



Contents lists available at ScienceDirect

Clinical Radiology

journal homepage: www.clinicalradiologyonline.net

Review

Prostatitis: imaging appearances and diagnostic considerations

A. Shakur^{a,*}, K. Hames^b, A. O'Shea^a, M.G. Harisinghani^a^a Department of Radiology, Division of Abdominal Imaging, Massachusetts General Hospital, 55 Fruit Street, Boston, MA, 02114, USA^b Department of Radiology, Hamilton General Hospital, 237 Barton Street E, Hamilton, Ontario, L8L 2X2, Canada

ARTICLE INFORMATION

Article history:

Received 17 July 2020

Accepted 14 January 2021

Acute and chronic inflammation of the prostate gland can be attributed to several underlying aetiologies, including but not limited to, bacterial prostatitis, granulomatous prostatitis, and Immunoglobulin G4-related prostatitis. In this review, we provide an overview of the general imaging appearances of the different types of prostatitis, their distinguishing features and characteristic appearances at cross-sectional imaging. Common imaging pitfalls are presented and illustrated with examples.

© 2021 The Royal College of Radiologists. Published by Elsevier Ltd. All rights reserved.

Introduction

Prostatitis is an inflammatory condition of the prostate with a prevalence of 8.7%,¹ which encompasses several conditions including acute and chronic bacterial prostatitis, granulomatous prostatitis, and Immunoglobulin G4 (IgG4)-related prostatitis.^{2,3} Although recent advances in multiparametric prostate magnetic resonance imaging (MRI) have improved significantly, the diagnostic accuracy of prostatitis and its mimics is limited by the many overlapping radiological features, which can make the differentiation from clinically significant prostate carcinoma challenging. The purpose of this review is to illustrate the multimodality imaging appearances of the various subtypes of prostatitis and when to consider this diagnosis. We also aim to emphasise the key imaging features that can help make a distinction of prostatitis from other conditions, namely prostate carcinoma.

Bacterial prostatitis

Bacterial prostatitis is a bacterial infection of the prostate gland occurring in a bi-modal distribution in younger and older men and comprises 5%–10% of all cases of prostatitis.⁴ It can be acute or chronic and can result in significant morbidity without adequate diagnosis and treatment. Risk factors for bacterial prostatitis include prostate manipulation, urethral stricture, benign prostatic hyperplasia (BPH), phimosis, urethritis, diabetes, and other immunocompromising states. Infection most commonly occurs from intra-prostatic reflux of urine infected with organisms. *Escherichia coli* is the most commonly isolated organism, but other Gram-negative organisms, such as *Klebsiella*, *Proteus*, and *Pseudomonas*, and Gram-positive *Enterococcus* species are often isolated.^{5,6}

Acute bacterial prostatitis often presents with symptoms of urinary irritation (e.g., dysuria, urinary frequency, urgency) and/or urinary obstruction (e.g., hesitancy, poor or

* Guarantor and correspondent: A. Shakur, Department of Radiology, Addenbrooke's Hospital, Hills Rd, Cambridge, CB2 0QQ, UK. Tel.: 07581179735. E-mail address: amreen.shakur@addenbrookes.nhs.uk (A. Shakur).

interrupted stream, straining to void, incomplete emptying). Systemic symptoms (fever, malaise, nausea) may also be present. Inadequately treated, it can lead to overwhelming sepsis, the development of prostatic abscess or chronic bacterial prostatitis.⁷

Chronic bacterial prostatitis is defined as persistent bacterial infection of the prostate lasting >3 months.^{8,9} In contrast to acute bacterial prostatitis, the obstructive/irritative urinary symptoms are less severe and systemic symptoms are often absent.

Imaging features

The challenge of distinguishing bacterial prostatitis from other diseases of the prostate is well documented in the literature. Alternate diagnoses to prostate cancer have been identified in up to 44% of one reported cohort, where patients were interpreted as having prostate cancer on imaging appearances alone.¹⁰ Recent advances including multiple iterations of the Prostate Imaging Reporting and Data System (PI-RADS) reporting tool have focused on helping distinguish prostate cancer from prostatitis.¹¹ The apparent diffusion coefficient (ADC) values have been highlighted as a useful parameter in distinguishing the two; restricted diffusion with ADC values of $>900 \text{ mm}^2/\text{s}$ have

been reported to be a useful indicator for prostatitis; however, there is a degree of overlap in ADC values of prostatitis and prostate cancer. Accurate measurement of ADC values in a clinical setting also remains challenging, thereby limiting its use in routine clinical practice.^{11,12}

On multiparametric MRI, acute bacterial prostatitis is identified most frequently in the peripheral zone and demonstrates focal or diffuse low T2 signal intensity with patchy enhancement. There is mild to moderate diffusion restriction due to the increased inflammatory cellular infiltrates, with associated signal loss on ADC maps (Figs 1–3). Morphological characteristics that can guide the diagnosis of prostatitis include a diffuse, band-like or wedge-like shape in comparison to the more commonly rounded, oval or irregular appearance of prostate cancer.¹³ Less commonly, bacterial prostatitis can occur in the transitional zone where the homogeneous low signal intensity can appear identical to the “erased charcoal” sign of prostate carcinoma.¹⁴

Although rare, acute bacterial prostatitis can progress to prostatic abscess in approximately 6% of cases and should be considered when patients fail to improve with antibiotic therapy.¹⁵ Prostate abscess carries significant morbidity and has a mortality rate ranging from 1–16%.^{16,17} Transrectal ultrasound (TRUS) is not indicated for the initial diagnosis of

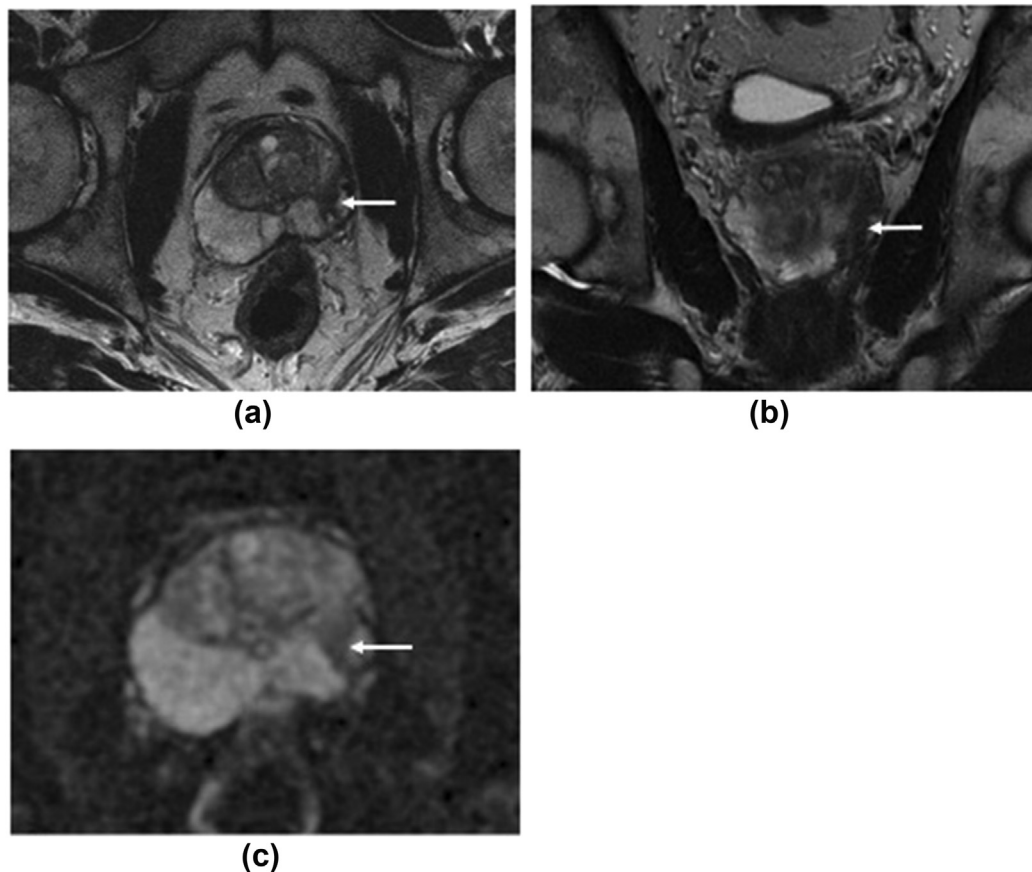


Figure 1 (a) Axial and (b) coronal mpMRI images of a 65-year-old man demonstrated a well-demarcated T2 hypointense lesion in the left peripheral zone (arrow) with corresponding signal loss on the ADC maps (c, arrow). The band-like shaped morphology and absence of mass effect is most consistent with bacterial prostatitis.

prostatitis; however, in the setting of suspected prostatic abscess it has a valuable diagnostic and therapeutic role. This technique is preferable to transperineal or trans-abdominal ultrasound as TRUS involves direct contact with the prostate, thereby improving the resolution. TRUS has been reported to provide an accurate diagnosis in 80–100% of patients, particularly for larger walled-off abscesses⁷; however, in the initial stages of abscess formation, it may be inconclusive.¹⁸ The most common TRUS finding is one or more hypoechoic areas with internal septa and well-defined, thick walls with increased colour Doppler flow signals and intraglandular calcifications.^{7,18,19} Abscesses are typically located in the transitional and central zones, and can cause anatomical distortion.

Cross-sectional imaging in the form of computed tomography (CT) or MRI is often needed in suspected abscess to detect contiguous spread of the infection in nearby organs as well as to provide an objective overview of abscess size and interval progression over time. CT will usually demonstrate prostate enlargement with septa or multiple fluid-like collections, often with peripheral enhancement and determine extra-prostatic involvement. MRI is superior in identifying abscesses of varying sizes and it will typically

demonstrate T1 hypointensity and heterogeneous T2 hyperintensity. After contrast medium administration there will be peripheral enhancement and diffusion weighted imaging will demonstrate diffusion restriction and loss of signal on the ADC maps (Fig 4).

Management

The majority of patients with acute bacterial prostatitis are managed in an outpatient setting with oral antibiotics. Less than 1/6 patients, however, require hospitalisation due to non-response to oral therapy, sepsis, or acute urinary retention.⁹ Prostatic abscesses are a rare complication of acute bacterial prostatitis and surgical intervention is considered when the abscess size exceeds 1 cm.^{16,20} The methods of surgical drainage can be broadly categorised as transurethral resection of the prostate (TURP), transurethral drainage, percutaneous drainage (transrectal and transperineal) or open drainage.^{16,21}

Classically TURP was considered a definitive treatment for prostatic abscess, particularly in elderly patients who have concomitant benign prostatic hypertrophy (BPH); however, this method has been shown to be associated with

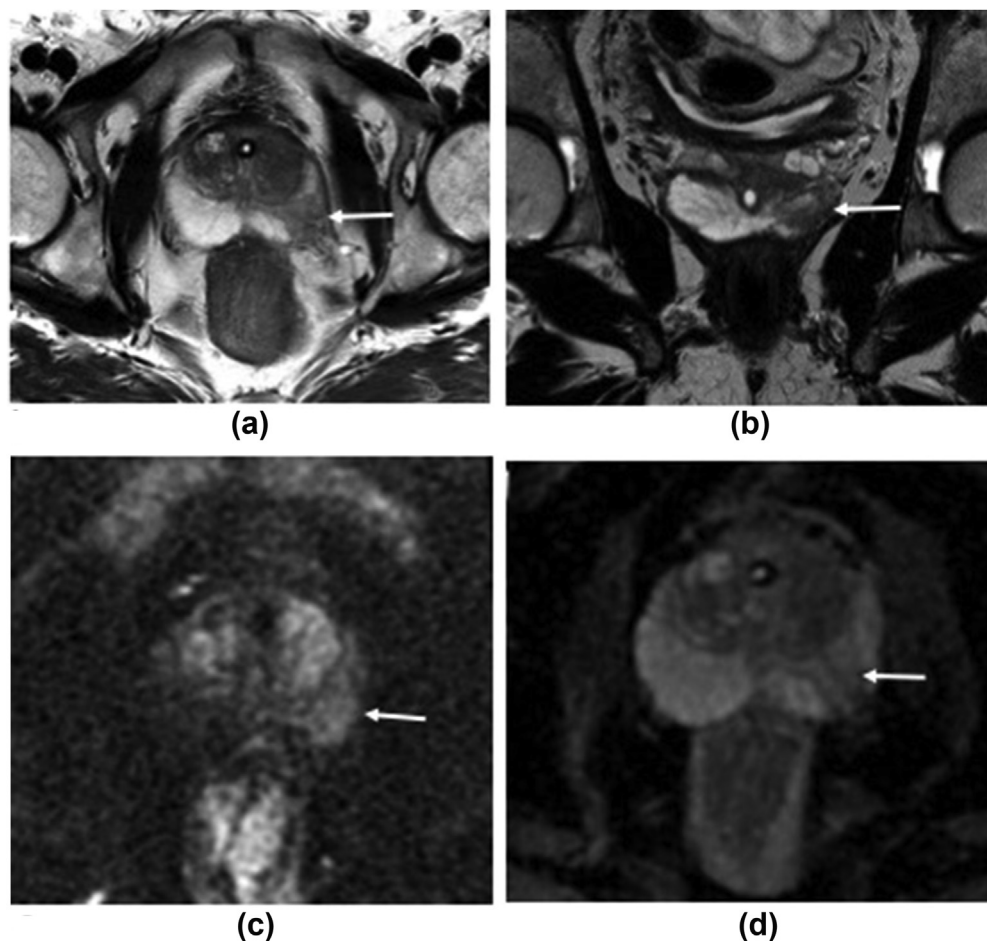


Figure 2 (a) Axial and (b) coronal mpMRI images of 57-year-old man demonstrated a T2 hypointense lesion (white arrows) in the left peripheral zone with corresponding restricted diffusion (c, arrow) and signal loss on the ADC maps (d, arrow). The wedge-shaped morphology, together with the lack of contour deformity of the adjacent prostate tissue and capsule is most consistent with bacterial prostatitis.

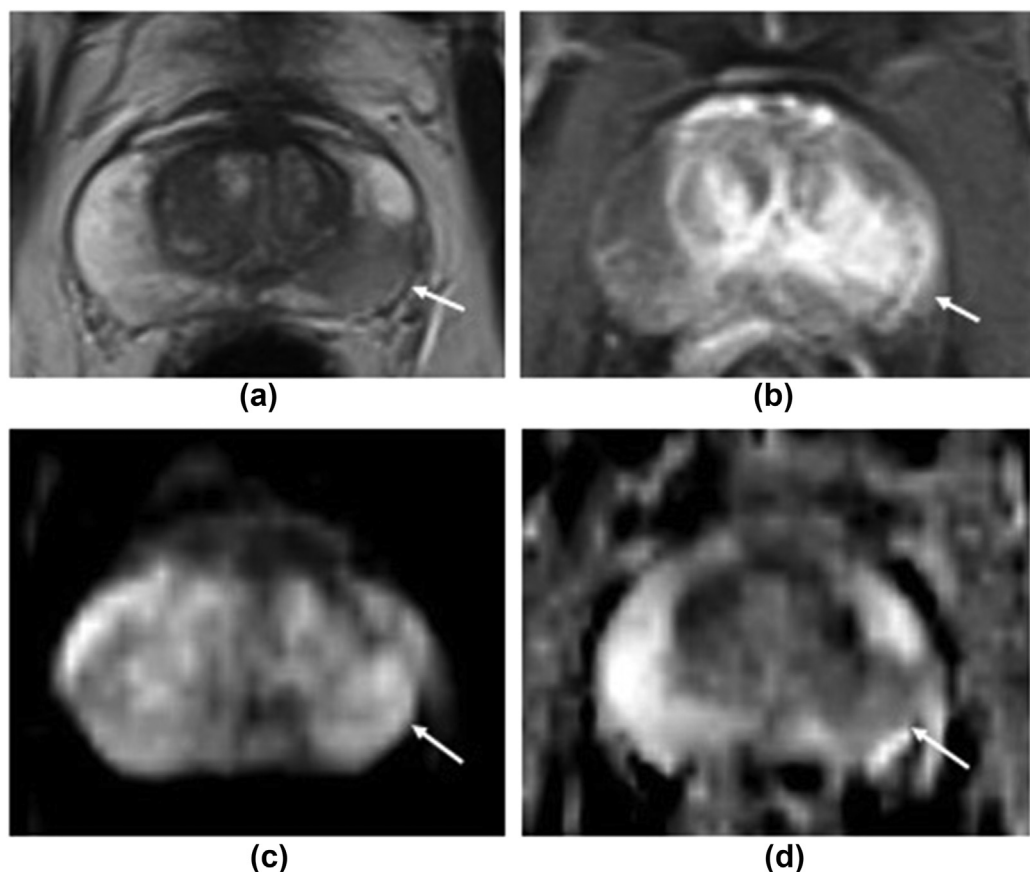


Figure 3 A 65-year-old man with a history of metastatic colorectal adenocarcinoma. MRI pelvis as part of surveillance imaging demonstrated prostatomegaly with a new 1.5 cm T2 hypointense focus in the left posterolateral peripheral zone (a, arrow) with abnormal enhancement (b, arrow), restricted diffusion (c, arrow) and signal loss on ADC maps (d, arrow). This patient was treated for prostatitis and follow-up imaging confirmed interval resolution of signal abnormalities in the prostate gland.

a high incidence of transient urinary incontinence in up to 50% of patients.²² With the changing epidemiology of the disease and younger patients being affected, as well as the evolution of minimally invasive techniques, TURP is usually now only considered in refractory cases with multiple abscesses.²³

Transurethral drainage, otherwise known as un-roofing was considered the most successful method of drainage and was largely the method choice for urologists²⁴; however, due to its more invasive nature and requirement for general anaesthesia, it has largely been superseded by percutaneous ultrasound methods.²⁵ Trans-rectal drainage involves the use of TRUS to guide a needle through the rectal wall and into the prostate abscess for drainage. It can be performed under local anaesthesia and can be repeated with ease in case of failure or incomplete drainage.²⁶ Transperineal drainage also involves the use of TRUS guidance to guide a needle puncturing the perineum into the prostatic abscess. The procedure is more painful than the trans-rectal approach and may require the use of general anaesthesia. The TRUS-guided approach confers many

advantages over the other methods described including a lower risk of retrograde ejaculation and incontinence, which are all potential risks with a transurethral approach.^{25,27} The success rate of TRUS-guided aspirations range from 84–86%^{25,28} and for TRUS-guided drainage is reported to be 83.3%.^{29,30} The main disadvantage of this approach is the inability to adequately allow for complete drainage of multi-loculate abscesses in which case, a transurethral approach may have a role.³¹ Open surgery is very rarely performed and is considered when the abscess has penetrated through the levator ani. The choice of therapeutic intervention therefore depends on a number of factors including the size, location, and number of abscesses, age of the patient, coexisting conditions such as BPH with a preference for TRUS guided drainage as first line.

Granulomatous prostatitis

Granulomatous prostatitis is a rare, benign inflammatory condition of the prostate and accounts for <1% of overall

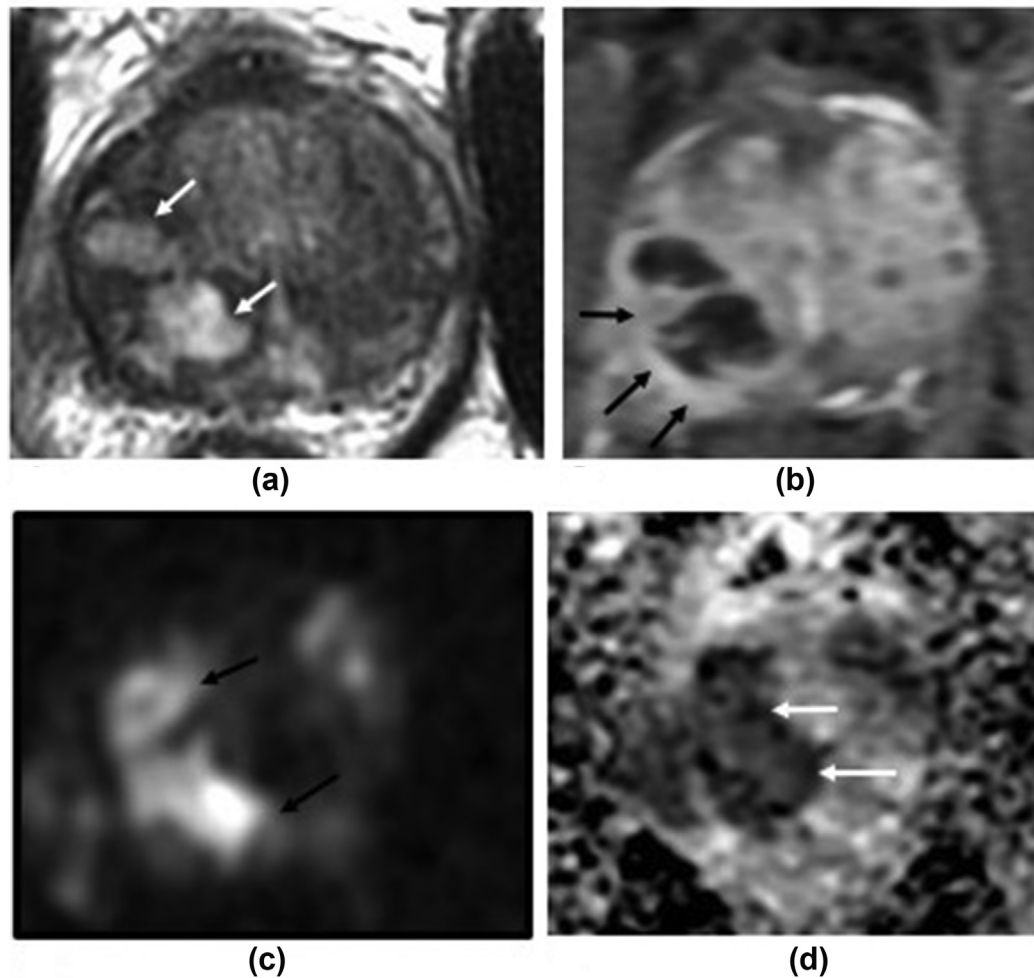


Figure 4 A 60-year-old man with acute bacterial prostatitis and sepsis. Follow-up mpMRI 2 months after treatment with antibiotics, demonstrated asymmetric enlargement of the right prostate gland with interval development of a 2 cm T2 hyperintense (a, arrows) rim-enhancing (b, arrows) fluid collection in the right mid peripheral zone with restricted diffusion (c, arrows) and loss of signal on the ADC maps (d, arrows). These findings are likely in keeping with post-infectious/inflammatory focal fluid collections.

prostatitis cases.^{32,33} It is characterised by well-formed granulomas with epithelioid cell and multinucleated giant cell infiltration with or without central necrosis.³⁴ The main subtypes of granulomatous prostatitis are idiopathic (comprising non-specific and xanthogranulomatous prostatitis), iatrogenic, and infective (necrotic and necrotic).

The most common subtype is idiopathic, making up 60–77.7% of granulomatous prostatitis cases and is often asymptomatic and self-limiting.^{33,35} Iatrogenic is the second most common subtype, comprising 22% of granulomatous prostatitis cases.³⁶ It occurs secondary to transurethral resection of the prostate (TURP) or prostate biopsy. Infective granulomatous prostatitis most commonly occurs as a complication of Bacillus Calmette-Guérin (BCG) immunotherapy for bladder carcinoma but can also be caused by *Mycobacterium tuberculosis* via haematogenous

spread or direct extension from adjacent organs. Other rare infectious agents include *Treponema pallidum*, viruses (herpes zoster), and fungi (*Cryptococcus*, *Candida*, *Aspergillus* spp.).

Imaging features

Reports in the literature have described variable imaging appearances of granulomatous prostatitis. The most common appearance, regardless of subtype is a discrete mass with focal or diffuse hypo-intense signal on T2-weighted imaging (WI) and restricted diffusion with corresponding signal loss on ADC (Fig 5).

Suzuki *et al.* evaluated MRI patterns of BCG-induced granulomatous prostatitis specifically and they identified three main types: diffuse, nodular, and cystic with a mural

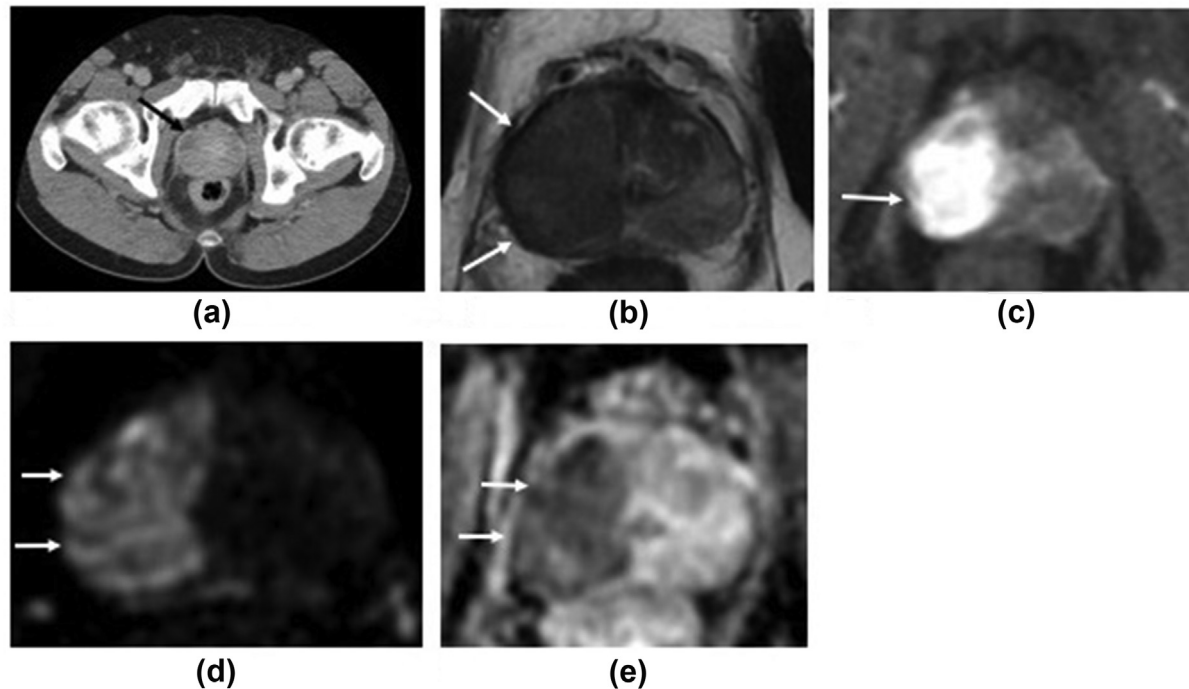


Figure 5 A 59-year-old man with fever, urinary tract infection, and pyuria. (a) The contrast-enhanced CT demonstrated a heterogeneous prostate gland (arrow). (b) The follow-up mpMRI demonstrated a 3.2 cm T2 hypointense lesion (arrows) involving the majority of the right prostate lobe, which demonstrated abnormal enhancement (arrow, (c) and restricted diffusion (d, arrows) with corresponding signal loss on ADC (e, arrows). Prostate biopsy revealed benign prostate tissue with dense granulomatous and lymphoplasmacytic inflammation with scattered micro-abscesses in the right gland in keeping with non-specific granulomatous prostatitis.

nodule. The diffuse subtype was the most common and these lesions showed high signal intensity on T1W, low signal intensity on T2 and higher signal on diffusion-weighted imaging (DWI). With the nodular subtype, they described a polygonal shape with marked hypo-intensity on T2WI to be characteristic³⁷ (Fig 6). Overall, however, granulomatous prostatitis showed mixed signal intensities on T1, T2, and DWI. This variability in signal on T2WI and DWI is seen in tuberculomas elsewhere in the body and is thought to reflect differing amounts of acute inflammation and caseous necrosis^{38,39} (Fig 7).

The most important differential diagnosis to consider is prostate carcinoma, and although there are certain radiological features that may point to one diagnosis over another, biopsy is often required for a definitive diagnosis unless there is a high index of suspicion for BCG-induced granulomatous prostatitis. In a retrospective study performed by Rais-Bahrami *et al.*, they compared the MRI findings of biopsy-proven granulomatous prostatitis cases to Gleason Grade Group 3 (Gleason score $\geq 4 + 3$) or higher cases. They identified that mean ADC values were higher for granulomatous prostatitis, whereas higher stage features, such as extracapsular extension, were common with prostate carcinoma.⁴⁰ Kawada *et al.* compared radiological features specifically of BCG-induced granulomatous prostatitis

with prostate carcinoma. The BCG-induced lesions showed early and prolonged ring enhancement following contrast-enhanced MRI with gadolinium, in contrast to the early enhancement and rapid contrast medium washout seen with prostate carcinoma. Additionally, they also showed BCG-induced granulomatous lesions to reduce in size with anti-tuberculous therapy, which could also help in differentiating them from prostate cancer.⁴¹

IgG4-related prostatitis

IgG4-related disease is rare immune-mediated inflammatory condition affecting a wide range of organ systems. It is characterised by enlargement of the affected organs, tissue IgG4 plasma cell deposition, a variable degree of tissue fibrosis, and often a significantly raised serum IgG4.⁴² Genitourinary system involvement is rare and includes a variety of entities including retroperitoneal fibrosis of the ureter or kidney, tubulointerstitial nephritis, pseudotumour of the bladder, and hypovascular renal lesions. Prostatitis is a relatively newly described manifestation of IgG4 disease and was first reported by Yoshimura *et al.*, in 2006, with a reported incidence ranging from 1–35% in Asian men with IgG4 disease.⁴³ There is, however, limited data regarding

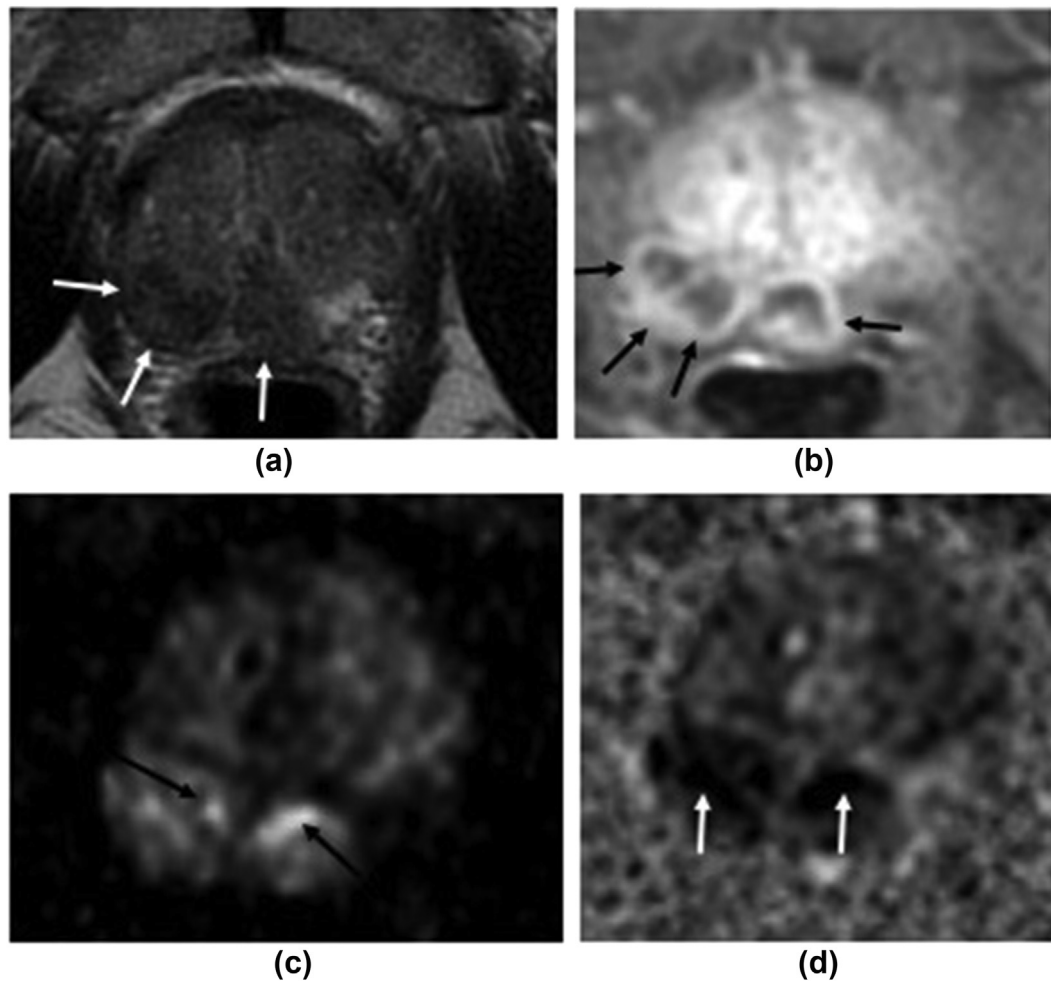


Figure 6 (a) A 65-year-old man with a history of bladder transitional cell carcinoma and BCG treatment underwent mpMRI, which demonstrated background T2 hypointensity involving the majority of the bilateral peripheral zones. There are superimposed nodules in the mid and right posterior peripheral zone that demonstrate more pronounced T2 hypointensity (a, arrows), smooth rim enhancement (b, arrows), and restricted diffusion (c, arrows) with signal loss on the ADC maps (d, arrows). Prostate biopsy demonstrated prostatic tissue with atrophic changes and chronic inflammation with granulomatous inflammation in keeping with BCG granulomatous prostatitis. The smooth rim enhancement of nodules represents the pseudo-capsule with granulation tissue.

the incidence in populations outside Asia. Several studies have reported most cases of IgG4-related prostatitis to be associated with autoimmune pancreatitis or IgG4-associated cholangitis.^{44,45} The clinical presentation is similar to that of BPH or chronic prostatitis, with lower urinary tract symptoms including dysuria, urinary urgency, and a feeling of incomplete emptying.⁴⁶ Corticosteroids are the first-line therapy for symptomatic patients; however, the response varies according to the affected organs and degree of fibrosis. Immunomodulators can also be considered to avoid the effects of long-term corticosteroid use.^{47,48}

Imaging features

Multiparametric MRI often shows non-specific diffuse enlargement of the prostate, focal or diffuse T2

hypointensity and may demonstrate diffusion restriction as well as signal loss on ADC maps.^{49,50} Due to the abundance of inflammatory cells, integrated 2-[¹⁸F]-fluoro-2-deoxy-D-glucose (FDG) positron-emission tomography (PET)-CT is a useful tool in the management of IgG4-related diseases. It can be used to assess organ involvement, monitor therapeutic response, and guide interventional treatment. In the prostate, FDG-PET/CT shows diffusely increased FDG uptake, which can help distinguish it from focal increased uptake with malignancy.⁵¹ When considering a diagnosis of IgG4-related prostatitis, it is crucial to search for synchronous lesions as patients may experience localised symptoms due to infiltration/fibrosis of the affected organ and delay in the diagnosis and treatment can lead to major organ dysfunction and failure⁴⁷ (Figs 8 and 9).

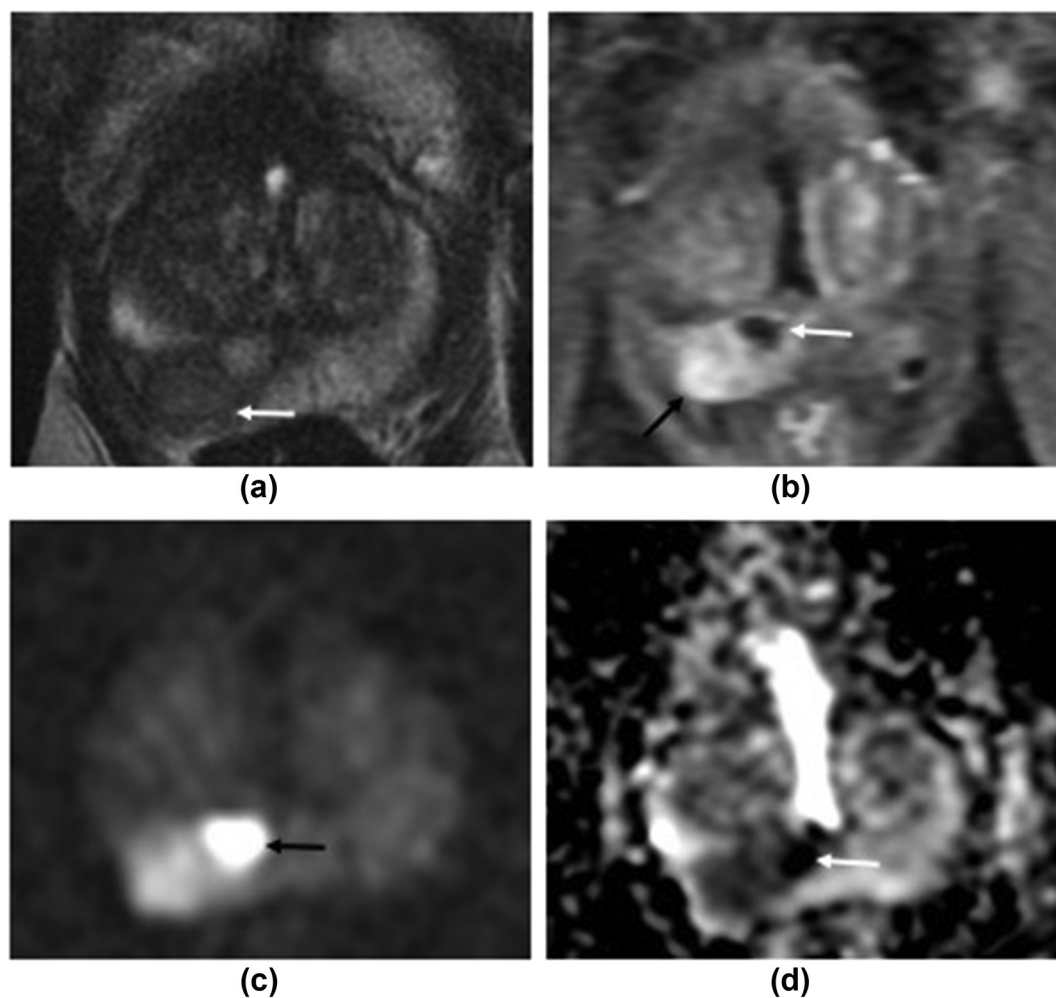


Figure 7 A 71-year-old man with a history of bladder transitional cell carcinoma receiving BCG therapy presented for mpMRI that demonstrated a 2 cm T2 hypointense lesion (a, arrows) in the right peripheral zone with abnormal enhancement (b, black arrow) with a focal area of hypo-enhancement medially (b, white arrow), restricted diffusion with increased signal intensity medially (c, arrow) and corresponding signal loss on the ADC maps (d, arrow) representing focal necrosis. Prostate biopsy demonstrated necrotising granulomas in keeping with BCG treatment effect.

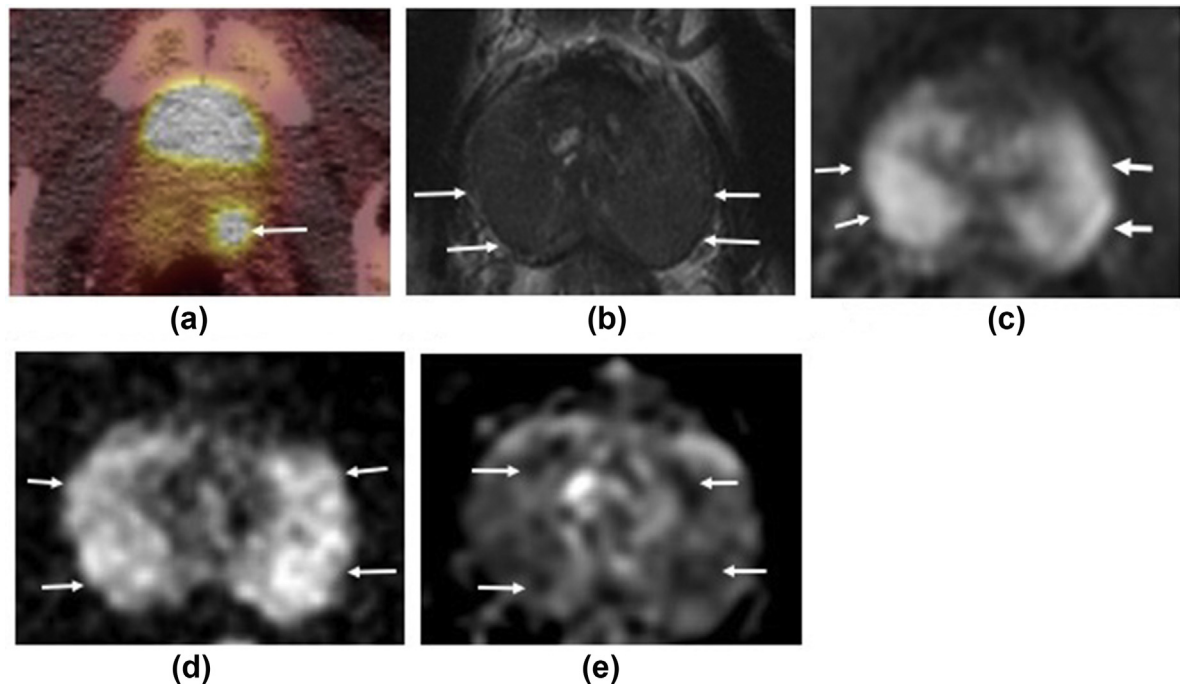


Figure 8 A 32-year-old man with flu-like myalgias and headache in the setting of VGKC autoantibodies. PET-CT was performed to assess for paraneoplastic syndrome and demonstrated asymmetric intense FDG uptake in the left prostate gland (a, arrow). mpMRI, 9 months later, demonstrated diffuse abnormal T2 hypointensity (b, arrows) involving nearly the entirety of the bilateral peripheral zones with associated abnormal enhancement (c, arrows), restricted diffusion (d, arrows) and signal loss on ADC maps (e, arrows). These findings were most compatible with IgG4 related prostatitis (prostate-specific antigen 0.8 ng/ml).

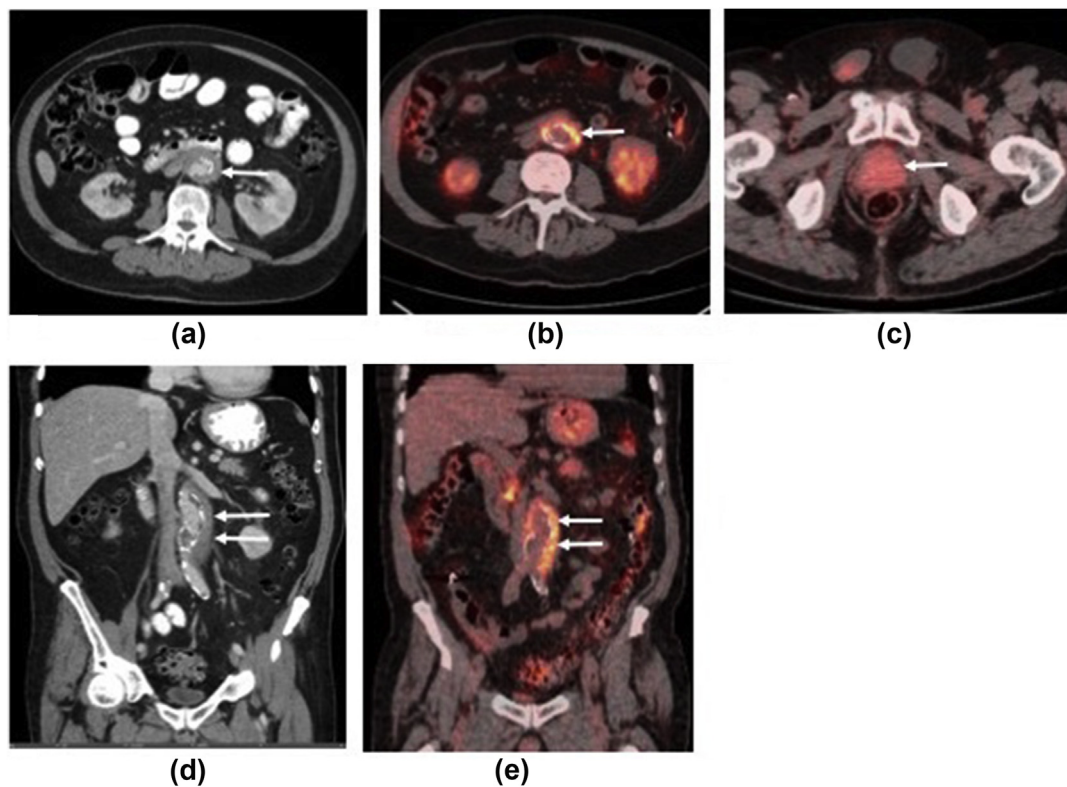


Figure 9 A 78-year-old man with IgG4 disease. PET-CT demonstrated peri-aortic enhancing soft tissue (a,b, arrows) with corresponding increased FDG activity (c,d, arrows) in keeping with IgG4-related aortic vascular disease. The same PET-CT also demonstrated increased FDG activity within the prostate gland, in keeping with IgG4 prostatitis (e, arrow).

Conclusion

Prostatitis encompasses several entities, which have overlapping clinical and radiological features. Accurate diagnosis of the specific type of prostatitis is important so the correct therapy can be initiated, and unnecessary surgical intervention avoided. The main MRI findings include focal or diffuse prostate enlargement, T2 signal hypointensity, diffusion restriction and corresponding low signal intensity on ADC maps. A previous history of BCG immunotherapy, confirmed TB infection, or TURP should prompt consideration of a diagnosis of granulomatous prostatitis. In patients with metachronous organ involvement outside of the prostate gland, IgG4-related disease should be considered. Synchronous disease should also be sought, as IgG4-related disease responds well to corticosteroid therapy and immunomodulators.

The radiological features of prostatitis often overlap with prostate carcinoma, which is the most important differential diagnosis to consider. On T2WI, the hypointense T2 signal areas in prostatitis are usually geographic and ill-defined and generally do not exert mass effect on the adjacent normal prostate tissue in contrast with prostate carcinoma. Although both diseases demonstrate diffusion restriction, in prostatitis it is usually to a lesser degree than seen in prostate carcinoma.^{52,53} Similarly the ADC values tend to be higher in prostatitis patients compared with prostate carcinoma patients^{11,12}. Although prostatitis can mimic prostate carcinoma radiologically, it is important to bear in mind the two may coexist.⁵⁴

Conflict of interest

The authors declare no conflict of interest.

References

- Krieger JN, Lee SWH, Jeon J, et al. Epidemiology of prostatitis. *Int J Antimicrob Agents* 2008;**31**(Suppl. 1):S85. <https://doi.org/10.1016/j.ijantimicag.2007.08.028>.
- Ho D-R. Prostate inflammation: a brief review. *Urol Sci* 2017;**28**(3):113–8. <https://doi.org/10.1016/j.urols.2017.04.003>.
- Adam A, Dixon AK, Gillard JH, et al. *Grainger & Allison's diagnostic radiology*. London: Elsevier Health Sciences; 2020.
- Davis NG, Silberman M. Bacterial acute prostatitis. In: *StatPearls*. Treasure Island, FL: StatPearls Publishing; 2020.
- Dévora Ruano O, de Diego García A, Hernando Real S. [Acute bacterial prostatitis by *Ralstonia pickettii*: clinical and epidemiological considerations of exceptional observation]. *Med Clin (Barc)* 2009;**133**(7):277–8. <https://doi.org/10.1016/j.medcli.2008.07.014>.
- Magri V, Boltri M, Cai T, et al. Multidisciplinary approach to prostatitis. *Arch Ital Urol E Androl* 2018;**90**(4):227–48. <https://doi.org/10.4081/aiua.2018.4.227>.
- Ackerman AL, Parameshwar PS, Anger JT. Diagnosis and treatment of patients with prostatic abscess in the post-antibiotic era. *Int J Urol* 2018;**25**(2):103–10. <https://doi.org/10.1111/iju.13451>.
- Clemens JQ, Meenan RT, O'Keeffe Rosetti MC, et al. Prevalence of and risk factors for prostatitis: population based assessment using physician assigned diagnoses. *J Urol* 2007;**178**:1333–7. <https://doi.org/10.1016/j.juro.2007.05.140>.
- Coker TJ. Acute bacterial prostatitis: diagnosis and management. *Am Fam Physician* 2016;**93**(2):114–20.
- Ikonen S, Kivisaari L, Tervahartiala P, et al. Prostatic MR imaging: accuracy in differentiating cancer from other prostatic disorders. *Acta Radiol* 2001;**42**(4):348–54. <https://doi.org/10.1080/028418501127346972>.
- Meier-Schroers M, Kukuk G, Wolter K, et al. Differentiation of prostatitis and prostate cancer using the prostate imaging-reporting and data system (PI-RADS). *Eur J Radiol* 2016;**85**(7):1304–11. <https://doi.org/10.1016/j.ejrad.2016.04.014>.
- Nagel KNA, Schouten MG, Hambrock T, et al. Differentiation of prostatitis and prostate cancer by using diffusion-weighted MR imaging and MR-guided biopsy at 3 T. *Radiology* 2013;**267**(1):164–72. <https://doi.org/10.1148/radiol.12111683>.
- Lovegrove CE, Matanhelia M, Randeve J, et al. Prostate imaging features that indicate benign or malignant pathology on biopsy. *Transl Androl Urol* 2018;**7**(Suppl. 4):S420–35. <https://doi.org/10.21037/tau.2018.07.06>.
- Sakala MD, Dyer RB, Tappouni R. The “erased charcoal” sign. *Abdom Radiol* 2017;**42**(3):981–2. <https://doi.org/10.1007/s00261-016-0938-x>.
- Ha U-S, Kim ME, Kim CS, et al. Acute bacterial prostatitis in Korea: clinical outcome, including symptoms, management, microbiology and course of disease. *Int J Antimicrob Agents* 2008;**31**(Suppl. 1):S96–101. <https://doi.org/10.1016/j.ijantimicag.2007.07.041>.
- Abdelmoteleb H, Rashed F, Hawary A. Management of prostate abscess in the absence of guidelines. *Int Braz J Urol Off J Braz Soc Urol* 2017;**43**(5):835–40. <https://doi.org/10.1590/S1677-5538.IBJU.2016.0472>.
- Reddivari AKR, Mehta P. Prostate abscess. In: *StatPearls*. Treasure Island, FL: StatPearls Publishing; 2020.
- Singh P, Yadav MK, Singh SK, et al. Case series: diffusion weighted MRI appearance in prostatic abscess. *Indian J Radiol Imag* 2011;**21**(1):46–8. <https://doi.org/10.4103/0971-3026.76054>.
- Jang K, Lee DH, Lee SH, et al. Treatment of prostatic abscess: case collection and comparison of treatment methods. *Kor J Urol* 2012;**53**(12):860–4. <https://doi.org/10.4111/kju.2012.53.12.860>.
- Chou Y-H, Tiu C-M, Liu J-Y, et al. Prostatic abscess: transrectal color Doppler ultrasonic diagnosis and minimally invasive therapeutic management. *Ultrasound Med Biol* 2004;**30**(6):719–24. <https://doi.org/10.1016/j.ultrasmedbio.2004.03.014>.
- Choudhry M, Pellino G, Simillis C, et al. Prostatic abscesses. A case report and review of the literature on current treatment approaches. *Cent Eur J Urol* 2017;**70**(1):118–22. <https://doi.org/10.5173/cej.2016.934>.
- Goyal NK, Goel A, Sankhwar S, et al. Transurethral resection of prostate abscess: is it different from conventional transurethral resection for benign prostatic hyperplasia? *ISRN Urol* 2013 Jun 11;**2013**:109505. <https://doi.org/10.1155/2013/109505>.
- Bhagat SK, Kekre NS, Gopalakrishnan G, et al. Changing profile of prostatic abscess. *Int Braz J Urol* 2008;**34**(2):164–70. <https://doi.org/10.1590/S1677-55382008000200006>.
- El-Shazly M, El-Enzy N, El-Enzy K, et al. Transurethral drainage of prostatic abscess: points of technique. *Nephro-Urol Mon* 2012;**4**(2):458–61. <https://doi.org/10.5812/numonthly.3690>.
- Vyas JB, Ganpule SA, Ganpule AP, et al. Transrectal ultrasound-guided aspiration in the management of prostatic abscess: a single-center experience. *Indian J Radiol Imag* 2013;**23**(3):253–7. <https://doi.org/10.4103/0971-3026.120262>.
- Basiri A, Javaherforoozhadeh A. Percutaneous drainage for treatment of prostate abscess. *Urol J* 2010;**7**(4):278–80.
- Oshinomi K, Matsui Y, Unoki T, et al. Treatment strategy for prostatic abscess: eighteen cases' report and review of literature. *Urol Sci* 2018;**29**(4):206. https://doi.org/10.4103/UROS.UROS_59_18.
- Lim JW, Ko YT, Lee DH, et al. Treatment of prostatic abscess: value of transrectal ultrasonographically guided needle aspiration. *J Ultrasound Med Off J Am Inst Ultrasound Med* 2000;**19**(9):609–17. <https://doi.org/10.7863/jum.2000.19.9.609>.
- Göğüş C, Özden E, Karaboğa R, et al. The value of transrectal ultrasound guided needle aspiration in treatment of prostatic abscess. *Eur J Radiol* 2004;**52**(1):94–8. [https://doi.org/10.1016/S0720-048X\(03\)00231-6](https://doi.org/10.1016/S0720-048X(03)00231-6).
- Collado A, Palou J, García-Penit J, et al. Ultrasound-guided needle aspiration in prostatic abscess. *Urology* 1999;**53**(3):548–52. [https://doi.org/10.1016/S0090-4295\(98\)00570-6](https://doi.org/10.1016/S0090-4295(98)00570-6).
- Lau P, Weiss E. Procedural approaches to drainage of prostatic abscesses. *78*(2):4.

32. Lee S-M, Wolfe K, Acher P, et al. Multiparametric MRI appearances of primary granulomatous prostatitis. *Br J Radiol* 2019;**92**(1098):20180075. <https://doi.org/10.1259/bjr.20180075>.
33. Oppenheimer JR, Kahane H, Epstein JI. Granulomatous prostatitis on needle biopsy. *Arch Pathol Lab Med* 1997;**121**(7):724–9.
34. Kitzing YX, Prando A, Varol C, et al. Benign conditions that mimic prostate carcinoma: MR imaging features with histopathologic correlation. *RadioGraphics* 2016;**36**(1):162–75. <https://doi.org/10.1148/r.2016150030>.
35. Mohan H, Bal A, Punia RPS, et al. Granulomatous prostatitis—an infrequent diagnosis. *Int J Urol Off J Jpn Urol Assoc* 2005;**12**(5):474–8. <https://doi.org/10.1111/j.1442-2042.2005.01068.x>.
36. Shanggar K, Zulkifli MZ, Razack AH, et al. Granulomatous prostatitis: a reminder to clinicians. *Med J Malaysia* 2010 Mar;**65**(1):21–2.
37. Suzuki T, Takeuchi M, Naiki T, et al. MRI findings of granulomatous prostatitis developing after intravesical Bacillus Calmette-Guérin therapy. *Clin Radiol* 2013;**68**(6):595–9. <https://doi.org/10.1016/j.crad.2012.12.005>.
38. Diaz de Leon A, Costa DN, Francis F, et al. Case 258: granulomatous prostatitis. *Radiology* 2018;**289**(1):267–71. <https://doi.org/10.1148/radiol.2018161272>.
39. Bour L, Schull A, Delongchamps N-B, et al. Multiparametric MRI features of granulomatous prostatitis and tubercular prostate abscess. *Diagn Interv Imag* 2013;**94**(1):84–90. <https://doi.org/10.1016/j.diii.2012.09.001>.
40. Rais-Bahrami S, Nix JW, Turkbey B, et al. Clinical and multiparametric MRI signatures of granulomatous prostatitis. *Abdom Radiol* 2017;**42**(7):1956–62. <https://doi.org/10.1007/s00261-017-1080-0>.
41. Kawada H, Kanematsu M, Goshima S, et al. Multiphase contrast-enhanced magnetic resonance imaging features of Bacillus Calmette-Guérin-induced granulomatous prostatitis in five patients. *Kor J Radiol* 2015;**16**(2):342–8. <https://doi.org/10.3348/kjr.2015.16.2.342>.
42. Ezaki T, Akatsuka S, Sanjo T, et al. Symptomatic IgG4-related prostatitis simultaneously diagnosed with aggressive prostate cancer. *Case Rep Urol* 2020 Mar 3;**2020**:6045328. <https://doi.org/10.1155/2020/6045328>.
43. Yoshimura Y, Takeda S, Ieki Y, et al. IgG4-associated prostatitis complicating autoimmune pancreatitis. *Intern Med Tokyo Jpn* 2006;**45**(15):897–901. <https://doi.org/10.2169/internalmedicine.45.1752>.
44. Uehara T, Hamano H, Kawakami M, et al. Autoimmune pancreatitis-associated prostatitis: distinct clinicopathological entity. *Pathol Int* 2008;**58**(2):118–25. <https://doi.org/10.1111/j.1440-1827.2007.02199.x>.
45. Buijs J, Maillette de Buy Wenniger L, van Leenders G, et al. Immunoglobulin G4-related prostatitis: a case–control study focusing on clinical and pathologic characteristics. *Urology* 2014;**83**(3):521–6. <https://doi.org/10.1016/j.urology.2013.10.052>.
46. Hara N, Kawaguchi M, Takeda K, et al. Retroperitoneal disorders associated with IgG4-related autoimmune pancreatitis. *World J Gastroenterol* 2014;**20**(44):16550–8. <https://doi.org/10.3748/wjg.v20.i44.16550>.
47. Zhang W, Stone JH. Management of IgG4-related disease. *Lancet Rheumatol* 2019;**1**(1):e55–65. [https://doi.org/10.1016/S2665-9913\(19\)30017-7](https://doi.org/10.1016/S2665-9913(19)30017-7).
48. Kamisawa T, Zen Y, Pillai S, et al. IgG4-related disease. *Lancet* 2015;**385**(9976):1460–71. [https://doi.org/10.1016/S0140-6736\(14\)60720-0](https://doi.org/10.1016/S0140-6736(14)60720-0).
49. Inui K, Nakagawa Y, Watanabe H, et al. Retroperitoneal fibrosis associated with IgG4-related disease diagnosed by prostate biopsy developed with acute post-renal renal failure: a case report. *Urol Case Rep* 2018;**16**:9–11. <https://doi.org/10.1016/j.eucr.2017.09.017>.
50. Saito T, Stone JH, Nakashima H, et al. *IgG4-Related kidney disease*. London: Springer; 2016.
51. Martínez-de-Alegría A, Baleato-González S, García-Figueiras R, et al. IgG4-related disease from head to toe. *RadioGraphics* 2015;**35**(7):2007–25. <https://doi.org/10.1148/r.357150066>.
52. Shukla-Dave A, Hricak H, Eberhardt SC, et al. Chronic prostatitis: MR imaging and ¹H MR spectroscopic imaging findings—initial observations. *Radiology* 2004;**231**(3):717–24. <https://doi.org/10.1148/radiol.2313031391>.
53. Rosenkrantz AB, Taneja SS. Radiologist, be aware: ten pitfalls that confound the interpretation of multiparametric prostate MRI. *AJR Am J Roentgenol* 2014;**202**(1):109–20. <https://doi.org/10.2214/AJR.13.10699>.
54. Medlicott SAC, Oryschak A, Trpkov K. IgG4 prostatitis associated with prostatic adenocarcinoma: a case report and literature review. *Hum Pathol Case Rep* 2018;**14**:8–11. <https://doi.org/10.1016/j.ehpc.2018.05.005>.