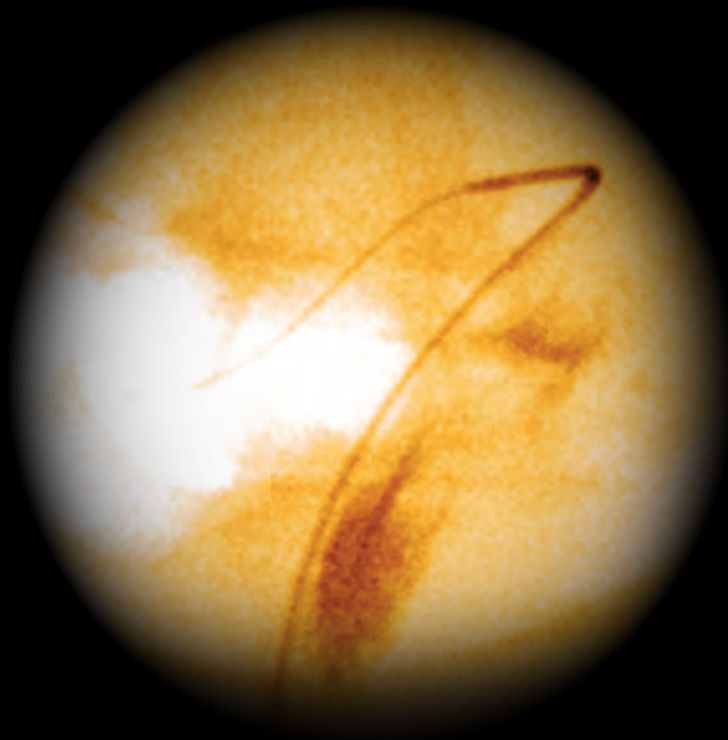


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Artery Embolization in the Treatment of Refractory Internal Hemorrhoids

Layth Alkhani, BS; Jessica Stewart, MD;
Osman Ahmed, MD

Hemorrhoidal artery embolization (HAE) is a minimally invasive, image-guided treatment for patients with bleeding internal hemorrhoids that are refractory to conservative and office-based therapies. In this review, the authors present emerging evidence that HAE provides symptom control comparable to hemorrhoidectomy with less postprocedural pain and shorter recovery, supporting its growing role in interventional radiology practice.

Tubal Infertility: A Review for Interventional Radiologists

Justin G. Roberts, MD; Jessica K. Stewart, MD

Proximal fallopian tube obstruction (pFTO) is a common and potentially reversible cause of female infertility, accounting for a significant proportion of tubal factor subfertility. This article reviews the anatomy and pathophysiology of pFTO and presents fluoroscopic transcervical fallopian tube recanalization (FTR) as a safe, minimally invasive, first-line therapy with favorable pregnancy outcomes in appropriately selected patients, while also discussing role of interventional radiologists in its diagnosis and management.

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BI-RADS 2025: A Language That Endures

Nina S. Vincoff, MD

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Some advances in medicine arrive with fanfare. Others change practice more quietly, gradually reshaping how we think, how we speak, and ultimately how we care for patients. The story of the Breast Imaging Reporting and Data System (BI-RADS®) proves that disruption in medicine does not always require cutting-edge technology, but can simply emerge from a shared commitment to better care.

BI-RADS and I share a few things in common. The original atlas was introduced in 1993, the year I graduated from medical school and began my career as a physician. I first encountered BI-RADS as a resident at UCSF the following year, learning breast imaging at the elbow of Dr Edward Sickles, one of the system's original architects and a true luminary in our field. In those days, BI-RADS was not yet the universal framework we now take for granted, but its authors had an ambitious vision: not merely a lexicon or reporting tool, but a foundation for clinical practice.

What began in breast imaging did not remain there. BI-RADS offered radiology a roadmap for quality, consistency, and patient- and family-centered care. Over time, the RADS framework spread across subspecialties, including LI-RADS®, PI-RADS®, TI-RADS®, Lung-RADS®, and O-RADS®. Each echoes the same essential promise that clear language, evidence-based categories, and measurable outcomes can elevate care.

More than 3 decades later, the arrival of BI-RADS 2025 feels less like a revision and more like a moment of reflection for our specialty. In this issue, *Applied Radiology* features the highlights of an interview with Dr Dana Smetherman, breast imager and Chief Executive Officer of the American College of Radiology, exploring why this update matters now, and how BI-RADS continues to guide breast imaging into the future. A video of the full discussion from RSNA 2025, facilitated by Editor-in-Chief Emeritus Dr Erin Simon Schwartz and Publisher Kieran Anderson, can be found on the *Applied Radiology* YouTube channel.

Breast imaging practice has transformed markedly since the last edition. Techniques once considered emerging, including digital breast tomosynthesis, contrast-enhanced mammography, and abbreviated MRI, are now woven into routine care. Treatment pathways have evolved as well, with neoadjuvant therapy and new surgical and minimally invasive approaches reshaping the radiologist's role. BI-RADS has expanded to incorporate these advances while maintaining the clarity, consistency, and clinical relevance that define its value.

The 2025 transition from atlas to a living digital manual reflects more than a change in format. It signals that BI-RADS is prepared to meet the broader currents shaping modern medicine, from the rapid rise of artificial intelligence to the growing emphasis on personalized care.

For those of us who have spent our careers in breast imaging, BI-RADS is woven so completely into daily work that it can feel almost invisible. Yet its impact is present in every clear recommendation, every measurable outcome, and every patient who leaves an imaging center with understanding instead of uncertainty.

In an era of rapid change, BI-RADS endures as something rare in medicine: an approach proven across decades and adaptable enough to guide what comes next.

Artery Embolization in the Treatment of Refractory Internal Hemorrhoids

Layth Alkhani, BS; Jessica Stewart, MD; Osman Ahmed, MD

Abstract

Hemorrhoids are naturally occurring vascular cushions within the anal canal, but today have become synonymous with the pathology of symptomatic hemorrhoid disease. Internal hemorrhoids are classified using the Goligher system, and management traditionally begins with conservative and office-based therapies. Many patients, however, experience recurrent or refractory symptoms, and surgical hemorrhoidectomy carries risks of pain, complications, and prolonged recovery. Hemorrhoid artery embolization (HAE) has emerged as a minimally invasive, nonsurgical option for patients with bleeding internal hemorrhoids. Appropriate patient selection is crucial, with ideal candidates experiencing significant bleeding refractory to conservative medical therapy. A comprehensive evaluation, including colonoscopy and bleeding/prolapse grading, is required. HAE involves selective embolization of hypertrophied superior rectal artery branches supplying the corpus cavernosum recti, using microspheres, coils, or both. The endpoint is decreased hemorrhoidal blush without complete stasis, followed by durable occlusion with coils. Data from nearly 400 patients demonstrate technical success rates of 93-100% and clinical success rates of 63-94%. Outcomes include significant reductions in bleeding scores, hemorrhoid size, prolapse, and pain. Patient satisfaction is high, with rates up to 94%. Early randomized controlled trial data show efficacy comparable to hemorrhoidectomy, but with shorter recovery, reduced pain, and fewer complications. HAE is a safe and effective treatment for internal hemorrhoids, particularly in patients with refractory bleeding who are not ideal surgical candidates or wish to avoid surgery. Future studies should refine patient selection, standardize procedural technique, and establish long-term comparative outcomes.

Keywords: hemorrhoids, hemorrhoid artery embolization (HAE), hemorrhoidectomy, Goligher classification, French bleeding score

Introduction

Hemorrhoids are naturally occurring clusters of vascular tissue within the anal canal consisting of smooth muscle, connective tissue, and arteriovenous connections.^{1,2} The hemorrhoidal cushions are naturally present and provide bulk to the anal canal that is believed

to aid in stool continence. Classified by their location, either proximal or distal to the dentate line, hemorrhoids have become synonymous with symptomatic hemorrhoid disease.³

Proximal hemorrhoids, also known as internal hemorrhoids, are most commonly characterized by rectal bleeding, particularly after defecation.^{4,5}

They are graded based on the degree of prolapse according to the Goligher classification system: grade I hemorrhoids bleed without prolapse, grade II hemorrhoids prolapse but spontaneously reduce, grade III hemorrhoids prolapse and require manual reduction, and grade IV hemorrhoids prolapse irreducibly.⁶ Owing to the asymptomatic nature of up to 40% of cases, hemorrhoidal prevalence is difficult to quantify.⁶ However, estimates of prevalence range from 4% to 40% globally; US studies report hemorrhoids to be the third-most common outpatient gastrointestinal diagnosis, with

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an estimated 3-4 million office and emergency department visits annually.^{3,6-10}

First-line treatment for symptomatic internal hemorrhoids typically consists of conservative medical management, including dietary changes to increase fiber and water intake, topical treatments such as corticosteroids or anti-inflammatory drugs, and other over-the-counter medications to relieve acute symptoms.^{4,6,11,12} For patients with grade I-III internal hemorrhoids and symptoms refractory to conservative treatments, office-based therapies such as rubber band ligation, sclerotherapy, and infrared photocoagulation are often offered. These procedures, however, can be painful and have recurrence rates of up to 50%.¹²⁻¹⁵ For grade III-IV internal hemorrhoids or recurrent hemorrhoids refractory to office-based treatments, surgical intervention is often required.^{1,3,4} However, surgery is invasive and painful, and carries a risk of complications, with a median recovery time of approximately 6 weeks.^{16,17}

Here, we review the potential of hemorrhoid artery embolization (HAE) as a minimally invasive option for patients with bleeding internal hemorrhoids but who otherwise are either not ideal candidates for, or do not wish to pursue, traditional surgical treatment.

Patient Selection

Appropriate patient selection is crucial in ensuring a successful clinical response to HAE. While many patients with internal hemorrhoids report pain, HAE is most successful in those experiencing bleeding. In those without bleeding, the superior rectal arteries that supply the corpus cavernosum recti may not be hypertrophied, thus limiting the efficacy of embolization. Ideal candidates for HAE include those whose quality of life has been severely impacted by bleeding and who either want to avoid surgery or are nonsurgical candidates. Notably, conservative management continues to be an important adjunct to HAE, as with

any treatment method, including diet modifications, lifestyle changes, sitz baths, and topical creams.

A comprehensive physical and medical history should be taken during the initial consultation with an interventional radiologist. Patients with a history of radiation to the pelvis should not be offered embolization, owing to potential compromise of collateral vasculature to the rectum, which increases the risk of ischemic complications. Patients must also undergo a colonoscopy to confirm the hemorrhoid grade and location, as well as ensure the absence of anorectal cancer. In addition to the degree of prolapse assessed using the Goligher classification (grade I-IV), the severity of bleeding should be measured using the French bleeding score, which ranges from 0 to 9. Both results should be documented during the initial visit.^{18,19}

As with any angiography-based procedure, patients with allergies to iodinated contrast media or renal failure have relative contraindications for HAE.

Procedural Technique

HAE seeks to reduce blood flow to the arteriovenous network within the internal hemorrhoidal cushions, known as the corpus cavernosum recti, via selective treatment of the corresponding terminal branches of the superior rectal arteries.

The interventional radiologist begins by obtaining femoral or radial arterial access using US guidance, after which they will place a vascular sheath and perform an aortogram to identify the origin of the inferior mesenteric artery (IMA). The IMA is then catheterized and a microcatheter is advanced into the superior rectal artery (SRA). Contrast is injected to identify the hypertrophied branches of the SRA supplying an area of contrast blush representing the corpus cavernosum recti within the hemorrhoidal cushions. The terminal branches of the SRA are then selectively catheterized, and angiography is used to confirm hemorrhoidal blush within the corpus cavernosum recti.

Embolization can be performed with microcoils, microspheres, or a combination of both. Microspheres are often diluted with iodinated contrast to a 1:20 ratio and are used sparingly to occlude distal arterial anastomoses within the middle and/or inferior rectal arteries. Microspheres range from 300 to 1200 μm in size; notably, larger microspheres showed better long-term efficacy and no minor ischemic findings compared with smaller sizes in one study.²⁰ Rather than complete stasis, the desired endpoint of microsphere embolization is decreased blush within the corpus cavernosum recti, which reduces the risk of ischemic complications. Microcoils are then used to embolize the distal SRAs to stasis and ensure durable occlusion.

Following embolization, the microcatheter is carefully withdrawn and angiography is performed to confirm major vessel patency and the absence of residual hemorrhoidal tissue blush below the pubic symphysis. The internal iliac arteries may also be catheterized to check for the presence of significant contributory middle rectal arteries. These may also be embolized using a technique similar to the one described. Some practices choose to perform middle rectal artery embolization only if they are visualized during SRA catheterization and/or if traditional superior rectal embolization fails to control bleeding.

Patients are observed for 2-6 hours and may be discharged the same day. Mild postprocedure discomfort can be treated with nonsteroidal anti-inflammatory drugs as needed. Follow-up is conducted 1-3 months post procedure.

Case Example

An adult patient presented with complaints of bright red bleeding upon defecation despite undergoing sclerotherapy and banding procedures for prolapsing internal hemorrhoids (Goligher grade II) 2 months prior. An arteriogram of the IMA (Figure 1) demonstrated dominant hypertrophied

Figure 1. (A) Inferior mesenteric arteriogram demonstrates dominant, hypertrophied superior rectal arteries supplying the corpus cavernosum recti below the pubic symphysis (white arrows). (B) Selective arteriogram of right-sided superior rectal arteries reveals blush of the corpus cavernosum recti (black arrows). (C) Two dominant terminal branches embolized using a combination of microspheres and coils. (D) Selective arteriogram of a dominant left superior rectal artery (SRA) branch reveals opacification of blush within the corpus cavernosum recti (thin white arrow). (E) This branch was also embolized using a combination of microspheres and coils. (F) Final arteriogram from the inferior mesenteric artery demonstrates no opacification of the corpus cavernosum recti following embolization of the dominant terminal branches of the SRAs.



SRA branches supplying the corpus cavernosum recti below the pubic symphysis. These branches were embolized using a combination of a small amount of 500-700 μm microspheres and microcoils. A second arteriogram of the IMA demonstrated absence of blush in corpus cavernosum recti. Anoscopy performed 1 month post procedure demonstrated shrinkage of the internal hemorrhoids to Goligher grade I, with no evidence of anorectal complications. The procedure resulted in complete resolution of bleeding.

Clinical Efficacy

Literature, including nearly 400 patients, describes the feasibility and efficacy of treating hemorrhoids with HAE.^{21,22} Rates of technical success, defined as embolization of at least one artery supplying the corpus cavernosum recti, range from 93% to 100% across all studies. Clinical success, often defined as reduction in French bleeding scores, is lower, ranging from 63% to 94%.²³⁻²⁷ The leading cause of clinical failure in these studies was rebleeding; however,

repeat embolization procedures have led to clinical success in some patients.²³ Meta-analyses have found statistically significant reductions in French bleeding scores (averaging ~ 2.7).^{21,22} However, studies have also reported improvements in pain, prolapse scores, and prolapse size of $>40\%$.^{25,27} The variability in clinical success rates can be attributed to patient selection (most failures occurred in advanced cases) and technique.

Patient satisfaction rates correlate with high clinical success; studies report satisfaction rates as high as

94%.^{25,28,29} A 2021 meta-analysis reported lower rebleeding rates for HAE using microspheres and coils.²²

Hemorrhoid Artery Embolization vs Surgery

Emerging evidence suggests efficacy rates for HAE comparable to surgical hemorrhoidectomy. A 2023 randomized control trial of 33 patients found no significant difference in hemorrhoidal symptom control between the embolization group and the surgical Ferguson hemorrhoidectomy group at 1 year post procedure.³⁰ By 12 months post treatment, the rates of bleeding, prolapse, and pruritus were similar between both groups. Along with similar clinical outcomes, the HAE group demonstrated a shorter recovery time. By 6 months post procedure, satisfaction rates between the 2 groups were also similar. The embolization group, however, reported lower pain and significantly lower medication usage. Patients undergoing hemorrhoidectomy report an average visual analog pain score of 6.6/10 during their first bowel movement compared with 0/10 in the embolization group.³⁰ These findings support the use of HAE as a clinically viable procedure that offers reduced pain and recovery time compared with hemorrhoidectomy.

Safety

Since 2015, major complications of HAE have been exceptionally rare, with only one case of rectosigmoidal ischemia reported.^{31,32} In this instance, the patient developed severe sigmoidal stenosis and was treated with 3 endoscopic dilations using a balloon dilator.³² This risk can be mitigated by careful embolic selection, placement, and coil use. Small superficial ulcerations have been reported in upward of 45% of patients.²⁰ However, these lesions are often asymptomatic and

heal without intervention. The risk of ulceration may be reduced through the use of larger beads (>900 µm), according to one study. Notwithstanding these complications, HAE maintains a favorable safety profile. Some patients report mild transient symptoms such as pelvic or anal aching. This often resolves within a few days after conservative management. In contrast to surgery, severe pain following HAE is rare. Owing to the nature of the procedure, HAE does not cause anal sphincter damage or mucosal resection. The minimally invasive nature of the procedure also eliminates the risks of wound infection or delayed healing.

Conclusion

Data from nearly 400 patients suggest that HAE is a safe and effective treatment option for patients with internal hemorrhoids, particularly those with refractory bleeding and those seeking less invasive hemorrhoid treatment. Early evidence has shown outcomes comparable to hemorrhoidectomy, along with shorter recovery time, reduced pain, and fewer complications. Future studies should focus on the identification of ideal patient demographics, standardization of procedural techniques such as access and embolic, and compare long-term outcomes against traditional management to establish HAE's role in hemorrhoid treatment.

References

- Fontem RF, Eyvazzadeh D. Internal hemorrhoid. In: *StatPearls*. StatPearls Publishing. Accessed September 7, 2025. <http://www.ncbi.nlm.nih.gov/books/NBK537182/>
- Sandler RS, Peery AF. Rethinking what we know about hemorrhoids. *Clin Gastroenterol Hepatol*. 2019;17(1):8-15. doi:10.1016/j.cgh.2018.03.020
- Sun Z, Migaly J. Review of hemorrhoid disease: presentation and management. *Clin Colon Rectal Surg*. 2016;29(1):22-29. doi:10.1055/s-0035-1568144
- Mott T, Latimer K, Edwards C. Hemorrhoids: diagnosis and treatment options. *Am Fam Physician*. 2018;97(3):172-179.

- Sheikh P, Régnier C, Goron F, Salmat G. The prevalence, characteristics and treatment of hemorrhoidal disease: results of an international web-based survey. *J Comp Eff Res*. 2020;9(17):1219-1232. doi:10.2217/ce-2020-0159
- Riss S, Weiser FA, Schwameis K, et al. The prevalence of hemorrhoids in adults. *Int J Colorectal Dis*. 2012;27(2):215-220. doi:10.1007/s00384-011-1316-3
- Johanson JF, Sonnenberg A. The prevalence of hemorrhoids and chronic constipation. An epidemiologic study. *Gastroenterology*. 1990;98(2):380-386. doi:10.1016/0016-5085(90)90828-o
- Elbataa H, Enaba M, Elkassas G, El-Kalla F, Elfert AA. Indications and outcome of colonoscopy in the middle of Nile delta of Egypt. *Dig Dis Sci*. 2011;56(7):2120-2123. doi:10.1007/s10620-010-1538-1
- Lee J-H, Kim H-E, Kang J-H, Shin J-Y, Song Y-M. Factors associated with hemorrhoids in Korean adults: Korean national health and nutrition examination survey. *Korean J Fam Med*. 2014;35(5):227. doi:10.4082/kjfm.2014.35.5.227
- Peery AF, Crockett SD, Barritt AS, et al. Burden of gastrointestinal, liver, and pancreatic diseases in the United States. *Gastroenterology*. 2015;149(7):1731-1741. doi:10.1053/j.gastro.2015.08.045
- Acheson AG, Scholefield JH. Management of haemorrhoids. *BMJ*. 2008;336(7640):380-383. doi:10.1136/bmj.39465.674745.80
- MacRae HM, McLeod RS. Comparison of hemorrhoidal treatment modalities. A meta-analysis. *Dis Colon Rectum*. 1995;38(7):687-694. doi:10.1007/BF02048023
- Pastor Peinado P, Ocaña J, Abadía Barno P, et al. Quality of life and outcomes after rubber band ligation for haemorrhoidal disease. *Langenbecks Arch Surg*. 2023;408(1):243. doi:10.1007/s00423-023-02990-6
- Brown SR, Tiernan JP, Watson AJM, et al. Haemorrhoidal artery ligation versus rubber band ligation for the management of symptomatic second-degree and third-degree haemorrhoids (HubBLE): a multicentre, open-label, randomised controlled trial. *Lancet*. 2016;388(10042):356-364. doi:10.1016/S0140-6736(16)30584-0
- Albuquerque A. Rubber band ligation of hemorrhoids: a guide for complications. *World J Gastrointest Surg*. 2016;8(9):614-620. doi:10.4240/wjgs.v8.i9.614
- Cristea C, Lewis CR. Hemorrhoidectomy. In: *StatPearls*. StatPearls Publishing. Accessed September 7, 2025. <http://www.ncbi.nlm.nih.gov/books/NBK549864/>
- Bouchard D, Abramowitz L, Castinel A, et al. One-year outcome of haemorrhoidectomy: a prospective multicentre french study. *Colorectal Dis*. 2013;15(6):719-726. doi:10.1111/codi.12090

- 18) Fathallah N, Beaussier H, Chatellier G, et al. Proposal for a new score: hemorrhoidal bleeding score. *Ann Coloproctol.* 2021;37(5):311-317. doi:10.3393/ac.2020.08.19
- 19) Goligher JC. *Surgery of the Anus, Rectum, and Colon.* Bailliere Tindall; 1980.
- 20) Küçükay MB, Küçükay F. Superior rectal artery embolization with tris-acryl gelatin microspheres: a randomized comparison of particle size. *J Vasc Interv Radiol.* 2021;32(6):819-825. doi:10.1016/j.jvir.2021.02.011
- 21) Nguyenhuy M, Xu Y, Kok HK, et al. Clinical outcomes following rectal artery embolisation for the treatment of internal haemorrhoids: a systematic review and meta-analysis. *Cardiovasc Interv Radiol.* 2022;45(9):1351-1361. doi:10.1007/s00270-022-03154-7
- 22) Makris GC, Thulasidasan N, Malietzis G, et al. Catheter-directed hemorrhoidal dearterialization technique for the management of hemorrhoids: a meta-analysis of the clinical evidence. *J Vasc Interv Radiol.* 2021;32(8):1119-1127. doi:10.1016/j.jvir.2021.03.548
- 23) Vidal V, Sapoval M, Sielezneff Y, et al. Emborrhoid: a new concept for the treatment of hemorrhoids with arterial embolization: the first 14 cases. *Cardiovasc Interv Radiol.* 2015;38(1):72-78. doi:10.1007/s00270-014-1017-8
- 24) Sun X, Xu J, Zhang J, Jin Y, Chen Q. Management of rectal bleeding due to internal haemorrhoids with arterial embolisation: a single-centre experience and protocol. *Clin Radiol.* 2018;73(11):985. doi:10.1016/j.crad.2018.07.105
- 25) Zakharchenko A, Kaitoukov Y, Vinnik Y, et al. Safety and efficacy of superior rectal artery embolization with particles and metallic coils for the treatment of hemorrhoids (Emborrhoid technique). *Diagn Interv Imaging.* 2016;97(11):1079-1084. doi:10.1016/j.diii.2016.08.002
- 26) Moussa N, Bonnet B, Pereira H, et al. Mid-term results of superior rectal artery and coils for hemorrhoidal embolization with particles bleeding. *Cardiovasc Interv Radiol.* 2020;43(7):1062-1069. doi:10.1007/s00270-020-02441-5
- 27) Bagla S, Pavidapha A, Lerner J, et al. Outcomes of hemorrhoidal artery embolization from a multidisciplinary outpatient interventional center. *J Vasc Interv Radiol.* 2023;34(5):745-749. doi:10.1016/j.jvir.2023.01.023
- 28) Tradi F, Louis G, Giorgi R, et al. Embolization of the superior rectal arteries for hemorrhoidal disease: prospective results in 25 patients. *J Vasc Interv Radiol.* 2018;29(6):884-892. doi:10.1016/j.jvir.2018.01.778
- 29) Panneau J, Mege D, Di Bisceglie M, et al. Hemorrhoidal disease: what role can rectal artery embolization play? *Front Surg.* 2024;11:1474799. doi:10.3389/fsurg.2024.1474799
- 30) Falsarella PM, Nasser F, Affonso BB, et al. Embolization of the superior rectal arteries versus closed hemorrhoidectomy (Ferguson technique) in the treatment of hemorrhoidal disease: a randomized clinical trial. *J Vasc Interv Radiol.* 2023;34(5):736-744. doi:10.1016/j.jvir.2023.01.022
- 31) Talaie R, Torkian P, Moghadam AD, et al. Hemorrhoid embolization: a review of current evidences. *Diagn Interv Imaging.* 2022;103(1):3-11. doi:10.1016/j.diii.2021.07.001
- 32) Eberspacher C, Ficuccilli F, Tessieri L, et al. Annoyed with haemorrhoids? Risks of the emborrhoid technique. *Dig Dis Sci.* 2021;66(11):3725-3729. doi:10.1007/s10620-021-07208-7

Tubal Infertility: A Review for Interventional Radiologists

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Abstract

Proximal fallopian tube obstruction (pFTO) is a common and potentially reversible cause of female subfertility. Image-guided techniques have established interventional radiology as a central discipline in the diagnosis and treatment of this condition. This review summarizes the pathophysiology, imaging evaluation, and management of pFTO, with a primary focus on fluoroscopy-guided transcervical fallopian tube recanalization (FTR). Normal tubal anatomy, mechanisms of proximal obstruction, and the diagnostic and therapeutic roles of hysterosalpingography are reviewed. Technical aspects of FTR, including patient selection, procedural technique, periprocedural care, and follow-up, are described. Published outcomes demonstrate technical success rates exceeding 90% with favorable pregnancy outcomes in appropriately selected patients and low complication rates. Given its safety profile, cost-effectiveness, and ability to immediately restore tubal patency, fluoroscopic FTR represents an effective first-line, minimally invasive treatment for women with isolated pFTO and an important application of image-guided therapy in infertility care.

Keywords: tubal infertility, fallopian tube obstruction, fallopian tube recanalization

Introduction

Subfertility at a Glance

Subfertility is defined as the inability to conceive within 12 months of frequent, unprotected intercourse in women younger than 35 years, or within 6 months in women 35 years and older, and can be either primary (never achieved pregnancy) or secondary (unable to achieve subsequent pregnancy).^{1,2} More

than 15% of child-seeking couples are affected by infertility globally,² and the incidence is thought to be increasing, in part due to a trend in couples rearing offspring at a later age, when female fertility and male sperm counts are both in decline, and cumulative exposure to sexually transmitted infections is increased.³ Most subfertility is attributable to a male factor (~25%), ovarian factor (~25%), or fallopian tube factor (~25%). Another 10% arise from a uterine or

peritoneal cause, and in 40% of cases, both male and female factors are thought to contribute. Despite a wealth of knowledge on the causes of infertility, still up to one-third of workups yield no identifiable male or female factor, known as unexplained infertility.⁴⁻⁶

The American College of Obstetricians and Gynecologists, American Society for Reproductive Medicine (ASRM), and National Institute for Health and Care Excellence (NICE) recommend that any child-seeking woman who meets criteria for infertility, or who has a condition known to cause infertility, should be offered an evaluation.^{2,4,7} The workup consists of 4 essential components: (1) a comprehensive medical history of both partners (if one exists), (2) targeted physical examination of the female partner, (3) semen analysis, (4) and testing to exclude common ovarian, tubal, and uterine causes of infertility. The latter

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generally includes, but is not limited to, fluoroscopic hysterosalpingography (HSG) to assess the uterine and tubal structures.

Fallopian Tube Obstruction

Tubal factor infertility accounts for 25-35% of all female subfertility worldwide, among which 10-25% is due to proximal fallopian tube obstruction (pFTO).^{3,5,8,9} There are many causes of pFTO, including inspissated debris (eg mucus, blood), tubal spasm (pseudo-obstruction), salpingitis isthmica nodosa (SIN), pelvic inflammatory disease (PID), endometriosis, polyps, and congenital Müllerian anomalies. However, it is estimated that tubal spasm and mucus plugs alone account for up to 40% of cases.^{6,10} It is also estimated that half of patients with bilateral obstructions have isolated short segment disease and no pelvic adhesions.¹¹ The remaining causes relate to distal fallopian tube obstruction (dFTO), which is virtually always caused by chronic adhesions from PID or other inflammatory processes.⁹ It is important to distinguish this entity from pFTO, as dFTO leads to hydrosalpinges and is managed surgically.⁹

Before discussing treatment options for pFTO, it is important to first understand normal tubal anatomy, physiology, and underlying mechanisms that lead to subfertility. Fallopian tubes are 7-14 cm in length, trumpet-shaped, musculomembranous conduits between the ovaries and the uterus. Each tube is comprised of an inner mucosal membrane made up of 2 simple columnar epithelial cell types, a middle muscularis comprised of 2 smooth muscle layers, and an outer serosa that is contiguous with the mesosalpinx of the broad ligament.¹⁰ Their course is straight or slightly curved in 60% of women and tortuous in 40%. This nonlinear anatomy is thought to assist in preventing access of vaginal flora into the peritoneum. However, this also makes the oviduct prone to accumulation of secretions and inflammation.¹²

The proximal fallopian tube comprises roughly the first 4 cm of duct with a diameter of about 1 mm and consists of

a proximal 1.5-2.5 cm intramural segment (also known as the uterine or interstitial segment) and distal 2-3 cm isthmic (or extrauterine) segment. The intramural segment has a thick muscular wall relatively devoid of ciliated epithelium and empties into the uterus through an ostium at the apex of the uterine cornu or horn. This combination of features renders the intramural segment prone to spasm and transient obstruction by upstream secretions generated at the junction of the intramural and isthmic segments, or uterotubal junction, during the follicular phase of the menstrual cycle.⁶

The distal fallopian tube is comprised of an ampulla, infundibulum, and fimbriae that gradually increase in caliber toward the ovary. The distal fimbriated end is open to the peritoneal cavity, where it performs a sweeping motion over the ovary allowing an ovulated egg to be drawn into the oviduct. Once in the duct, the ovum becomes available for fertilization, where a coordinated effort between ciliary action, secretory flow, and smooth muscle contractions optimizes the environment for sperm ascent, ovum descent, and transportation of a developing embryo to the endometrium. These multiple essential functions of the fallopian tube are modulated by an intricate network of sympathetic and parasympathetic nerve fibers, estrogen, progesterone, and prostaglandins, all of which play a vital role in achieving spontaneous pregnancy.^{10,13} The focus of this article is to discuss the role of the interventional radiologist (IR) in the workup and management of pFTO.

Treatment of Proximal FTO: Then and Now

Since its inception in the early 20th century, diagnostic transcervical HSG was recognized for having therapeutic value in the management of pFTO.^{14,15} Its therapeutic effect on infertility was first described in 1923 when a report noted increased pregnancy rates following successful intrauterine insufflation of carbon dioxide to overcome tubal occlusions.¹⁶ In 1931, a landmark paper detailed a similar phenomenon

using oil-based contrast media.¹⁷ The therapeutic advantage of lipid-soluble contrast over aqueous-based agents was subsequently determined.^{5,18} Today, the safety and efficacy of HSG using lipid-soluble contrast is well established. Current evidence from randomized clinical trials comparing HSG to no treatment suggests at least 3-fold increased odds of achieving both clinical pregnancy and live birth in the setting of pFTO.⁵

In the 1980s, with the advent of improved low-profile coaxial catheter and wire technologies, selective salpingography and tubal recanalization emerged as a superior treatment option for tubal infertility.¹⁹⁻²¹ The contemporary fluoroscopy-guided transcervical fallopian tube recanalization (FTR) technique used today combines standard HSG and coaxial angiography techniques from the IR's armamentarium.²¹ The FTR procedure augments the therapeutic benefit of conventional HSG via selective tubal flushing of contrast and catheter- and wire-directed mechanical clearance of tubal debris and adhesions. Moreover, selective salpingography has a greater positive predictive value for diagnosing tubal obstruction compared to HSG.^{5,22} While there are many underlying etiologies and nuances of tubal obstruction, histopathologic findings in the proximally occluded fallopian tube include only 3 entities: fibrotic adhesions, amorphous intraluminal debris, or normal histology (in which case transient spasm or dislodged debris is presumed).²³ Several mechanisms have been proposed to explain the improved fertility outcomes from FTR, which include not only direct mechanical effects, but also indirect effects on the endometrium and peritoneum.⁵ With regard to the endometrium, there is evidence to suggest that changes in the local immunological environment may increase endometrial receptivity by favorably modulating leukocyte populations essential to early embryo development.²⁴ With regard to the peritoneum, oil-based contrast media modulate dendritic and regulatory T cell populations specific to pregnancy.²⁵

There is also evidence for inhibition of sperm phagocytosis by peritoneal macrophages by altering interleukin and prostaglandin production.²⁶

Guidelines and Indications for FTR

Current ASRM and NICE infertility guidelines recommend the use of selective salpingography and recanalization as a first-line approach to diagnosis and management of proximal tubal obstruction.^{4,7} FTR is indicated for women with primary or secondary infertility who have confirmed unilateral or bilateral proximal tubal obstruction and no other significant infertility factors.^{7,27} The procedure can be performed as a standalone therapy, or as a neoadjuvant with microsurgical tubal anastomosis or in vitro fertilization (IVF) and intracytoplasmic sperm injection (ICSI).^{6,28} Tubal cannulation can be performed fluoroscopically or hysteroscopically; however, a fluoroscopic approach is preferred as it allows simultaneous diagnosis and treatment. Moreover, fluoroscopy is less costly and mitigates the need for sedation or anesthesia.⁷ Contraindications to FTR include active pelvic infection, pelvic malignancy, or pregnancy.⁶ Relative contraindications include the presence of concomitant dFTO, cornual fibroids, severely distorted uterine anatomy, Müllerian anomalies, or severe SIN.⁷

Patient Evaluation

Patients are preferably seen for preprocedural consultation in a dedicated IR clinic. Key historical factors include duration of infertility, coital frequency and timing, pregnancy history, menstrual history, sexual and gynecologic history, prior contraceptive methods, prior abdominopelvic surgeries, and drug and alcohol use.² Current medications and any supplements should be reviewed for potential teratogenic agents. Any prior infertility studies should be reviewed for evidence of other contributing factors to

infertility, including results of ovulatory function testing, ovarian reserve testing, and male semen analysis, which will aid in the prognostication of FTR outcomes and appropriate patient counseling. Perhaps the most important role during the clinical encounter is to set expectations and to explain to the patient and her family the risks, benefits, and alternatives to FTR. This ensures an open dialogue that allows for shared decision-making and informed consent.⁶

Prior imaging should be reviewed for procedural planning and to identify any findings that could potentially compromise the success of FTR, such as hydrosalpinges, pelvic adhesions, endometriosis, severe SIN, or complex uterine anatomy.⁶ The diagnosis of pFTO is established by demonstrating a lack of patency within the first 4 cm of the fallopian tube, evaluated either by HSG, hysterosalpingo-contrast sonography, or diagnostic laparoscopy and chromotubation. Fallopian tube recanalization is optimally performed during the follicular phase of the ovarian cycle and proliferative phase of the endometrial cycle, that is, days 7-14. This increases the chances of conception and decreases the risk of contrast intravasation.¹⁸ A preprocedural pregnancy test is not routinely required provided that FTR is performed during the follicular phase.²⁷ Note that ensuring optimal timing requires a detailed menstrual history of the patient's ovulatory cycle, especially if intervals are irregular.

Technique

Patient Preparation

Five days total of doxycycline antibiotic prophylaxis is recommended for all patients undergoing FTR. Regimens can include 100 mg twice daily beginning 2 days before, or a single 200 mg dose immediately prior to the procedure followed by 100 mg twice daily for 4 additional days.^{6,28} Patients should

fast for a minimum of 6 hours in the event that sedation is needed. If sedation is administered, standard moderate intravenous sedation with midazolam and fentanyl titrated to comfort is recommended.

In the angiography suite, the patient is placed in the lithotomy position with the lower extremities fastened in table stirrups. The perineum is prepped and draped in the usual sterile fashion. At this stage, patient communication is crucial to minimize discomfort. An appropriately sized, lubricated speculum is then inserted slowly into the vagina and gently opened until the cervix is in clear view. Optional warming of the speculum can be performed prior to insertion by rinsing it under warm water or holding it in a gloved hand. Once in view, the cervix is cleansed using sterile betadine swabs.⁶

Intrauterine Access and HSG

Transcervical uterine access is obtained using a balloon occluding sheath or catheter, such as a 9-French intrauterine access balloon catheter (Cook Medical, Bloomington, Indiana) or similar device that can accommodate the passage of a 5- or 6-French diagnostic catheter while occluding the internal os. A tenaculum should be on standby in the event that excessive mobility or difficult anatomy prohibits successful cervical cannulation. If this step is required, a topical anesthetic applied to the cervix is encouraged prior to engaging the tenaculum at the 12 o'clock position to fix the cervix in place. The tenaculum can also help stabilize the uterus for tubal cannulation. Next, a conventional hysterosalpingogram is performed to confirm the presence of pFTO; gentle back tension can be applied to the balloon catheter to prevent leakage of contrast through the os.^{6,12} Water- or lipid-soluble contrast is injected through the catheter into the uterine cavity until all its contours are well-delineated, and fluoroscopic runs are reviewed for evidence of tubal patency (Figure 1). Contrast should be prepared as a 50% volume dilution in normal saline to ensure

Figure 1. Initial hysterosalpingogram. Initial hysterosalpingogram with iodinated contrast demonstrates absence of opacification of the proximal aspect of the bilateral fallopian tubes, compatible with proximal fallopian tube obstruction.



adequate visualization. A slow and steady injection is recommended to minimize tubal spasm and prevent a false positive result.⁶ If the tubes are not visualized and spasm is suspected, turning the patient prone will occasionally result in their opacification.¹²

Selective Salpingography and Recanalization

Selective catheterization of the tubal ostium is then preferably performed with a triaxial system that includes an angled 5-French catheter, 0.035-inch hydrophilic guidewire, 3-French microcatheter, and 0.018 microwire in order to engage the laterally oriented cornu and cross the obstruction. The Rösch-Thurmond fallopian tube catheterization set (Cook Medical, Bloomington, Indiana) is ideal for this purpose. First, the 5-French catheter is advanced over the 0.035-inch guidewire into the uterine cornu (Figure 2), and a salpingogram is performed. In a minority of cases, a patent tube will be revealed, and no further intervention is needed.²⁸ Assuming persistent tubal obstruction, the microcatheter and microwire are then coaxially advanced across the proximal occlusion, with gentle probing movements of the microwire as necessary to clear the obstruction (Figure 3). If the proximal tube is acutely angulated or the site of obstruction is within the isthmic segment, exchanging the system for a softer, tapered guidewire and catheter may be preferred.^{6,12}

Figure 2. Selective catheterization. A 5-French angled catheter and 0.035-inch guidewire are used to selectively catheterize the right fallopian tube.

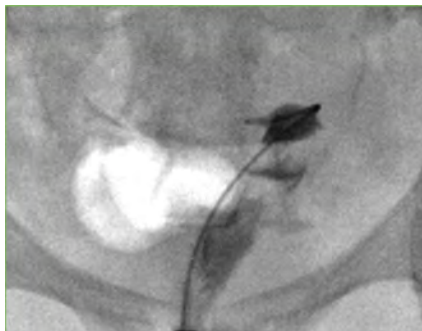


Figure 4. Selective salpingography. Selective salpingography performed via the 5-French catheter from the ostium of the right fallopian tube reveals opacification of the entire length of the fallopian tube with free spillage of contrast into the peritoneum following successful fallopian tube recanalization.



Following recanalization, the crossing wire is removed and selective salpingography through the 3-French catheter is performed to confirm luminal patency (Figure 4). Lastly, with the 5-French catheter still wedged in the tubal ostium, the 3-French catheter is removed, and a final salpingogram is performed (Figure 5). Full-strength oil-based contrast is preferred over water-soluble contrast due to its association with higher clinical pregnancy rates.⁵ Rarely, tubal perforation may give the false impression of a patent tube, in which case enough of the agent should be injected to ensure spillage of contrast through the distal fimbriated end of the tube.

If bilateral obstruction is present, attention is then turned to the contralateral side, and the procedure is repeated

Figure 3. Wire recanalization. The glide wire is advanced to the distal aspect of the right fallopian tube, clearing the proximal fallopian tube of debris.

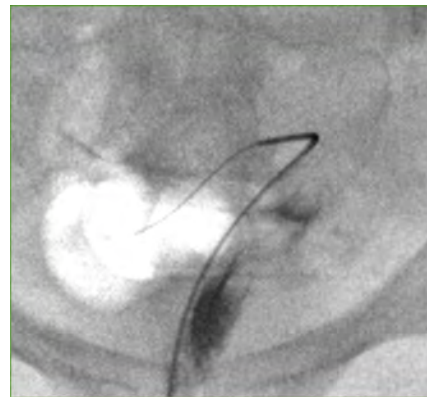


Figure 5. Final salpingogram. Final salpingogram again demonstrates complete opacification of the right fallopian tube with free spillage of contrast into the peritoneum following treatment of proximal fallopian tube obstruction with recanalization.



in the same manner. All devices are then removed from the uterus and vagina, the patient is cleaned, and the procedure is concluded. Total procedure time is approximately 30 minutes, with fluoroscopy time usually under 10 minutes.¹²

Postprocedural Care and Follow-Up

Patients are discharged the same day following a brief observation period to monitor pain levels and ensure no complications. Postprocedural expectations and return precautions should be reviewed with patients prior

to discharge. Mild uterine cramping and vaginal bleeding or discharge is expected within the first 24 hours and may continue for up to 3 days. Oral nonsteroidal anti-inflammatory agents are prescribed to be taken as needed for pain control. Patients are encouraged to abstain from sexual intercourse for 24 hours but can otherwise resume their usual physical activity after the procedure.⁶ If not already doing so, patients should be advised to start taking daily prenatal vitamins, as the chance of pregnancy is highest immediately following FTR. Given the diminishing chances of spontaneous conception over time, repeat FTR or an alternative therapy should be considered 6-12 months after successful recanalization if pregnancy has not yet ensued.⁷ In cases of failed FTR, one study revealed that 93% of patients had evidence of SIN, chronic salpingitis, or oblitative fibrosis on excised proximal tube specimens.²⁹ Lastly, patients should be advised to continue follow-up with their fertility specialist, as couples may have additional fertility treatments planned after tubal patency is restored.¹²

Outcomes

Technical success for restoring tubal patency via fluoroscopic FTR ranges from 70% to 100%,^{8,23} with most contemporary reports citing success rates of well over 90%.^{30,31} However, tubal re-occlusion is common following FTR and may warrant early reintervention in certain cases, as repeat recanalization is feasible and has resulted in successful pregnancies.¹¹ Among women who do not conceive within one year, rates of tubal re-occlusion are as high as 25%, 50%, and 60% at 3, 6, and 12 months, respectively.^{11,32}

Due to differences in reporting methods and heterogeneity among study populations, clinical success rates of FTR vary considerably within the literature. One meta-analysis found a clinical pregnancy rate of 27% at 12 months, with a live birth rate of 22%³³; however, many women had a combination of infertility

factors and therefore did not reflect true pFTO rates. In carefully selected patients who have undergone a full workup with no other identifiable causes of infertility other than proximal tubal occlusion, 12 month clinical pregnancy rates in the range of 41-58% are widely reported.^{30-32,34} In general, favorable factors are thought to include younger age (< 35 years), secondary infertility, duration of infertility < 5 years, and pFTO as the sole cause of infertility,³⁰ although this has yet to be elucidated by large-scale randomized data. At this time, there is no convincing evidence for a difference in outcomes between unilateral and bilateral pFTO.^{30,33} While FTR is an effective treatment for unilateral pFTO, it should be noted that similar pregnancy rates have been achieved with ovarian stimulation therapy and intrauterine insemination in this population.⁷ Lastly, multiple retrospective studies have demonstrated no significant difference in pregnancy outcomes between fluoroscopic and hysteroscopic recanalization techniques, which is important to consider when counseling patients and communicating with colleagues across disciplines.^{7,33}

Given the substantial heterogeneity within the literature, it is best to advise patients with a conservative estimate of pregnancy outcomes. Patients should be counseled only after a routine infertility workup has demonstrated that they are a good candidate for FTR. In general, patients should be advised that their chance of conception is at least 30% shortly after recanalization, and that if they do not achieve pregnancy after 6 months, there is a 50% chance that re-occlusion has occurred.³⁵

Adverse Events

Primary complications include ectopic pregnancy, spontaneous abortion, tubal perforation, and pelvic infection. Ectopic pregnancy has been reported to occur in 3-9% of patients.^{4,33} However, the percentage directly attributable to FTR is unclear, as patients with tubal disease

have a baseline elevated risk of ectopic pregnancy prior to intervention, and similar ectopic rates are seen following IVF. The rate of spontaneous abortion ranges from 4% to 8% among large cohorts and does not appear to differ from hysteroscopic techniques.³³ Asymptomatic tubal perforation occurs in 2-11% of cases and requires no additional monitoring or intervention.^{4,7,11} It is unclear at this time whether perforation has an impact on subsequent pregnancy outcomes, as case numbers are overall low. The rate of pelvic infection, although not reported in the literature, is a theoretical risk of any transcervical procedure and is generally assumed to be < 1% following FTR.⁶

Historically, intravasation, or the backflow of contrast into the blood or lymphatic vessels, was considered an important risk factor of HSG, said to occur in approximately 1-7% of cases.⁵ While intravasation occasionally occurs, it is nearly always asymptomatic. Nevertheless, there is a potential, although mostly historical, risk of allergic reaction or embolic phenomena. Lastly, there is a potential risk of harm from radiation exposure. However, studies have shown that the dose absorbed by the ovaries during FTR is < 1 Rad (10 Gy), or roughly equivalent to one barium enema, and is within the acceptable limits for women of reproductive age.¹¹ Importantly, while a fertilized gamete is highly radiosensitive, an unfertilized ovum is relatively radioresistant.⁶

Alternatives and Future Applications

It is imperative that IRs performing FTR have fundamental knowledge of both the pathophysiology of and treatments for proximal tubal disease. Microsurgical tubocornual anastomosis is a well-established procedure for pFTO that involves careful resection of the occluded proximal segment and surgical conjoining of the non-diseased tube to the uterine cornu, with pregnancy rates of up to 58%

over the course of months to years, and an ectopic rate of 4%.¹¹ While the procedure has fallen out of favor in recent years due to its invasiveness and lag time in efficacy, it is still used as a first-line treatment for tubal ligation reversal.⁷

The number of couples seeking IVF and ICSI treatment for tubal infertility has dramatically increased over the past few decades and continues to increase in popularity and efficacy. Recent global estimates of successful per-cycle pregnancy rates and live birth rates are 24% and 18% for IVF, and 26% and 19% for ICSI, respectively.³⁶ The main disadvantages of these therapies include high cost, potential need for multiple treatments and several weeks of monitoring, and the risks of twin gestation and ovarian hyperstimulation syndrome.^{7,36} Thus, for infertility due to isolated proximal tubal obstruction, FTR is preferred as the first-line option, with microsurgical anastomosis and IVF/ICSI reserved for refractory cases.

A number of recent developments have emerged as potential future treatment options for pFTO. Fertiloscopy and fallopscopy are among such developments.⁴ Similar to conventional hysteroscopy, fallopscopy allows direct visualization of the working field, but on a much smaller scale. The new low-profile scopes are designed to navigate into the fallopian tube and can be used to cross tubal obstructions under direct view. However, further study is needed to demonstrate feasibility, safety, and efficacy. Another area of ongoing research is in the development of solutions to prevent re-occlusion after FTR. A number of small studies have used various biochemical agents injected into the fallopian tubes after recanalization. A recent 4-arm randomized controlled trial studied rates of pregnancy and re-occlusion following injection of beta-(1-4)-2-amino-2-deoxy-D-glucosechitosan (chitosan) and *Salvia miltiorrhiza* (Dan-shen), 2 commonly used Eastern medicinal compounds.³²

At 12 months post-FTR, cumulative pregnancy rates for each group were 31% (control), 47% (chitosan), 43% (Dan-shen), and 64% (chitosan plus Dan-shen), which was statistically significant. For patients who had not conceived by 12 months, tubal patency rates were 39% (control), 78% (chitosan), 77% (Dan-shen), and 94% (chitosan plus Dan-shen). While the mechanisms by which these agents might work to prevent re-occlusion are not well understood, these results show promise for future research into adjuvant agents other than oil-based contrast.

Conclusions

pFTO is a common cause of female infertility in which IRs have a primary role in the diagnosis, treatment, and management of. Due to its favorable safety and efficacy profile, fluoroscopic transcervical FTR has become the first-line treatment of choice for pFTO. In experienced hands, FTR is associated with a technical success rate of up to 100% and 1-year pregnancy rate of up to 60%, with immediate onset of efficacy and extremely low rate of complications. Tubal re-occlusion is common after FTR, occurring in up to 50% of patients after 6 months. Therefore, patients should be closely followed by their IR and infertility specialist in the immediate postprocedural period and offered repeat intervention or prompt referral for appropriate treatment if pregnancy is unlikely to occur.

References

- 1) Farquhar CM, Bhattacharya S, Repping S, et al. Female subfertility. *Nat Rev Dis Primers*. 2019;5(1):7. doi:10.1038/s41572-018-0058-8
- 2) American College of Obstetricians and Gynecologists. Infertility workup for the women's health specialist: ACOG committee opinion, number 781. *Obstet Gynecol*. 2019;133(6):e377-e384. doi:10.1097/AOG.0000000000003271
- 3) Evers JLH. Female subfertility. *Lancet*. 2002;360(9327):151-159. doi:10.1016/S0140-6736(02)09417-5

- 4) National Institute for Health and Care Excellence. *Fertility Problems [Quality Standard No. QS73]*; 2014. <https://www.nice.org.uk/guidance/qs73/resources>
- 5) Wang R, Watson A, Johnson N, et al. Tubal flushing for subfertility. *Cochrane Database Syst Rev*. 2020;10(10):CD003718. doi:10.1002/14651858.CD003718.pub5
- 6) Kohi MP. Interventional radiologist's approach to fallopian tube recanalization. *Tech Vasc Interv Radiol*. 2021;24(1):100736. doi:10.1016/j.tvir.2021.100736
- 7) Practice Committee of the American Society for Reproductive Medicine. Role of tubal surgery in the era of assisted reproductive technology: a committee opinion. *Fertil Steril*. 2021;115(5):1143-1150. doi:10.1016/j.fertnstert.2021.01.051
- 8) Hou HY, Chen YQ, Li T-C, et al. Outcome of laparoscopy-guided hysteroscopic tubal catheterization for infertility due to proximal tubal obstruction. *J Minim Invasive Gynecol*. 2014;21(2):272-278. doi:10.1016/j.jmig.2013.09.003
- 9) Role of tubal surgery in the era of assisted reproductive technology: a committee opinion. *Fertil Steril*. 2015;103(6):e37-e43. doi:10.1016/j.fertnstert.2015.03.032
- 10) Honoré GM, Holden AEC, Schenken RS. Pathophysiology and management of proximal tubal blockage. *Fertil Steril*. 1999;71(5):785-795. doi:10.1016/S0015-0282(99)00014-x
- 11) Thurmond AS. Fallopian tube recanalization: technique, results, and complications. *J Vasc Interv Radiol*. 2002;13(2):P252-P254. doi:10.1016/S1051-0443(02)70162-0
- 12) Thurmond AS. Fallopian tube catheterization. *Semin Intervent Radiol*. 2008;25(4):425-431. doi:10.1055/s-0028-1102995
- 13) Lyons RA, Saridogan E, Djahanbakhch O. The reproductive significance of human fallopian tube cilia. *Hum Reprod Update*. 2006;12(4):363-372. doi:10.1093/humupd/dml012
- 14) Rindfleisch W. Darstellung des cavum uteri. *Klin Wochenschr*. 1910;47:780.
- 15) Smith WT. On a new method of treating sterility, by the removal of obstructions of the fallopian tubes. *Lancet*. 1849;53(1342):529-531. doi:10.1016/S0140-6736(02)65312-7
- 16) Peterson R. Therapeutic value of transuterine gas inflation. *JAMA*. 1923;81(12):980. doi:10.1001/jama.1923.02650120012004
- 17) Robins SA, Shapira AA. The value of hysterosalpingography. *N Engl J Med*. 1931;205(8):380-395. doi:10.1056/NEJM193108202050804
- 18) Gillespie HW. The therapeutic aspect of hysterosalpingography. *Br J Radiol*. 1965;38(448):301-302. doi:10.1259/0007-1285-38-448-301

- 19) Platia MP, Krudy AG. Transvaginal fluoroscopic recanalization of a proximally occluded oviduct. *Fertil Steril*. 1985;44(5):704-706. doi:10.1016/s0015-0282(16)48992-2
- 20) Jansen R, Anderson J. Catheterisation of the fallopian tubes from the vagina. *Lancet*. 1987;330(8554):309-310. doi:10.1016/S0140-6736(87)90893-2
- 21) Thurmond AS, Novy M, Uchida BT, Rösch J. Fallopian tube obstruction: selective salpingography and recanalization. *Radiology*. 1987;163(2):511-514. doi:10.1148/radiology.163.2.3562835
- 22) Woolcott R, Fisher S, Thomas J, Kable W. A randomized, prospective, controlled study of laparoscopic dye studies and selective salpingography as diagnostic tests of fallopian tube patency. *Fertil Steril*. 1999;72(5):879-884. doi:10.1016/s0015-0282(99)00382-9
- 23) Thurmond AS. Selective salpingography and fallopian tube recanalization. *AJR Am J Roentgenol*. 1991;156(1):33-38. doi:10.2214/ajr.156.1.1898568
- 24) Yun AJ, Lee PY. Enhanced fertility after diagnostic hysterosalpingography using oil-based contrast agents may be attributable to immunomodulation. *AJR Am J Roentgenol*. 2004;183(6):1725-1727. doi:10.2214/ajr.183.6.01831725
- 25) Izumi G, Koga K, Takamura M, et al. Oil-Soluble Contrast Medium (OSCM) for hysterosalpingography modulates dendritic cell and regulatory T cell profiles in the peritoneal cavity: a possible mechanism by which OSCM enhances fertility. *J Immunol*. 2017;198(11):4277-4284. doi:10.4049/jimmunol.1600498
- 26) Johnson JV, Montoya IA, Olive DL. Ethiodol oil contrast medium inhibits macrophage phagocytosis and adherence by altering membrane electronegativity and microviscosity. *Fertil Steril*. 1992;58(3):511-517. doi:10.1016/s0015-0282(16)55254-6
- 27) Thurmond AS, Machan LS, Maubon AJ, et al. A review of selective salpingography and fallopian tube catheterization. *Radiographics*. 2000;20(6):1759-1768. doi:10.1148/radiographics.20.6.g00nv211759
- 28) Thurmond AS. Fallopian tube recanalization BT. In: Jafri SZH, Diokno AC, Amendola MA, eds. *Lower Genitourinary Radiology: Imaging and Intervention*. Springer New York. 1998:501-507. doi:10.1007/978-1-4612-1648-3_29
- 29) Letterie GS, Sakas EL. Histology of proximal tubal obstruction in cases of unsuccessful tubal canalization. *Fertil Steril*. 1991;56(5):831-835. doi:10.1016/S0015-0282(16)54651-2
- 30) Al-Omari MH, Obeidat N, Elheis M, Khasawneh RA, Gharaibeh MM. Factors affecting pregnancy rate following fallopian tube recanalization in women with proximal fallopian tube obstruction. *J Clin Med*. 2018;7(5):110. doi:10.3390/jcm7050110
- 31) Shen H, Cai M, Chen T, et al. Factors affecting the success of fallopian tube recanalization in treatment of tubal obstructive infertility. *J Int Med Res*. 2020;48(12):300060520979218. doi:10.1177/0300060520979218
- 32) Huang C, He X, Luo W, Chen H, Huang Y. Combined chitosan and dan-shen injection for long-term tubal patency in fallopian tube recanalization for infertility. *Drug Deliv Transl Res*. 2019;9(4):738-747. doi:10.1007/s13346-018-00611-0
- 33) De Silva PM, Chu JJ, Gallos ID, et al. Fallopian tube catheterization in the treatment of proximal tubal obstruction: a systematic review and meta-analysis. *Hum Reprod*. 2017;32(4):836-852. doi:10.1093/humrep/dex022
- 34) Hayashi M, Hoshimoto K, Ohkura T. Successful conception following fallopian tube recanalization in infertile patients with a unilateral proximally occluded tube and a contralateral patent tube. *Hum Reprod*. 2003;18(1):96-99. doi:10.1093/humrep/deg006
- 35) Thurmond AS, Rösch J. Nonsurgical fallopian tube recanalization for treatment of infertility. *Radiology*. 1990;174(2):371-374. doi:10.1148/radiology.174.2.2296648
- 36) Adamson GD, de Mouzon J, Chambers GM, et al. International committee for monitoring assisted reproductive technology: world report on assisted reproductive technology, 2011. *Fertil Steril*. 2018;110(6):1067-1080. doi:10.1016/j.fertnstert.2018.06.039

Foundation Models Emerge as the “Electricity” of Radiology AI

Claudia Stahl

Note: this is the first in a series of articles based on interviews conducted at RSNA 2025 by Lawrence Tanenbaum, MD, Associate Editor of Applied Radiology’s Artificial Intelligence section. The interview can be viewed at Applied Radiology’s YouTube channel.

Fans of the PBS television series *Downton Abbey*, set in early 20th-century England, witnessed the far-reaching effects of the Second Industrial Revolution—particularly innovations in electricity, mechanization, and communication—on the lives of its characters. What fueled this all-encompassing change, and what does it have in common with artificial intelligence in health care?

During a discussion with Nina Kottler, MD, Chief Medical AI Officer at Mosaic Clinical Technologies, Lawrence Tanenbaum, MD, raised the comparison as a way to describe how foundational technologies perhaps, not so quietly, reshape professional life.

Kottler, whose academic pathway started out in applied mathematics and shifted direction to medicine, said that she “wanted to be a radiologist because on every rotation, we went down into the reading room,” where she was captivated by the collegial interaction among radiologists and their clinical colleagues.

Kottler’s early career in radiology was rooted in data and technology, including work at the then-emerging teleradiology company NightHawk. She became an early adopter of AI in 2016, when the technology first entered radiology. In 2017, she partnered with data scientists to develop an AI model that helped radiologists apply population health guidelines at the point of care, and she has remained deeply engaged in the AI space ever since.

Despite her early interest in the potential of technology to improve workplace efficiency, Kottler acknowledged that the advent of PACS, while necessary and transformative for imaging, had an unintended consequence. “It separated us from all of the other clinicians in the hospital.” Although foundation models won’t bring back the reading room, the technology can “change how we communicate with other physicians and how we do our job,” she said.

AI models in medicine, especially radiology, learn patterns from diverse datasets (eg, millions of images and reports) that can then be adapted to specific tasks such as detection, segmentation, reporting, and workflow support. Unlike earlier, task-specific models built for a single challenge, foundation models, trained on various

types of inputs, are capable of multiple, comprehensive functions across diseases, modalities, and institutions.

In Kottler’s view, today’s AI opportunity is not about improving legacy processes, but about rebuilding workflows using modern technology. “If you were going to create (a radiology workflow solution) from scratch today,” she said, “you would create something cloud-native, AI-native.”

Much of that reinvention centers on reducing the countless inefficiencies that contribute to radiologist burnout. Fragmented data—scanned worksheets, incomplete exams, siloed clinical information—force radiologists to act as manual integrators, and “that’s really silly,” Kottler noted. “It doesn’t mean we don’t need the information, but we need something that helps us understand what that information is.”

At Mosaic, the technology and AI services division of Radiology Partners, that “something” increasingly takes the form of foundational models, including both large language models (LLMs) and vision-language models (VLMs). Kottler explained that AI-driven reporting tools powered by LLMs are already delivering meaningful efficiency gains. “We’re seeing about an 18% increase in efficiency from reporting alone,” she said, with early signs of even greater improvements as VLMs begin assisting with x-ray and head CT interpretation. In some settings, she noted, preliminary results show gains of nearly 30%.

Most AI used in radiology today focuses on narrow, single-purpose tasks, such as pneumothorax detection on a CXR or intracranial hemorrhage triage on a

Dr Kottler is Chief Medical AI Officer at Mosaic Clinical Technologies, a division of Radiology Partners, Austin, TX; Dr Tanenbaum is Associate Editor of *Applied Radiology*’s Artificial Intelligence section and former Chief Technology Officer and Director of Advanced Imaging at Radnet Inc; Claudia Stahl is Managing Editor of *Applied Radiology*.

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noncontrast head CT. These tools provide a binary output, flagging whether the finding is present. Even in combination, narrow AI models generated “about a 1% capacity increase. That didn’t solve our problem,” Kottler said.

Foundation models, by contrast, help address the widening gap between imaging volume growth and radiologist capacity by generating a more holistic output that closely resembles what a radiologist would produce. In the future, both LLMs and VLMs are likely to become ubiquitous, embedded directly into radiology workflow across all imaging exams.

So, why the *Downton Abbey* reference? Tanenbaum likened the shift to foundation models in practice to the arrival of electricity in the homes of the last century

—initially novel, but soon invisible once fully integrated into virtually everything. Kottler agreed. “These foundation models are kind of the electricity of radiology,” she said. “In the next year or two, most people are not going to be dictating their studies. They’re going to ... be using a solution that will be creating a structured report and overseeing them.”

Beyond workflow efficiency, Kottler sees foundation models enabling a more fundamental shift in the role of radiology. She described a move beyond population-based guidelines toward precision radiology, where AI extracts predictive insights from imaging pixels that humans cannot perceive. “There is far more information in the pixels than we’re consuming as radiologists,” she said, pointing to emerging tools that

can predict disease risk years before clinical manifestation.

With these technologies, radiologists could find their profession evolving into one that is centered more on disease prevention than detection, in partnership with patients. “It’s hard to get patients to change their lifestyle until a disease has manifested; but once they have a disease, it’s already too late,” said Kottler.

Predictive technology gives radiologists a tool with which to engage directly with patients about their future health. “If we can show them information that helps them understand their trajectory, maybe we can get them to alter their lifestyle and be more involved in their personal care to prevent diseases from developing in the first place.”

BI-RADS v2025: A Modern Framework for a Data-Driven Era

Kerri Reeves

Kerri Reeves is a freelance writer based in Ambler, Pennsylvania.

A lot has changed in breast imaging since the American College of Radiology (ACR) released its 5th edition of the Breast Imaging Reporting and Data System (BI-RADS®) Atlas in 2013. Digital breast tomosynthesis is now routine. Contrast-enhanced mammography and advanced breast MRI techniques, including evolving diffusion-weighted imaging protocols, have expanded diagnostic capabilities.

Artificial intelligence (AI) applications are increasingly embedded in workflows, while multimodality treatments have become the norm and expectations for metrics reporting have significantly increased.

Given this progression, much excitement accompanies December's BI-RADS v2025 manual release, updated to meet the complex needs of today's practice.

"This is really a big deal for the breast imaging community," says Dana H. Smetherman, MD, MPH, MBA, Chief Executive Officer of the ACR. While remaining true to the foundational principles of BI-RADS, the new edition incorporates "lots of new descriptors, including new modalities, new auditing and outcomes," she notes.

The most comprehensive update to BI-RADS in over a decade, the atlas-turned-manual—900 pages complete with deeper guidance, illustrations, and structured terminology to help address day-to-day practice demands—has modernized breast imaging reporting standards. The guide's transition from print to digital format also reflects a progressive approach to managing the continuously evolving field.

According to Melissa B. Reichman, MD, Associate Professor of Clinical Radiology at Weill Cornell

Medicine in New York City, "the BI-RADS 6th edition represents a major step forward for the breast imaging community, incorporating more than a decade of robust, evidence-based research."

The expanded lexicon reflects contemporary imaging techniques, while reducing ambiguity and improving communication across practices, notes Dr Reichman. Of equal importance, Reichman emphasizes, is the enhanced focus on performance metrics and targeted audits—particularly for BI-RADS 3 assessments, extent-of-disease evaluation, and the addition of method of detection. Together, these refinements represent a meaningful step forward in "improving diagnostic quality, radiologist performance, and patient-centered care."

Clinical Clarity and Quality Measurement

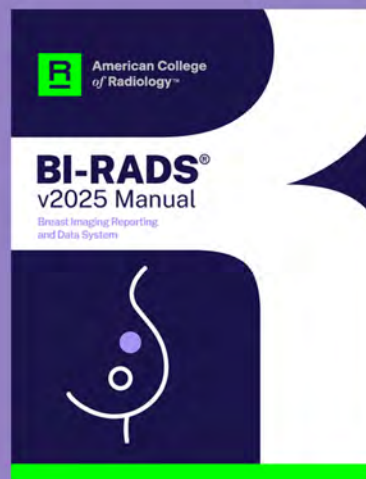
The BI-RADS manual encompasses currently used methods of detection as well as clinically relevant terminology that help accomplish clear, consistent communication. The updates closely align with the ACR National Mammography Database, giving practices an "easy and very standardized way that they look at the outcomes of the work that they are doing," which, in turn, impacts daily reporting, Dr Smetherman says.

New auditing guidance and outcomes measures are designed to support accountability and longitudinal benchmarking. For example, the manual expands the use of BI-RADS Category 6 to better account for patients who may have completed neoadjuvant chemotherapy, aligning reporting with today's common care pathways. It "provides clearer direction for reporting additional ipsilateral

“... the BI-RADS 6th edition represents a major step forward for the breast imaging community, incorporating more than a decade of robust, evidence-based research.”

— Melissa B. Reichman, MD

Image/courtesy the American College of Radiology



findings in patients with biopsy-proven cancer, improving communication with breast surgeons and helping determine when findings will change [disease] management,” explains Dr Reichman.

Dr Smetherman says these improvements are a direct result of the ACR’s stakeholder review efforts. “We have really tried to listen to the feedback of our members of the breast imaging community to make these updates and to incorporate the way they’re practicing breast imaging today.”

A Digital Framework for an Evolving Field

The format of BI-RADS v2025 supports timely updates for continued evolution of imaging technologies and clinical practice.

“In the past, updates meant mailed inserts that you had to physically add to the book,” Dr Smetherman recalls. “Now, those changes can happen much more easily.”

Not only does the digital format enable streamlined updating, it supports data consistency and change implementation amid rapid technological development. The ACR’s broad oversight across the radiology ecosystem—from clinical practice to coding, regulation to technology development—makes this possible, allowing the organization to “be thoughtful about what gets added [to BI-RADS] and when,” Dr Smetherman reports.

For example, use of AI will continue to expand in breast imaging for detection, triage, and risk prediction, among other applications. The format of BI-RADS v2025 facilitates integration of AI applications based on ACR clinical validation processes, not simply in response to market trends.

“Our role [is] to make sure that it is implemented in ways that are safe, effective, and responsible for our patients and for radiologists and their practices,” she says, noting that ongoing technological updates must be anchored in the commitment to reporting and delivery of patient-centered care.

Setting a Global Standard

As new breast imaging technologies become standard of care, the need for consistent, internationally recognized reporting standards becomes even more essential.

“Enthusiasm for the new version [of BI-RADS] is most assuredly not confined to the United States,” Dr Smetherman says, noting there were immediate requests for translated versions, particularly from regions with limited resources. “At a certain level, having that standardization helps everyone.”

As breast imaging continues to advance, consistent terminology and measurable quality standards across borders will continue to ensure safe, harmonized, optimal clinical practice management worldwide.

“Alongside the many exciting and meaningful advancements in breast imaging, grounded in more than a decade of evidence-based medicine, BI-RADS remains a strong and enduring framework. It has withstood the test of time and continues to provide a solid foundation for future progress,” Dr Reichman concludes. “As technology advances and research generates new data, including AI and emerging breast imaging modalities, BI-RADS will continue to evolve, and the breast imaging community will be well prepared to move forward with it.”

Tubo-Ovarian Abscess

Daniel Reyes, BS; Connor Woodward, MD; Raul Galvez-Trevino, MD

Case Summary

A nonsexually active adolescent with a history of constipation, obesity, recurrent urinary tract infection (UTI), and recent treatment of UTI presented with acute, chronic right-sided abdominal pain, low back pain, and fever. On examination, the abdomen was soft and mildly tender to palpation at the periumbilical and right flank and lower quadrant regions. The patient denied sexual activity/sexual abuse. Gonorrhea and chlamydia testing were negative. Leukocytosis was present at 22.3K, and clean catch urine culture demonstrated > 100K *Escherichia coli*. The patient was found on CT and US to have an 8 cm right tubo-ovarian abscess (TOA), from which purulent fluid was aspirated and determined to be culture positive for *E. coli* and *Peptoniphilus asaccharolyticus*.

Imaging Findings

Pelvic CT coronal and sagittal views (Figure 1) demonstrated $6.0 \times 7.3 \times 7.0$ cm rim-enhancing fluid collections in the right lower pelvis, displacing the uterus slightly to the left of midline, with associated right moderate hydronephrosis and associated reactive adenopathy. US pelvis and power Doppler demonstrated 8.1 cm hypervascular right adnexa structure with cystic component.

Diagnosis

Tubo-ovarian abscess.

Depending on the clinical scenario, the differential diagnosis may include uterine or ovarian tumor, ovarian or fallopian tube cyst, malignancy, diverticular or appendiceal abscess or mass, endometrioma, ovarian cyst, hematoma, hemorrhagic cysts, hydrosalpinx, ectopic pregnancy, inflammatory bowel disease, pelvic inflammatory disease (PID), ovarian torsion, and pyelonephritis.^{1,2}

Discussion

TOA is a severe, potentially life-threatening infection of the fallopian tube and ovary, typically affecting sexually active females of reproductive age as a complication of PID. TOA is caused by ascending genital infections, mainly *Chlamydia trachomatis* and *Neisseria*, though *E. coli*, *Bacteroides* species, and *Peptostreptococcus* may also be involved.^{1,3,4} Symptoms classically include acute lower abdominal pain, vaginal discharge, chills, and fever, though these can vary, complicating diagnosis.¹ Conservative treatment with broad-spectrum antibiotics, and possibly drainage if antibiotics fail, is recommended to preserve fertility.^{1,4} Surgery is recommended in cases of large abscesses, hemodynamic instability, antibiotic failure,

or signs of rupture (eg, peritonitis and sepsis).^{1,3,4} Complications can include rupture, sepsis, and rarely, perihepatitis (Fitz-Hugh-Curtis syndrome).^{2,3} Prognosis is generally good with proper treatment, with a mortality rate of 1 in 740 and potential reproductive complications such as infertility and ectopic pregnancy.³

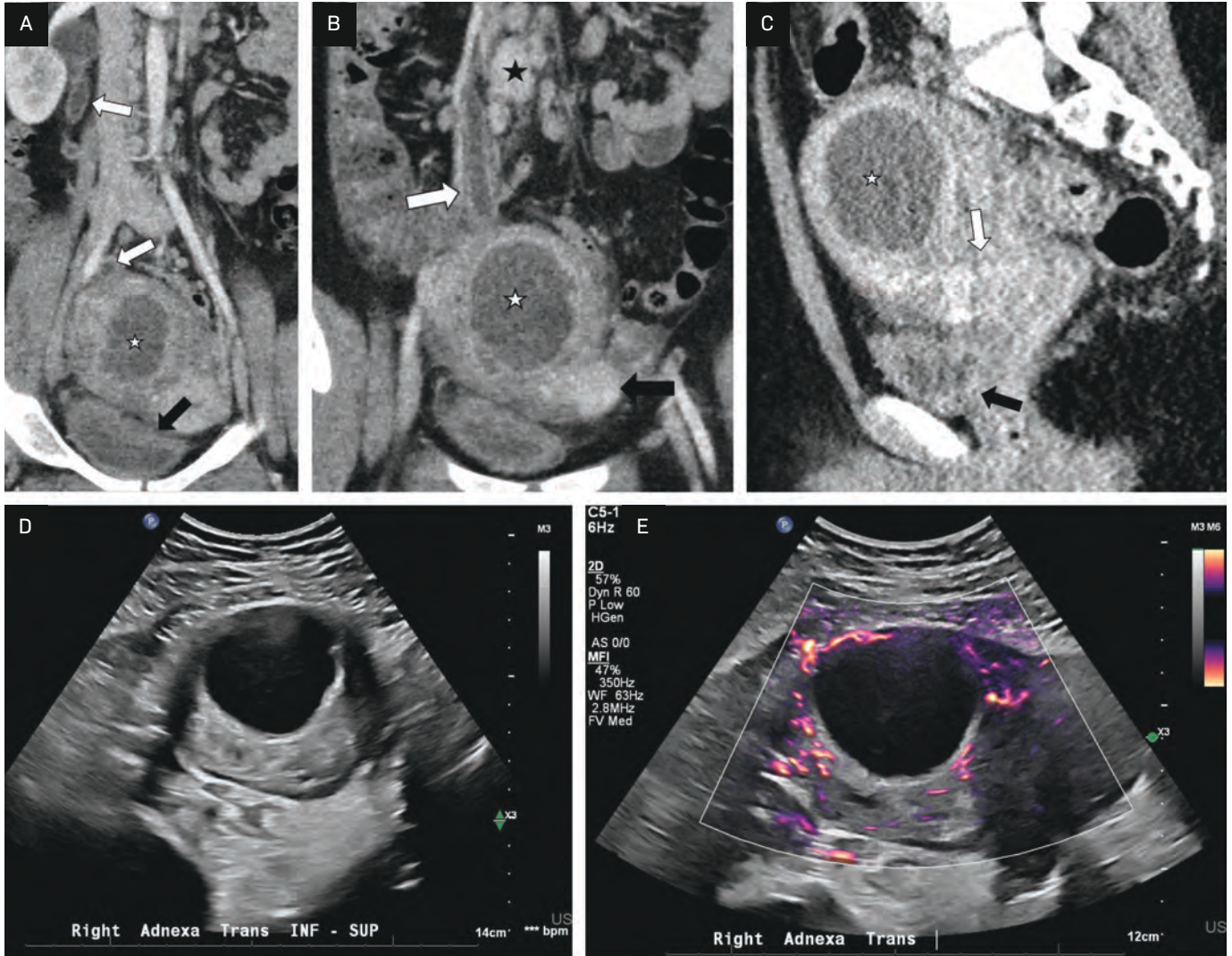
TOA is rare in nonsexually active females.^{1,5} Abdominal pain is the most common symptom, with fever, leukocytosis, nausea, vomiting, diarrhea, and abdominal tenderness also common.^{1,5} The mechanism of spread in such cases may include direct translocation through the intestines, ascension through the urogenital tract; for example, from vaginal urine pooling related to obesity, or hematogenous spread with bacteremia.⁶ The proposed risk factors include recurrent UTIs, obesity, inflammatory bowel disease, prior pelvic surgery, and poor hygiene, all of which can facilitate ascending infection and bacterial seeding from the gastrointestinal tract.^{1,4,5} The organisms found in nonsexually active females include *E. coli*, *Streptococcus milleri*, *Bacteroides uniformis*, coagulase-negative staphylococci, *Streptococcus viridans*, *Peptostreptococcus anaerobius*, *Streptococcus anginosus*, and *Streptococcus constellatus*.^{1,4,5}

Endovaginal and transabdominal US are the preferred initial imaging studies for work-up in a patient with symptoms of TOA (notably, at the authors' institution, vaginal US is not performed in patients who are not sexually active).² US is useful to distinguish between TOA and uncomplicated PID, as the clinical features are similar. US typically demonstrates a solid, cystic, or

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Figure 1. (A) Coronal CT pelvis with contrast demonstrating dilated, thickened ureter (white arrow) obstructed by tubo-ovarian abscess (white star). Note the relationship to the uterus (black star) and bladder (black arrow). (B) Coronal image demonstrating tubo-ovarian abscess (white star) and enlarged perivascular lymph nodes (black star). Also note hydroureter (white arrow) and uterus (black arrow). (C) Contrast-enhanced sagittal CT pelvis demonstrating the relationships of pelvic structures: tubo-ovarian abscess (white star), bladder (black arrow), and uterus (white arrow), which is poorly delineated owing to adjacent inflammatory changes. Ovaries were not distinctly visualized due to adjacent inflammation. (D) Pelvis US and (E) power Doppler US transverse views demonstrating 8.1 cm hypervascular right adnexa structure with cystic component.



complex adnexal or cul-de-sac mass with an adjacent fluid collection.⁷

Additional US findings suggestive of a typical TOA include a multilocular, complex, adnexal mass with debris, septations, and irregular/thick walls/margins, as well as loss of visualized tissue boundaries between pelvic organs, and thick, dilated fallopian tubes.^{5,7} These are commonly bilateral with echogenic debris within the pelvis.^{2,8} The masses may present as indistinct uterine margins, and there may be loss of midline

endometrial echoes.⁷ CT or MRI may be required to distinguish a TOA from ovarian malignancies.^{2,5,7}

CT most commonly demonstrates a pelvic mass with uniformly thick, enhancing abscess walls and internal septations.⁷ The pelvic mass typically has high attenuation and may contain fluid-fluid levels or gas. When present, internal gas helps differentiate TOA from other ovarian cystic masses. There may be adjacent or contained pyosalpinx, visualized as a fluid-filled tubular

multilocular lesion with enhancing, thick walls. Uterosacral ligament thickening is visible when the inflammation extends posteriorly. Other organ involvement may be seen, most commonly rectosigmoid colon and ureter.^{7,9}

MRI may be useful when US is inconclusive or if gas content is difficult to distinguish from bowel gas.¹⁰ On MRI, TOA typically appears as a mass with hypointense abscess contents on T1 images and heterogeneous or hyperintense abscess contents on T2 images.²

Abscess contents may vary in signal intensity depending on viscosity or protein concentration. Occasionally, TOA can contain areas of T1 hyperintensity owing to granulation tissue and hemorrhage products.⁷ Dense pelvic adhesions or fibrosis may create mesh-like strands in the pelvic fat planes that appear T2-hypointense but avidly enhance on postcontrast images.⁷

While no single finding on CT/MRI is pathognomic for TOA, the condition should be suspected in any febrile female with a complex pelvic fluid collection without an alternative source of infection (such as appendicitis).

In summary, while rare, our case of an 11-year-old girl with a history of obesity, recurrent UTIs, and constipation presenting with abdominal pain, tenderness, and fever has similar factors to other nonsexually active pediatric TOA cases reported in the literature; for example, case 2 in Hartmann et al of a 12-year-old girl with obesity, constipation, and recurrent UTIs.⁴ Of note, our case presented with a simpler, cystic appearance than a typical TOA secondary to PID. There does not appear to be a discussion in the literature regarding the differences in appearance on imaging in these cases versus typical TOA secondary to PID.

Conclusion

TOA is most common in reproductive-aged, sexually active females as a complication of PID, but it can also be seen in nonsexually active pediatric patients and should be considered in the differential diagnosis in patients with a concerning clinical presentation and imaging findings, especially in the presence of other risk factors. The proposed risk factors for TOA in nonsexually active females include a history of recurrent UTIs, obesity, inflammatory bowel disease, prior pelvic surgery, and poor hygiene. Modalities such as sonography and CT are useful in arriving at a diagnosis.

Antibiotics are used as the primary treatment, preferably with adequate anaerobic coverage given the large proportion of cases attributable to anaerobic gut flora. Surgical drainage may not always be required but could be used for diagnosis or when conservative measures do not result in improvement.

References

- 1) Bathan L, Mendez YS, Sacks MA, et al. Tubo-ovarian abscess in a virgin pediatric patient. *J Case Rep Images Obstet Gynecol*. 2020;6:100067Z08LB2020. doi:10.5348/100067Z08LB2020CR
- 2) Weerakkody Y, Sharma R, Nassar I, et al. Tubo-ovarian abscess. Reference article, Radiopaedia.org. Accessed January 6, 2025. <https://doi.org/10.53347/rID-9818>
- 3) Kairys N, Roepke C. Tubo-ovarian abscess. In: *StatPearls* [Internet]. StatPearls Publishing. 2025. <https://www.ncbi.nlm.nih.gov/books/NBK448125>
- 4) Hartmann KA, Lerand SJ, Jay MS. Tubo-ovarian abscess in virginal adolescents: exposure of the underlying etiology. *J Pediatr Adolesc Gynecol*. 2009;22(3):e13-e16. doi:10.1016/j.jpag.2008.03.006
- 5) Fei YF, Lawrence AE, McCracken KA. Tubo-ovarian abscess in non-sexually active adolescent girls: a case series and literature review. *J Pediatr Adolesc Gynecol*. 2021;34(3):328-333. doi:10.1016/j.jpag.2020.12.002
- 6) Fink D, Lim PP, Desai A, et al. Recurrent tubo-ovarian abscess in a nonsexually active adolescent. *Consultant*. 2021;62(1):e26-e28. doi:10.25270/con.2021.04.00010
- 7) Kim SH, Kim SH, Yang DM, Kim KA. Unusual causes of tubo-ovarian abscess: CT and MR imaging findings. *Radiographics*. 2004;24(6):1575-1589. doi:10.1148/rg.246045016
- 8) Stanley K, Morato D, Chilstrom M. Ultrasound diagnosis of bilateral tubo-ovarian abscesses in the emergency department. *West J Emerg Med*. 2013;14(6):641-642. doi:10.5811/westjem.2013.7.18487
- 9) Hiller N, Sella T, Lev-Sagi A, Fields S, Lieberman S. Computed tomographic features of tuboovarian abscess. *J Reprod Med*. 2005;50(3):203-208.
- 10) Telischak NA, Yeh BM, Joe BN, et al. MRI of adnexal masses in pregnancy. *AJR Am J Roentgenol*. 2008;191(2):364-370. doi:10.2214/AJR.07.3509

Hepatic Hydatid Disease

Alexander Vold, MD; Emily Alden; William Thompson, MD

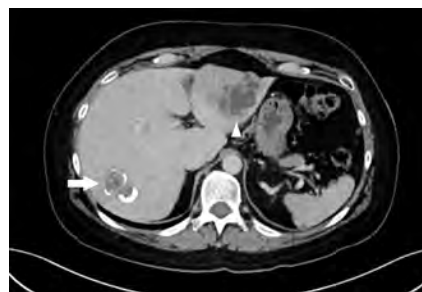
Case Summary

A previously healthy, middle-aged patient presented to the emergency department for chronic, intermittent worsening abdominal pain in the right upper quadrant with associated intermittent nausea, vomiting, diarrhea, and fatigue, without subjective fever. US and CT of the abdomen showed cystic structures within the liver. The patient was prescribed a 2-week course of oral doxycycline and discharged with a plan for outpatient follow-up. The patient returned to the emergency department on day 15 of the antibiotic course, reporting initial improvement but the return of symptoms the day after finishing the antibiotic course. At this time, it was noted that the patient worked as a sheep herder, butcher, and wool weaver. The patient also had a long history of work-related travel, including visits to international slaughterhouses. A CT of the abdomen again showed cystic disease within the liver. A complete blood count with differential showed eosinophilia of 2.0. The patient was started on albendazole (ABZ) and admitted for pain control. Serum *Echinococcus* antibody titer was drawn and later showed elevated titer of 6.14. The patient was discharged with an outpatient appointment for surgical evaluation. Later, the patient received outpatient surgery for resection of hepatic segments 2 and 3, along with elective cholecystectomy. A calcified segment 7 lesion was determined to be inactive without the need for surgical intervention.

Figure 1. Transverse grayscale US image over the epigastrium. The arrow shows a thick-walled multiloculated cystic structure within the parenchyma of the left liver.



Figure 3. Axial contrast-enhanced CT of the abdomen. The arrowhead shows a heterogeneous complex cystic structure in segments 2 and 3 of the liver, representing an active echinococcal cyst containing smaller anterior daughter cysts. The arrow shows a well-margined calcified cystic structure in segment 7 of the liver, representing chronic inactive disease.



Imaging Findings

Abdominal US of the right upper quadrant showed a multiloculated cystic lesion in the left lobe of the liver (Figure 1, arrow). US of the posterior right liver showed

Figure 2. Transverse grayscale US of the right upper abdomen. The arrow shows hyperechoic circular shadowing calcified lesions representing calcified cysts in the posterior right liver.



Figure 4. Frontal radiograph of the chest. The arrow shows a round, calcified lesion over the region of the right liver, corresponding to the calcified hydatid cyst seen on CT.



multiple circular hyperechoic shadowing calcified structures (Figure 2, arrow). Axial contrast-enhanced abdominal CT through the liver showed a multiloculated cystic lesion in the left lobe of the liver (Figure 3, arrowhead), as well as a calcified cystic lesion in the posterior right liver (Figure 4, arrow).

Diagnosis

Hepatic hydatid disease.

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Aside from hydatid disease, the main differential considerations for multifocal cystic hepatic lesions are hepatic abscesses from other etiologies or neoplasms.

Discussion

The tapeworm *Echinococcus granulosus* and its genetic variants are responsible for cystic hydatid disease. Humans are considered accidental intermediate hosts after they acquire infection similarly to other intermediate hosts through contaminated food and water.¹ The majority of human infection involves a dog-sheep-dog cycle, but other domestic animals may also be involved.¹ Most human cases of infection in the United States are diagnosed in immigrants from countries where cystic echinococcosis is endemic, who acquired infection in their country of origin.^{2,3} Primary infections in the United States mainly occur in California, Utah, Arizona, and New Mexico.² Hospital records indicate an estimated incidence of 1-4 cases per year.²

Clinical presentation is usually in the 3rd to 5th decade of life, but primary infection usually occurs in childhood.⁴ Most cysts are asymptomatic and are detected incidentally on imaging obtained for unrelated reasons or at autopsy. When patients are symptomatic, presentation depends on the sites involved, and symptoms are usually related to expanding mass pressure on adjacent structures or infection from rupture of cyst contents into surrounding areas.⁴ Cyst rupture into the hollow abdominal organs is rare, even in endemic areas, and can result in fever and nonspecific symptoms, such as abdominal pain.⁵

Imaging findings, along with a concurrent history of potential exposure, can be key to diagnosis. On US, cysts are typically well visualized, with primary cysts appearing as well-defined margins containing fluid, and daughter cysts appearing as multivesicular cystic structures.⁴ CT can reveal the cystic structures as well as the location of cysts that are not easily seen with US.⁴ Differential diagnosis of cystic hepatic lesions is broad, including benign and malignant etiologies such as simple hepatic cysts, polycystic liver disease, hepatic abscess, primary or metastatic neoplasms, biloma, and hepatic hydatid cyst, among others.⁶ Diagnosis with aspiration or biopsy is rarely performed due to the risk of anaphylaxis with cyst rupture.⁴ Diagnosis is further supported by serological detection of serum antibodies or detection of specific antigens.⁴

Cystic echinococcosis disease is treated using observation, benzimidazoles, drainage, and/or surgery depending on the patient's symptoms, the stage, size, and location of the cyst, and the risk of complications.⁵ Medical treatments are mainly limited to benzimidazole-carbamate compounds, which include ABZ and mebendazole.⁷ Prognosis is variable and dependent on each cystic lesion. Follow-up repeat imaging with CT or MRI, or with serologies, should be considered as local recurrence has been reported up to 10 years after apparently successful treatment.⁸

Conclusion

Hydatid disease is caused by the tapeworms of the genus *Echinococcus*, with typical clinical presentation in the 3rd to 5th decade of life, although primary infection usually occurs in childhood. Classic imaging features on US and CT

show the characteristic primary cystic structure containing multilocular daughter cysts. Calcification of the cysts can occur with long-standing chronic infection. If the classic daughter cysts are not seen or if there are alternative diagnoses, such as pyogenic abscess or neoplasm, diagnosis can be supported by appropriate history, including exposure to livestock and laboratory analysis showing eosinophilia and positive serum antibody titer.

References

- 1) World Health Organization. *Echinococcosis*; 2021. <https://www.who.int/news-room/fact-sheets/detail/echinococcosis>
- 2) Moro P, Schantz PM. Cystic echinococcosis in the Americas. *Parasitol Int*. 2006;55 Suppl:S181-S186. doi:10.1016/j.parint.2005.11.048
- 3) Centers for Disease Control and Prevention. *Parasites-Echinococcosis biology*; 2019. https://wwwnc.cdc.gov/eid/article/29/5/22-1828_article/
- 4) Khuroo MS. Hydatid disease: current status and recent advances. *Ann Saudi Med*. 2002;22(1-2):56-64. doi:10.5144/0256-4947.2002.56
- 5) Stojković M, Weber TF, Junghanss T. Clinical management of cystic echinococcosis: state of the art and perspectives. *Curr Opin Infect Dis*. 2018;31(5):383-392. doi:10.1097/QCO.0000000000000485
- 6) Knipe H. *Cystic lesions of the liver (differential)*. Radiopaedia; 2020. <https://radiopaedia.org/articles/cystic-lesions-of-the-liver-differential?lang=us>
- 7) Siles-Lucas M, Casulli A, Cirilli R, Carmena D. Progress in the pharmacological treatment of human cystic and alveolar echinococcosis: compounds and therapeutic targets. *PLOS Negl Trop Dis*. 2018;12(4):e0006422. doi:10.1371/journal.pntd.0006422
- 8) Rinaldi F, Brunetti E, Neumayr A, et al. Cystic echinococcosis of the liver: a primer for hepatologists. *World J Hepatol*. 2014;6(5):293-305. doi:10.4254/wjh.v6.i5.293

Erdheim-Chester Disease

Gary G. Ghahremani, MD, FACR; Stephen M. Dorros, MD; David S. Karow, MD, PhD

Case Summary

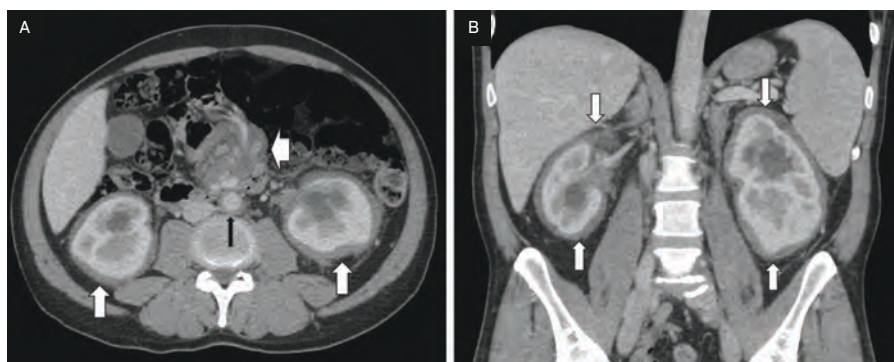
A middle-aged patient with diabetes presented with complaints of general malaise, dull abdominal pain, and weight loss of 12 pounds during the prior 3 months. Physical examination revealed periumbilical tenderness without a palpable mass, mild splenomegaly, and bilateral exophthalmos. Laboratory tests showed unremarkable hematologic and metabolic data, but abnormal renal function with an elevated creatinine level. The patient was referred to our institution for contrast-enhanced CT of the abdomen and MRI of the brain and orbits.

Imaging Findings

Contrast-enhanced CT of the abdomen demonstrated a thick rim of infiltrated subcapsular fat surrounding both kidneys. An ill-defined, 5 cm soft-tissue mass was present at the root of the small bowel mesentery, encasing the visceral vessels, extending into the retroperitoneal area, and surrounding the aorta, renal hilus, and proximal ureters, causing moderate hydronephrosis (Figure 1).

Contrast-enhanced MRI of the orbits demonstrated bilateral, cone-shaped, soft-tissue masses in the retrobulbar regions, resulting in a mild degree of exophthalmos. These lesions were

Figure 1. Abdominal CT features of Erdheim-Chester disease. (A, B) Axial and coronal sections of contrast-enhanced CT demonstrating a 6-8-mm-thick rim of infiltrated perinephric fat around both kidneys (arrows). Delayed nephrogram and hydronephrosis are present. An ill-defined 5 cm mesenteric mass encases the visceral vessels (arrowhead), and a circumferential periaortic infiltration is present (black arrow).



hypointense and homogeneous on T1 and T2 sequences but appeared hyperintense on T1 with fat suppression (Figure 2).

A biopsy of these masses revealed dense clusters of lipid-laden and foamy histiocytes with non-Langerhans features, Touton giant cells with multiple nuclei, and reactive inflammatory fibrosis. Genetic analysis disclosed BRAF-V600E mutation on chromosome 7, which controls cell growth and division in this particular entity. Further confirmation was provided by the characteristic osteosclerosis seen on the patient's knee radiographs (Figure 3).

Diagnosis

Erdheim-Chester disease.

The differential diagnosis includes Langerhans cell histiocytosis, Rosai-Dorfman disease, lymphoma, orbital pseudotumors, and neurosarcoidosis.

Discussion

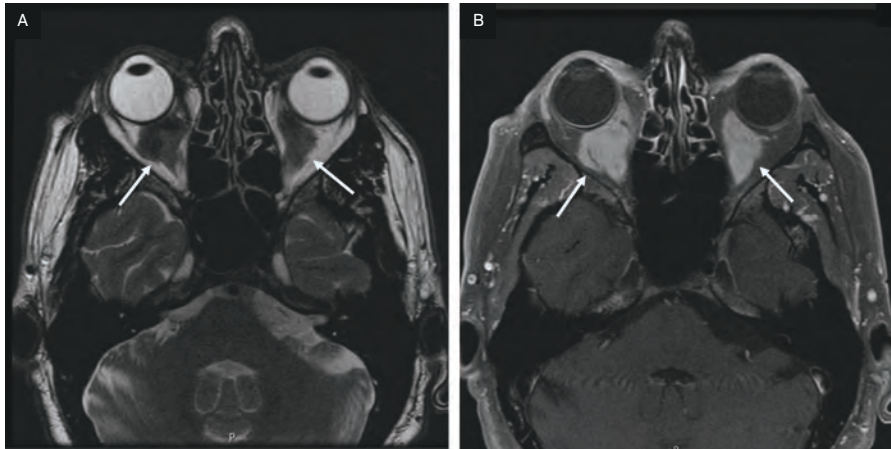
Erdheim-Chester disease is a rare neoplastic condition caused by genetic mutation of histiocytes that are normally found in the bone marrow, blood, liver, and spleen as an important part of the immune system. Their malignant transformation leads to infiltration of various organs by lipid-laden and foamy histiocytes and giant macrophages that incite an inflammatory reaction and fibrosis.^{1,2}

Since the disease's initial description as lipoid granulomatosis in 1930, about a thousand isolated cases and small series of patients with Erdheim-Chester disease have been reported. It develops predominantly in men (3:1 male/female ratio) during the 5th to 7th decade.¹⁻⁴

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Figure 2. Contrast-enhanced MRI of the orbits. (A) Axial T2 sequence demonstrating hypointense bilateral retrobulbar masses (arrows). (B) Axial T1 with fat suppression revealing hyperintense masses (arrows) measuring about 16 × 22 mm and causing moderate exophthalmos.



About 68% of patients present with renal and retroperitoneal involvement. The characteristic finding is the bilateral infiltration of perinephric fat that is called the “hairy kidney sign.” The concomitant pathological process in the peri-pelvic area and around the proximal ureters can cause hydronephrosis, encase the abdominal aorta as the “Coated aorta sign,” and simulate the appearance of retroperitoneal fibrosis (Figure 1). However, that condition can be differentiated from retroperitoneal xanthogranulomatosis in Erdheim-Chester disease, which has perinephric infiltration and other coexisting skeletal and orbital lesions. Occasional involvement of other abdominal organs such as the mesentery, adrenal glands, liver, and pancreas may also occur.³⁻⁶

About 50% of the reported cases had presented with symptoms of brain or eye involvement. This is often manifested as bilateral exophthalmos owing to histiocytic mass formation in the retrobulbar spaces (Figure 2). These lesions surround the optic nerves but do not invade ocular muscles or lacrimal glands. Their distinct MRI features as hypointense on T1 and T2 sequences and hyperintense on contrast-enhanced T1 with fat suppression will help in differentiating these entities from inflammatory orbital pseudotumors.⁷⁻⁹

In addition to retrobulbar masses, some patients develop brain lesions, and their various patterns on CT and MRI have been reported. It is also noted that brain stem involvement results in ataxia, while pituitary gland infiltration leads to diabetes insipidus.^{7,8}

The most common presenting complaint—in 96% of reported cases—has been bone and joint pain.^{3,4,10} This is caused by characteristic osteosclerotic changes best seen in the diaphyseal-metaphyseal area of the long bones, particularly in the femoral condyle and proximal tibia. About 8-10% of the cases also show some scattered lytic lesions caused by xanthogranulomas in the bone marrow.^{3,10} These findings were demonstrated on the subsequently obtained knee radiographs of this patient (Figure 3).

Treatment of Erdheim-Chester disease consists of targeted immunotherapy with BRAF mutation inhibitors, interferon-alpha, and corticosteroids.¹⁻³ Until 2 decades ago, most patients had a relatively poor prognosis, owing to renal failure or cardiopulmonary complications, but the new treatments have increased overall 5-year survival from 68% to 83%.^{1,5} Our patient died from congestive heart failure and pulmonary edema 4 years after presenting to our institution.

Figure 3. Anteroposterior radiographs of both knees demonstrating bilateral osteosclerosis in the metaphyseal regions of proximal tibias (arrows) and the femoral condyles. Multiple osteolytic foci are also visible.



Conclusion

Diagnosis of Erdheim-Chester disease depends upon the histology and genetic analysis of biopsied tissue. However, radiological imaging studies play a crucial role in demonstrating various sites of involvement and their subsequent monitoring. Hence, radiologists should be familiar with the spectrum of presented features of this entity.

References

- 1) Goyal G, Heaney ML, Collin M, et al. Erdheim-Chester disease: consensus recommendations for evaluation, diagnosis, and treatment in the molecular era. *Blood*. 2020;135(22):1929-1945. doi:10.1182/blood.2019003507
- 2) Mazor RD, Manevich-Mazor M, Shoenfeld Y. Erdheim-Chester disease: a comprehensive review of the literature. *Orphanet J Rare Dis*. 2013;8:137. doi:10.1186/1750-1172-8-137
- 3) Veyssier-Belot C, Cacoub P, Caparros-Lefebvre D, et al. Erdheim-Chester disease. Clinical and radiologic characteristics of 59 cases. *Medicine*. 1996;75(3):157-169. doi:10.1097/00005792-199605000-00005
- 4) Estrada-Veras JI, O'Brien KJ, Boyd LC, et al. The clinical spectrum of Erdheim-Chester disease: an observational cohort study. *Blood Adv*. 2017;1(6):357-366. doi:10.1182/bloodadvances.2016001784
- 5) Aswani Y, Patel A, Zhan X, et al. Imaging in Erdheim-Chester disease. *Radiographics*. 2024;44(9):1-19. doi:10.1148/rg.240011

- 6) Nikpanah M, Kim L, Mirmomen SM, et al. Abdominal involvement in Erdheim-Chester Disease (ECD): MRI and CT imaging findings and their association with BRAF^{V600E} mutation. *Eur Radiol*. 2018;28(9):3751-3759. doi:10.1007/s00330-018-5326-1
- 7) Drier A, Haroche J, Savatovsky J, et al. Cerebral, facial, and orbital involvement in Erdheim-Chester disease: CT and MR imaging findings. *Radiology*. 2010;255(2):586-594. doi:10.1148/radiol.10090320
- 8) Sedrak P, Ketonen L, Hou P, et al. Erdheim-Chester disease of the central nervous system: new manifestations of a rare disease. *AJNR Am J Neuroradiol*. 2011;32(11):2126-2131. doi:10.3174/ajnr.A2707
- 9) Fang Y, Shen B, Dai Q, et al. Orbital inflammatory pseudotumor: new advances in diagnosis, pathogenesis, and treatment. *Eur J Med Res*. 2023;28(1):395. doi:10.1186/s40001-023-01330.0
- 10) Dion E, Graef C, Miquel A, et al. Bone involvement in Erdheim-Chester disease: imaging findings including periostitis and partial epiphyseal involvement. *Radiology*. 2006;238(2):632-639. doi:10.1148/radiol.2382041525

Intra-Abdominal Rupture of a Uterine Leiomyoma

Kaitlin M. Zaki-Metias, MD; Tess Vindischman, DO; Lisa Deptula, MMS; Serguei Medvedev, MD; Yanruo Lu, MD; Leslie S. Allen, MD

Case Summary

A middle-aged G1P1 female presented to the emergency department with a 2-week history of worsening intermittent abdominal pain and constipation. Pelvic US showed an enlarged uterus with calcified fibroids. Cross-sectional imaging was performed and revealed findings suspicious for superinfection with a necrotic fibroid. Further evaluation with MRI and gynecology consultation was recommended; however, the patient declined administration of intravenous gadolinium-based contrast. She was given antibiotics for a potentially infected necrotic fibroid and was discharged with an outpatient referral to gynecology.

She returned to the emergency department one month after her initial presentation with severe, 10/10 pelvic pain, nausea, vomiting, and diaphoresis. A CT of the abdomen and pelvis with intravenous contrast performed at this time noted the necrotic fibroid had ruptured, resulting in complex ascites. The patient was taken to the operating room for a hysterectomy.

Imaging Findings

Abdominopelvic CT with intravenous contrast performed upon initial presentation showed a lobular enlarged uterus with a centrally necrotic, partially calcified intramural uterine fibroid at the uterine fundus, which contained a small locule of gas along the anterior myometrial mantle (Figure 1). A subsequent MRI of the pelvis was performed without intravenous contrast and demonstrated similar findings (Figure 2).

Upon re-presentation to the emergency department approximately one month later, abdominopelvic CT with intravenous contrast showed a small to moderate volume of complex free fluid in the abdomen and pelvis, suspicious for hemoperitoneum (Figure 3). This was suspected to be secondary to a perforated uterine fibroid, given a significant decrease in size and slight change in morphology of the known large necrotic uterine fibroid. Further CT angiographic imaging was performed and noted moderate volume hemoperitoneum without active extravasation.

Diagnosis

Intra-abdominal rupture of a uterine fibroid. The differential diagnosis included pyomyoma, pyometra, uterine leiomyosarcoma, and uterine sarcoma.

Discussion

Uterine fibroids, or leiomyomas, are the most common benign uterine neoplasm in women and are primarily composed of smooth muscle and fibrous connective tissue. Leiomyomas can be classified as submucosal, intramural, or subserosal depending on location.¹ Most leiomyomas are asymptomatic but may present with abnormal uterine bleeding, pain from pressure on abdominal organs, or a palpable abdominal mass.¹ Although benign, there are several complications of fibroids that may necessitate intervention. Intra-abdominal hemorrhage due to a ruptured uterine fibroid is extremely rare. Only 125 cases were identified in a 2020 systematic review of the literature for the past century.² An intraperitoneal hemorrhage from a fibroid is typically secondary to rupture of superficial veins due to an increase in abdominal pressure.³ The resulting bleeding can less frequently be arterial in nature or related to hypertension.³ Other etiologies for intra-abdominal hemorrhage from a fibroid include avulsion due to trauma, torsion of a pedunculated fibroid, and vessel rupture due to venous congestion.

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Figure 1. Sagittal (A) and axial (B) CT of the abdomen and pelvis after the administration of intravenous contrast shows an enlarged uterus with a centrally necrotic and partially calcified myometrial mass (straight arrow). There is a single locule of air within the necrotic portion of the fibroid anteriorly (curved arrow).

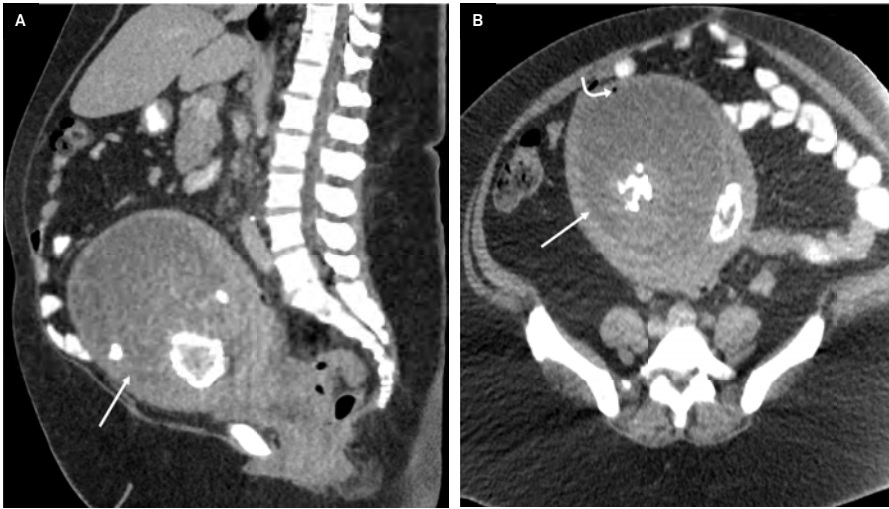
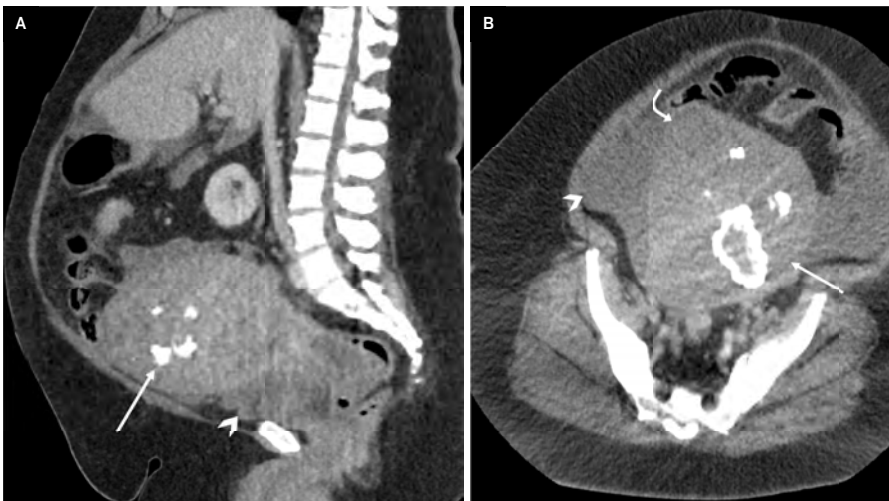


Figure 3. Sagittal (A) and axial (B) CT of the abdomen and pelvis after the administration of intravenous contrast, obtained one month after initial presentation, shows a significant decrease in size of the cystic component of the previously visualized large necrotic myometrial mass (straight arrow) with a small to moderate volume complex free fluid (arrowhead). Note the abnormal contour (curved arrow) along the right uterine fundus, suspicious for perforation.

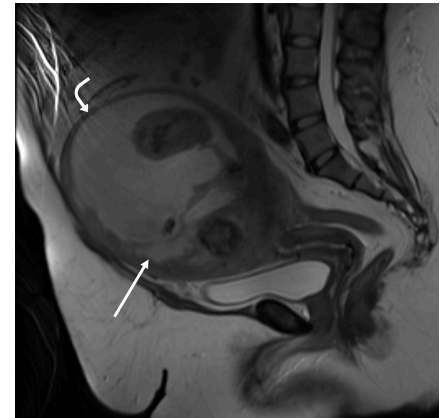


tion in menstruation, pregnancy, or strenuous exercise.^{3,4}

The spontaneous rupture of a fibroid is exceedingly rare, with only ten cases reported in the last half-decade.³ Cases of spontaneous perforation most commonly involve a pathologic provoking factor, such as necrosis or infection, as in our

case. Other possible provoking factors include post-dilation and curettage and increased intra-abdominal pressure.⁵ The bisected gross uterine specimen in our case exhibited uterine fibroids with areas of degeneration and necrosis. Microscopic examination of the surgical specimen from our patient demonstrated hyaline

Figure 2. Sagittal T2-weighted MRI of the pelvis without intravenous contrast shows an enlarged uterus with a large, heterogeneous necrotic myometrial mass with cystic, solid, and calcified components (straight arrow). The small locule of gas visualized on the CT was not visualized at the time of MRI. There is marked myometrial thinning along the anterior and superior margins of the necrotic mass (curved arrow).



degeneration and ischemic necrosis of the leiomyoma. Hyaline degeneration occurs when smooth muscle is replaced by fibrous connective tissue, resulting in decreased stromal support and tissue integrity.⁵ This compromised tissue integrity, along with the noted necrotic tissue, could have precipitated a rupture from within the mass. The pathogenesis resulting in the degeneration of fibroids is multifactorial. An enlarging leiomyoma may outgrow its blood supply, resulting in several types of degeneration, including hyaline, calcified, cystic, and red.¹ The pathogenesis may also involve steroid hormones.³ Fibroids are responsive to estrogen and progesterone, with symptomatic fibroids being most common in perimenopausal women aged 40 or older.⁶ Menstrual cyclicity is another important factor affecting the maintenance and growth of fibroids.³ As our patient was perimenopausal, the pathological changes seen in her fibroid might have been related to decreasing levels of estrogen and progesterone and associated with menstrual cycle irregularity. These hormonal changes could have affected the vascular blood

supply to her large fibroid, leading to degeneration and necrosis.

US, CT scans, and MRI have been used for the evaluation of ruptured leiomyomas.³ In our case, the CT scan was sufficient to identify the etiology of the small-volume hemoperitoneum, given the absence of the necrotic fibroid visualized on the previous CT. Pelvic US is safe and readily accessible, but the acuity of presentation led to prioritizing CT for a more definitive diagnosis in this scenario. One limitation of the US is the inability to assess for active bleeding and limitations in evaluating adjacent structures. CT angiography was performed to exclude active bleeding in our patient due to the presence of complex ascites. MRI can further delineate the nature of the diagnosis, but it is not appropriate in emergent cases due to acquisition time.⁴ Only 2 of the 125 patients identified by Lim et al. had an MRI performed.²

Conclusion

Spontaneous rupture of uterine fibroids resulting in hemoperitoneum is a rare complication, as most fibroids will necrose and extrude into the endometrial cavity, rather than rupture into the abdomen. Although acute intra-abdominal bleeding from a ruptured fibroid is rare, it should be included on the differential in any woman with acute hemoperitoneum and a history of fibroids. Prompt surgical intervention based on accurate and timely diagnosis can be life-saving in such circumstances.

References

- 1) Murase E, Siegelman ES, Outwater EK, Perez-Jaffe LA, Tureck RW. Uterine leiomyomas: histopathologic features, MR imaging findings, differential diagnosis, and treatment. *Radiographics*. 1999;19(5):1179-1197. doi:10.1148/radiographics.19.5.g99se131179
- 2) Lim WH, Cohen SC, Lamaro VP. Intra-abdominal haemorrhage from uterine fibroids: a systematic review of the literature. *BMC Surg*. 2020;20(1):70. doi:10.1186/s12893-020-00736-5
- 3) Schwartz M, Powell K. Spontaneous rupture of a leiomyoma causing life-threatening intra-abdominal hemorrhage. *Case Rep Obstet Gynecol*. 2017;2017:3701450. doi:10.1155/2017/3701450
- 4) Hu H, Choi JDW, Arrage N. Unusual presentation of hemoperitoneum secondary to ruptured uterine leiomyoma: a case report. *Gynecol Pelvic Med*. 2022;5:19-19. doi:10.21037/gpm-21-54
- 5) Shan A, Harjee R, McLaughlin P, Jones EC, Bedaiwy MA. Spontaneous rupture of leiomyoma diagnosed preoperatively with noncontrast computed tomography. *Case Rep Obstet Gynecol*. 2020;2020:5364165. doi:10.1155/2020/5364165
- 6) Choi JH, Liu HJ, Heo SM, Kim SA. Hemoperitoneum caused by spontaneous rupture of uterine leiomyoma in a perimenopausal woman. *J Menopausal Med*. 2021;27(1):42-45. doi:10.6118/jmm.20037

Misdiagnosed High-Grade Undifferentiated Sarcoma Mimicking Infantile Myofibroma: A Case Report

Soham Bhanvadia, BS; Mary Jane Risch, BS; Luis Goncalves, MD; Sudhen Desai, MD

Abstract

Accurate prenatal diagnosis of fetal masses is challenging due to their varied presentation and the limitations of current imaging modalities, which are typically restricted to obstetric US and non-contrast fetal MRI. We present a case involving a right forearm mass first identified on prenatal US and MRI, which initially appeared benign and was diagnosed as infantile myofibroma following excisional biopsy. However, due to rapid growth and persistent symptoms, the patient ultimately underwent transhumeral amputation, with final pathology revealing a high-grade undifferentiated sarcoma. This case highlights the diagnostic complexity of fetal soft tissue tumors and underscores the importance of maintaining a broad differential, including malignancy, even when imaging and initial pathology suggest a benign process.

Keywords: prenatal imaging, fetal tumor, infantile myofibroma, undifferentiated sarcoma, fetal MRI, diagnostic error, pediatric oncology, prenatal diagnosis, biopsy misdiagnosis, soft tissue tumor

Case Summary

A pregnant adult patient was referred at 20 weeks' gestation following abnormal anatomy US that revealed an enlarged right arm with clenched fingers. Fetal MRI and US confirmed right forearm enlargement, thickening of the radius and ulna, and dystrophic soft tissue calcifications. The remainder of the exam was unremarkable. At birth via cesarean section, a firm, nodular mass was noted on the right forearm. MRI 4 days

after delivery revealed bone remodeling and a hyperintense, heterogeneous soft tissue mass with calcifications and patchy enhancement. An excisional biopsy performed at 6 weeks yielded a diagnosis of infantile myofibroma, and the patient was observed over the next few months. By 4 months, the mass had enlarged and was causing pain and loss of function, necessitating transhumeral amputation. Final pathology demonstrated high-grade undifferentiated sarcoma. At 5 months, the patient developed a recurrence of the mass

requiring shoulder disarticulation and is currently undergoing chemotherapy.

Imaging Findings

Fetal MRI (Figure 1) and US (Figure 2) at 22 weeks' gestation showed enlargement of the right forearm with thickened radius and ulna. The mass appeared hyperintense on T2-weighted sequences. Postnatal MRI on day 4 (Figure 3) demonstrated deformity of the forearm bones and a hyperintense, heterogeneously enhancing mass with calcifications on T2-weighted sequences. At 5 months, MRI (Figure 4) revealed a lobulated, enhancing soft tissue mass surrounding the midhumerus without obvious proximal bony involvement.

Diagnosis

High-grade undifferentiated sarcoma.
Differential diagnoses: venous malformation with phleboliths,

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Informed Consent: Patient confidentiality was maintained according to institutional policy. Written informed consent was not obtained but is encouraged in future similar cases.

Figure 1. Sagittal T2-weighted fetal MRI demonstrates enlargement of the right forearm soft tissues with thickening of the radius and ulna.

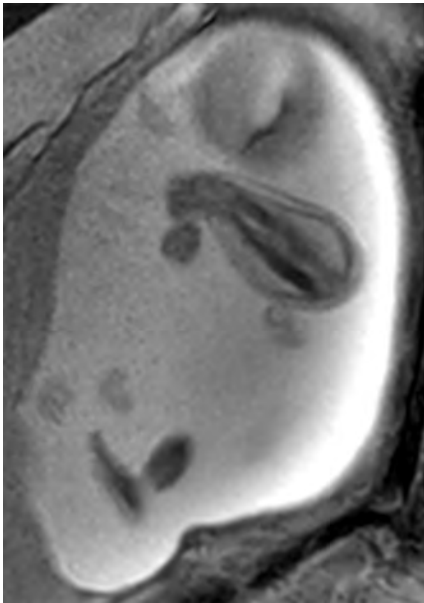


Figure 2. Coronal US confirms soft tissue enlargement of the right forearm and bony enlargement without associated fluid collection.

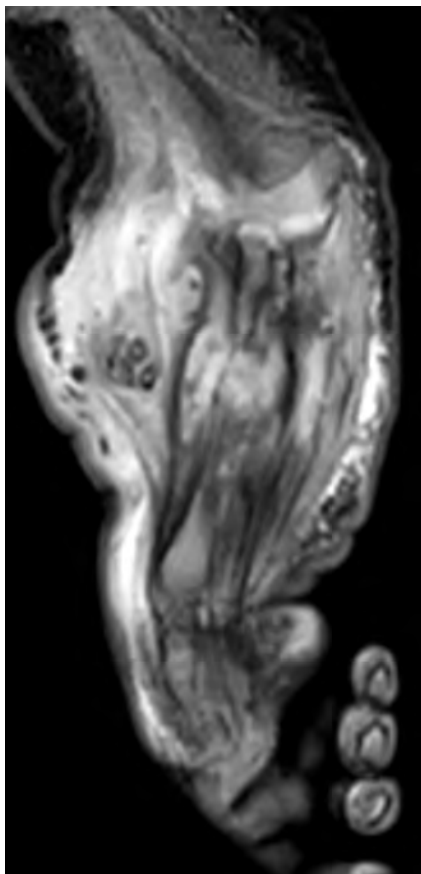


macrodystrophia lipomatosa, constriction ring from amniotic band sequence, kaposiform hemangioendothelioma, myofibroma, fibrosarcoma, rhabdomyosarcoma, and osteogenesis imperfecta type V.

Discussion

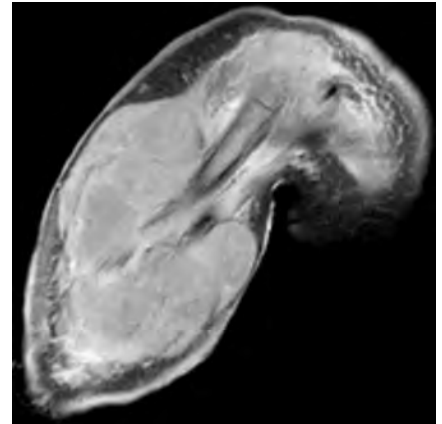
Diagnosis of prenatal masses presents significant challenges due to variability in presentation, limited imaging modalities (US and non-contrast MRI), and differences between fetal and pediatric tumors.¹ Prenatal imaging can guide postnatal management but cannot provide

Figure 3. Sagittal T2-weighted MRI demonstrates further enlargement of the forearm, irregular contouring of the radius and ulna, and dystrophic soft tissue calcifications 4 days after birth.



a definitive diagnosis, as fetal tumors differ from pediatric tumors in their biology, growth kinetics, and prognosis.^{1,2} In this case, prenatal US first identified the abnormality, and fetal MRI was instrumental in further characterizing the lesion, narrowing the differential to entities such as infantile myofibroma, fibrosarcoma, and vascular malformations. Prenatal imaging also guided delivery planning, prompting transfer to a specialized hospital for postnatal MRI and further evaluation of the lesion. Sarcomas, rare in the prenatal period, are malignant connective tissue tumors accounting for about 5% of childhood tumors.³ Undifferentiated sarcoma—a poorly differentiated subtype—is a diagnosis of exclusion and

Figure 4. Coronal T2-weighted MRI demonstrates a lobulated, circumferential, enhancing soft tissue mass surrounding the mid humerus without obvious bony involvement of the proximal humerus at 5 months. Additionally, a fluid collection of the distal arm near the amputation stump is present.



commonly occurs in individuals over 50 and in the lower extremities.⁴ Prenatal and initial postnatal findings suggested a benign mass due to a lack of invasive features, the presence of dystrophic calcifications, and slow growth.^{5,6} Initial biopsy findings were consistent with infantile myofibroma, possibly due to overlapping histopathological features with sarcomas such as hypercellularity and infiltrative growth.^{7,8} Sampling error may also have contributed to the initial misdiagnosis due to the well-documented phenomenon of intratumoral heterogeneity and the dynamic nature of pediatric tissues, which are characterized by ongoing growth and high cellular turnover.^{9,10} Postnatal progression, including rapid growth and pain, suggested malignancy.

Venous malformation with phleboliths may also present with soft tissue calcifications but typically demonstrates well-delineated phleboliths without associated skeletal anomalies, which were not observed in this case.⁶ The absence of additional lesions and the localized nature of the mass also lowered suspicion for multifocal or syndromic pathology. Ultimately, the worsening clinical course—marked

by increasing pain, functional limitation, and mass progression—necessitated transhumeral amputation.

The diagnosis was confirmed after resection and recurrence, illustrating the limitations of both imaging and single-site biopsies in heterogeneous tumors. Early consideration of malignancy could have prompted earlier definitive treatment and potentially reduced the extent of surgical morbidity.

This case highlights the importance of maintaining malignancy in the differential diagnosis for fetal soft tissue masses, even when imaging and initial pathology appear benign. Improved outcomes rely on advanced imaging techniques, such as diffusion-weighted MRI, magnetic resonance spectroscopy, and total-body MRI, alongside careful multidisciplinary evaluation and clinicopathologic correlation, which together enhance lesion characterization, detect multifocal disease, and guide biopsy to the most diagnostically relevant areas.¹¹⁻¹³

Conclusion

This case demonstrates the challenges in diagnosing fetal soft tissue masses and

emphasizes the potential for misdiagnosis, particularly when biopsy and imaging findings appear benign. Maintaining a broad differential and monitoring for clinical progression are crucial. Advances in imaging and multidisciplinary collaboration may improve early and accurate diagnosis.

References

- 1) Kamil D, Tepelmann J, Berg C, et al. Spectrum and outcome of prenatally diagnosed fetal tumors. *Ultrasound Obstet Gynecol*. 2008;31(3):296-302. doi:10.1002/uog.5260
- 2) Cho JY, Lee YH. Fetal tumors: prenatal ultrasonographic findings and clinical characteristics. *Ultrasonography*. 2014;33(4):240-251. doi:10.14366/usg.14019
- 3) Miser JS, Pizzo PA. Soft tissue sarcomas in childhood. *Pediatr Clin North Am*. 1985;32(3):779-800. doi:10.1016/s0031-3955(16)34836-2
- 4) Steele CD, Pillay N. The genomics of undifferentiated sarcoma of soft tissue: progress, challenges and opportunities. *Semin Cancer Biol*. 2020;61:42-55. doi:10.1016/j.semcancer.2019.11.009
- 5) Patel A. Benign vs malignant tumors. *JAMA Oncol*. 2020;6(9):1488. doi:10.1001/jamaoncol.2020.2592
- 6) Freire V, Moser TP, Lepage-Saucier M. Radiological identification and analysis of soft tissue musculoskeletal calcifications. *Insights Imaging*. 2018;9(4):477-492. doi:10.1007/s13244-018-0619-0
- 7) Linos K, Carter JM, Gardner JM, et al. Myofibromas with atypical features: expanding the morphologic spectrum of a benign entity. *Am J Surg Pathol*. 2014;38(12):1649-1654. doi:10.1097/PAS.0000000000000270
- 8) Alaggio R, Bisogno G, Rosato A, Ninfo V, Coffin CM. Undifferentiated sarcoma: does it exist? A clinicopathologic study of 7 pediatric cases and review of literature. *Hum Pathol*. 2009;40(11):1600-1610. doi:10.1016/j.humpath.2009.04.013
- 9) Palmerini E, Picci P, Reichardt P, Downey G. Malignancy in giant cell tumor of bone: a review of the literature. *Technol Cancer Res Treat*. 2019;18:1533033819840000. doi:10.1177/1533033819840000
- 10) Schmelz K, Toedling J, Huska M, et al. Spatial and temporal intratumour heterogeneity has potential consequences for single biopsy-based neuroblastoma treatment decisions. *Nat Commun*. 2021;12(1):6804. doi:10.1038/s41467-021-26870-z
- 11) Manisterski M, Benish M, Levin D, et al. Diverse presentation and tailored treatment of infantile myofibromatosis: a single-center experience. *Pediatr Blood Cancer*. 2021;68(2):e28769. doi:10.1002/pbc.28769
- 12) Masselli G, Vaccaro Notti MR, Zacharzewska-Gondek A, et al. Fetal MRI of CNS abnormalities. *Clin Radiol*. 2020;75(8):640. doi:10.1016/j.crad.2020.03.035
- 13) Rubesova E, Barth RA. Advances in fetal imaging. *Am J Perinatol*. 2014;31(7):567-576. doi:10.1055/s-0034-1371712

Variant of Left-Sided Scimitar Syndrome

Elise Dunning, BS; Randy Richardson, MD

Case Summary

A monochorionic diamniotic twin was born at 34 weeks, 1 day via C-section. On prenatal US, the patient was noted to have a ventricular septal defect (VSD), single umbilical artery, and oligohydramnios.

At birth, the patient's weight was 1720 g and APGARs were 5 and 9 at 1 and 5 minutes. The patient had poor color, shallow breaths, and increased secretions, with initial oxygen saturations in the 50s and a heart rate in the 80s.

Chest x-ray (CXR) showed mild persistent gaseous distention of the upper esophagus with left apical atelectasis and relative hypoaeration of the left lung compared with the right.

Tracheoesophageal fistula (TEF) was identified via echocardiogram on the second day of life, along with the VSD, a large atrial septal defect (ASD), patent ductus arteriosus (PDA), and a hypoplastic left pulmonary artery. Routine CXRs showed persistent left lung hypoplasia.

An initial CT scan of the chest was completed at nearly 2 weeks of life given the abnormal pulmonary artery on echocardiogram.

At 1.5 months of life, an echocardiogram was repeated, identifying an innominate malformation and pulmonary venous obstruction. A repeat CT scan (Figures 1,

2) was obtained at that time given the repeated echocardiogram findings.

During hospitalization, the patient had recurrent right pulmonary vein stenosis, as well as recurrent pulmonary infections requiring several rounds of antibiotics. After multiple surgeries and despite adequate mechanical ventilation, the patient developed severe pulmonary congestion and respiratory failure, succumbing at 7 months of age.

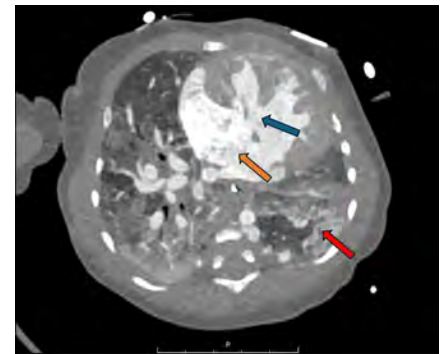
Imaging Findings

Initial chest CT scan with IV contrast at 2 weeks of life demonstrated left lung hypoplasia with diminutive left pulmonary veins, left pulmonary artery, and left mainstem bronchus, as well as tracheomalacia (images not included).

Chest CT scan with IV contrast at 1.5 months of life redemonstrated the left lung hypoplasia (Figures 1, 2), hypoplastic left mainstem bronchus and hypoplastic left pulmonary artery (Figure 3), as well as partial anomalous venous return of the left lung draining into the hemiazygos vein and back to the heart via a left superior vena cava (SVC) and left coronary sinus (Figures 2, 3).

Systemic arterial supply to the left lower lobe of the lung was via an aortopulmonary collateral from the proximal

Figure 1. Contrast-enhanced axial CT maximum intensity projection image of the chest demonstrating severely hypoplastic left lung (red arrow) with a large ventricular septal defect (blue arrow) and atrial septal defect (orange arrow). The pulmonary findings are thought to result from atelectasis in the lung bases, right middle lobe, and right upper lobe, with some right upper lobe air trapping noted.



abdominal aorta (Figures 2, 3). There was also a single right pulmonary vein that drained the superior and inferior right lung. There were associated congenital cardiac defects, including a VSD and ASD (Figure 1), as well as tracheomalacia resulting from compression by the brachiocephalic artery (not shown). Additional features, including a TEF and large PDA, were identified via echocardiogram (images not included).

Diagnosis

Left-sided scimitar syndrome variant.

The differential diagnosis includes scimitar syndrome and extralobar pulmonary sequestration resulting from other causes such as congenital

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Figure 2. (A) Contrast-enhanced oblique sagittal CT maximum intensity projection (MIP) image of the chest demonstrating a left-sided superior vena cava (SVC, red arrow) draining into an enlarged coronary sinus (blue arrow), which empties into the dilated right atrium (yellow arrow). Also shown is a severely hypoplastic left lung. (B) Contrast-enhanced sagittal chest CT MIP image demonstrating anomalous pulmonary veins draining into the hemiazygos vein (red arrow), which drains into the left-sided SVC (green arrow) and eventually empties into the coronary sinus (blue arrow).

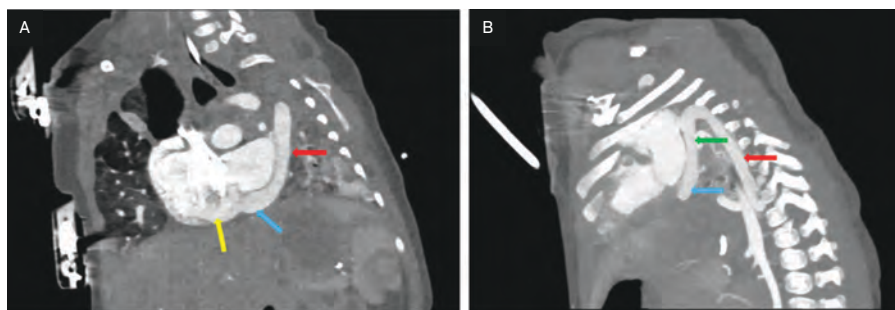
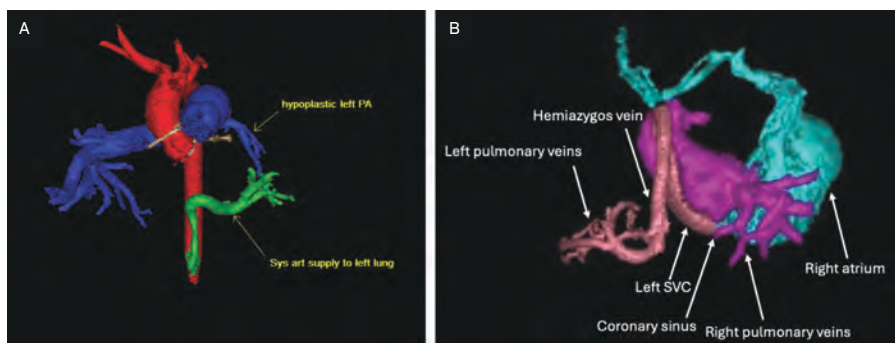


Figure 3. (A) Three-dimensional reconstruction of the patient's left-sided scimitar syndrome, with the diminutive left pulmonary artery shown and the anomalous systemic arterial supply to the left lung arising from the proximal abdominal aorta (red). (B) Three-dimensional reconstruction demonstrating the anomalous drainage of the left pulmonary veins into the hemiazygos vein, the left-sided superior vena cava, the coronary sinus, and ultimately into the dilated right atrium. The normal right-sided pulmonary veins (pink) are included for comparison.



pulmonary airway malformation. Other rare congenital anomalies include lobar atelectasis or unilateral absence of the pulmonary artery. Scimitar syndrome is less likely, given that the scimitar vein does not cross the diaphragm. There were associated cardiovascular defects and recurrent stenosis of the right pulmonary vein confluence.

Discussion

Scimitar syndrome, also known as congenital venolobar syndrome, Halasz syndrome, mirror-image lung syndrome, hypogenetic lung syndrome, or vena cava bronchovascular syndrome, is a

rare congenital anomaly presenting in 1-3/100,000 live births.¹ It consists of partial anomalous venous return to the heart, resulting in a left-to-right shunt.¹ The name “scimitar” derives from the European term describing a broad variety of Middle Eastern curved swords, which the anomalous pulmonary vein can resemble on imaging.¹ The anomaly is classically described on the right side, and its distinct features are as follows¹:

1. Partial or entire anomalous venous drainage of the right lung.
2. Variable degrees of the right lung and pulmonary artery hypoplasia.
3. Dextroposition of the heart.

4. Anomalous systemic blood supply to the ipsilateral lung.¹
5. Anomalies of the right bronchial tree, including sequestration.²

Presentation ranges from asymptomatic to symptoms of cyanosis, respiratory distress, tachypnea, recurrent pneumonia, and heart failure.¹ Those with infantile onset may have demonstrated symptoms of respiratory failure secondary to pulmonary sequestration with recurrent lung infection, as well as features of right ventricular volume overload, pulmonary hypertension, subsequent heart failure, and sometimes pulmonary vein stenosis.

The prognosis for scimitar syndrome presenting in infancy is poor regardless of surgical intervention¹; a recent meta-analysis of scimitar syndrome prognosis and treatment found a mortality rate of 31.8%.¹

Left-sided scimitar syndrome is exceedingly rare, with only 6 cases reported in the literature. In these cases, there were aortopulmonary collaterals to the left lung; left lung and pulmonary artery hypoplasia; and at least one anomalous left pulmonary vein that can drain to various sites.² These cases also reported the left scimitar vein draining into the left hepatic vein immediately after crossing the diaphragm,³ the pericardiophrenic vein,⁴ the azygos vein,⁵ the left hepatic vein close to the IVC,⁶ and passing directly into the IVC.^{7,8}

In our patient's case, the anomalous pulmonary venous system of the left lung drained into the hemiazygos vein, then into the left-sided superior vena cava, which then emptied into the coronary sinus and eventually passed into the right atrium.

Notably, all the previously reported cases had anomalous draining veins that crossed the diaphragm before returning to the heart; thus, this case does not meet the criteria for true left-sided scimitar syndrome. Although the draining vein in this case did not cross the diaphragm, however, it still resulted in the “scimitar” appearance typical of the condition

on imaging. In addition, characteristic findings such as left lung hypoplasia, left pulmonary artery hypoplasia, and anomalous systemic arterial supply to the left lobe were all present in this case, as were congenital cardiac anomalies often associated with infantile scimitar syndrome.^{1,3} Moreover, whereas right scimitar syndrome can be associated with left pulmonary vein stenosis, a rare but well-described phenomenon, our case was associated with recurrent right pulmonary vein stenosis.⁹ Given the findings, our case arguably suggests an interesting variant of scimitar syndrome.

Conclusion

Left-sided scimitar syndrome is an exceptionally rare variant of a classically right-sided condition. The presentation in this case involved another anomaly, in which the draining vein did not cross the

diaphragm, although the classic “scimitar” appearance was still present on imaging. Classic symptoms such as respiratory distress and recurrent pneumonia were present in this case, along with congenital cardiac anomalies and stenosis of the right pulmonary vein confluence. Surgical intervention is the only definitive treatment; however, survival rates remain poor for patients with infantile onset of the condition.

References

- 1) Wang K, Xu X, Liu T, Gao W, Guo Y. Treatment and prognosis of Scimitar syndrome: a retrospective analysis in a single center of East China. *Front Cardiovasc Med*. 2022;9:973796. doi:10.3389/fcvm.2022.973796
- 2) Bo I, Carvalho JS, Cheasty E, Rubens M, Rigby ML. Variants of the scimitar syndrome. *Cardiol Young*. 2016;26(5):941-947. doi:10.1017/S1047951115001651
- 3) Pandey S, Bharath AP, Mohata AP, et al. Left sided scimitar syndrome. *Ann Pediatr Cardiol*. 2023;16(4):303-305. doi:10.4103/apc.apc_73_23
- 4) Mardini MK, Sakati NA, Nyhan WL. Anomalous left pulmonary venous drainage to the inferior vena cava and through the pericardiophrenic vein to the innominate vein: left-sided scimitar syndrome. *Am Heart J*. 1981;101(6):860-863. doi:10.1016/0002-8703(81)90630-x
- 5) Bratincsák A, Rao RP, El-Said HG. Unusual variant of a rare constellation: a left-sided scimitar syndrome with connection to the azygos vein. *Congenit Heart Dis*. 2010;5(2):174-177. doi:10.1111/j.1747-0803.2010.00401.x
- 6) Rutledge JM, Hiatt PW, Wesley Vick G 3rd, Grifka RG. A sword for the left hand: an unusual case of left-sided scimitar syndrome. *Pediatr Cardiol*. 2001;22(4):350-352. doi:10.1007/s002460010245
- 7) Agayev A, Yekeler E. Left-sided scimitar syndrome. *Pediatr Radiol*. 2009;39(2):191. doi:10.1007/s00247-008-1031-6
- 8) Juraszek AL, Cohn H, Van Praagh R, Van Praagh S. Isolated left-sided scimitar vein connecting all left pulmonary veins to the right inferior vena cava. *Pediatr Cardiol*. 2005;26(6):846-847. doi:10.1007/s00246-005-0920-9
- 9) Mendoza A, Herrera D, Caro AT, et al. Scimitar syndrome and left pulmonary vein stenosis: a serious and rare association. *Ann Pediatr Cardiol*. 2022;15(1):80-83. doi:10.4103/apc.apc_53_21

"If something is worth saying, it is worth saying three times over."

—Steven Magee



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Normal STILL Means Normal

C Douglas Phillips, MD, FACR

Sure, Steven, but is it necessary? A few years ago, I got on a tirade about calling things normal; perhaps a few of you remember. Brief refresher: if you call something normal, it makes sense to most air-breathing, upright-walking human beings that there is nothing *bad* there. If you call a study normal, for god's sake, there isn't a tumor, a fracture, a hemorrhage. Right? At least nothing that you saw. I am not dealing with misses here. You looked. You looked twice. And you said it was normal.

Why is this a difficult concept? In a way, this is an already-lost argument. I have long ago knelt to the pressure of including negatives in my reports so that anyone who bothers to read them (and that isn't everyone, believe me) knows that although my impression was *normal*. I also went to the trouble of noting in there many things that go along with *normal*. (*There isn't an acute infarction or hemorrhage or fracture...on, and on, and on.*) So, why revisit this? Oh, you know exactly why. Because the list of things I need to spell out keeps growing. How long, oh lord? How long?

When I was in medical school, a wise attending showed me a technique for the "negative review of systems." At the VA I trained at, the intake form was a very lengthy, paper, system-by-system review of history (obviously way before the advent of tablet

PCs, smart phones, or even a good PC interface to work with). If you hit a system that the patient wasn't complaining of (or many of them if it was truly a negative review), this attending showed me you could take your government issue black pen and just start drawing a line down the page, covering all the boxes. Saved time. It was self-explanatory. Nothing to see here. I now understand it was the visual impact of that straight line that was the deal. If you looked at it, you immediately understood that there wasn't anything there to further concern yourself with.

We need that straight line or something similar. Visual impact. We are radiologists, and radiologists are visual people. So, I had an idea. A thing that would allow someone reading your report to click on an icon if they wanted to further inquire. It would be pre-configured as you saw fit. They might think, "Hey, I wonder if they thought about sarcoidosis?" and they could click the icon and a text box would appear. They could type in, "hey, smarty pants radiologist, did you think about sarcoidosis?" and then a big red field would appear with the words, "YES I DID! AND THIS ISN'T SARCOID! IT'S NORMAL!" would flash up.

Okay, enough of this again. Keep doing that good work. Mahalo.