



Rapidly recurrent prostatic obstruction due to granulomatosis with polyangiitis

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ABSTRACT

Granulomatosis with polyangiitis (GPA) is a rare cause of prostatitis. Our case illustrates a case of granulomatous prostatitis secondary to unrecognised GPA requiring multiple surgical interventions. The patient presented with lower urinary tract symptoms. They underwent two endoscopic prostatic resections for recurrent urinary obstruction due to granulomatous prostatitis before a diagnosis of GPA was made. The rarity of this pathology, challenges in making a diagnosis and the systemic nature of this disease are emphasised.

Introduction

Granulomatosis with polyangiitis is an autoimmune systemic vasculitic disease. The aetiology remains unknown. However there is a strong association with the cytoplasmic and perinuclear antineutrophil cytoplasmic auto-antibodies (c-ANCA, p-ANCA)¹ Diagnosis should be considered if there is clinical, serological (ANCA) and histological evidence of systemic involvement.¹ Differential diagnosis for GPA include the infective causes mycobacterium tuberculosis, blastomyces, dermatitidis brucella species and spirochetes. The systemic inflammatory disorder sarcoidosis should also be considered.²

Classically the upper respiratory tract (92%), lungs (85%) and kidneys (77%) are involved.¹ Prostatic involvement has rarely been reported in systemic GPA. Large series reviewing patients with GPA have noted less than 1% of cases with documented urogenital involvement.² A large study conducted by Stillwell et al. reviewed 25,000 biopsies and reported only 2 cases of histologically diagnosed prostatitis secondary to GPA.³

Treatment generally involves immunosuppressive therapy (glucocorticoid and cyclophosphamide) while surgical management is rarely required. Here we present a patient requiring multiple surgical interventions secondary to rapidly recurring prostatic GPA.

The case

A 78 year old male presented for urological assessment with a three

month history of lower urinary tract symptoms. Relevant past medical history included stable rheumatoid arthritis, scleritis, cryptogenic organizing pneumonia, chronic sinusitis, polymyalgia rheumatica and osteopenia.

Digital rectal exam revealed a benign feeling, moderately enlarged prostate gland. Examination findings were otherwise unremarkable.

Prostate specific antigen was 1.18 ng/ml and urine was sterile with mild leucocytosis and microscopic haematuria. Full blood count was normal while erythrocyte sedimentation rate (ESR) was elevated to 30 mm/hr. Urinary tract ultrasound reported prostatomegaly (volume 54 cc). The symptoms were thought to be due to benign prostatic hyperplasia and pharmacological therapy was commenced with dutasteride/tamsulosin.

Two months later the patient's voiding symptoms had worsened. Pharmacological treatment was ceased and transurethral resection of the prostate (TURP) scheduled. An enlarged prostate with a focal papillary lesion arising from the left lobe was noted. Twenty six grams of tissue was resected. Histopathology reported widespread necrotising granulomatous inflammation (Fig. 1.).

Despite initial post-operative symptomatic improvement, urinary symptoms recurred within four months. Cystoscopy revealed significant regrowth of prostatic tissue and a second TURP was performed. Eighteen grams of prostatic tissue was resected and histopathology once again reported necrotising, granulomatous inflammation.

Four weeks later, because of troublesome sinusitis, the patient underwent frontal sinus biopsy also showing granulomatous inflammation

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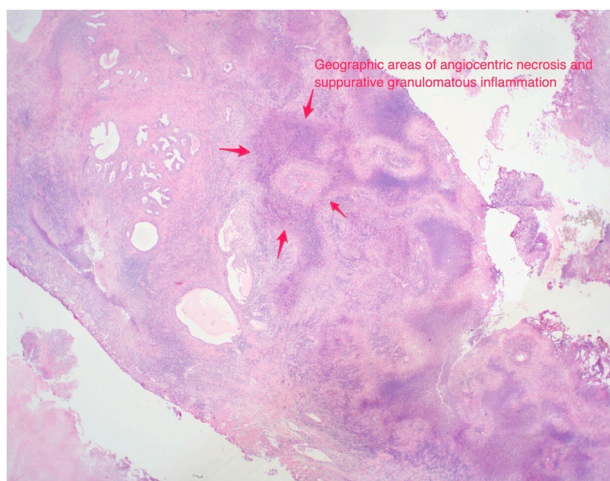


Fig. 1. Geographic areas of angiocentric necrosis and suppurative granulomatous inflammation.

with vasculitis (Fig. 2.). Repeat ANCA showed a moderate c-ANCA titre of 40 U/ml. Thus multisystem GPA was diagnosed.

Immunosuppressive treatment was initiated with prednisolone and rituximab. However 1 week following treatment induction the patient was admitted to hospital with epigastric pain and increasing dyspnoea. CT chest revealed large, thick walled cavitating lesions extending from right hilum to the upper right lobe apex (Fig. 3.). Considering the clinical context, radiology reported the likely diagnosis as pulmonary GPA.

The patient died at home several days later due to an uncertain cause.

Discussion

This report describes a case of unrecognised GPA complicated by prostatic involvement, recurring rapidly after surgical resection. Because of its rarity, there are few treatment outcome reports for this condition.

Medical therapy normally includes immunosuppressive therapy (cyclophosphamide and prednisone).² This combination is generally successful and leads to disease remission in most cases.² More recently rituximab in combination with prednisone has also proven to be effective.²

As GPA was not apparent at presentation the patient initially received surgical rather than medical therapy. Other documented

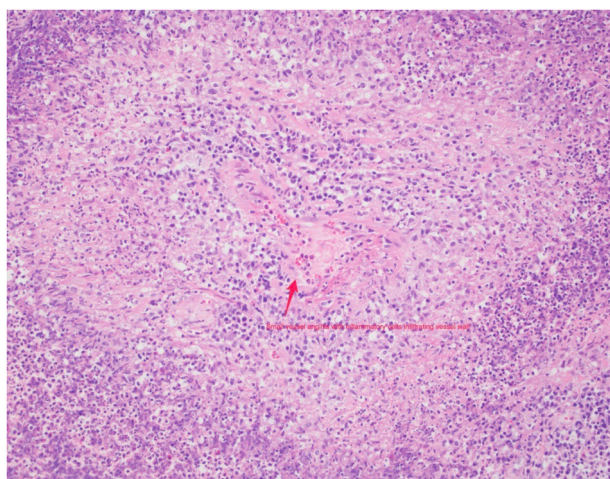


Fig. 2. Small vessel angiitis with inflammatory cells infiltrating vessel wall.

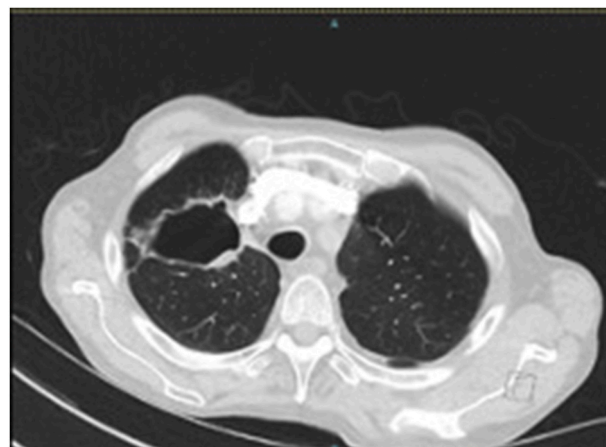


Fig. 3. CT imaging revealing a large thick walled cavitating lesion extending from the right hilum to the right upper lobe apex.

surgical interventions include suprapubic catheterization and radical prostatectomy.^{1,2} Khattak et al. documented only four of 26 patients (15%) with prostatitis secondary to GPA requiring TURP, one for gross haematuria, two for bladder outlet obstruction (BOO) and one for recurrent urinary tract infections and BOO.⁴ To our knowledge there have been no reported cases of rapid prostatic regrowth requiring multiple TURPs due to GPA. Our patient's inflammatory prostatic obstruction may not have recurred if the diagnosis of GPA was recognised earlier and effective immunosuppression implemented.

Yet the signs and symptoms of GPA can be vague and definitive serological and pathological results are not always reliable. While high levels of c-ANCA have a specificity of 99%, their sensitivity of 41%–96% varies with disease extent and severity.¹ Pulmonary biopsy has a sensitivity ranging from 10 to 80% while the sensitivity of biopsies from the nose or sinuses is estimated at 20–50%.⁵

Conclusion

GPA can rarely involve the prostate gland causing obstruction with the potential to recur rapidly after endoscopic resection. Early recognition and effective immunosuppression may minimise the need for surgical intervention. GPA should be considered in patients with unexplained prostatitis; however diagnosing this systemic autoimmune disease can be challenging.

Consent for publication

Written informed consent was obtained from the patient's family for publication of this case report and the accompanying images. A copy has been provided to the editor of this journal and can be obtained for review on request.

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Declaration of competing interest

None.

Abbreviations:

GPA	Granulomatosis with polyangiitis
c-ANCA, p-ANCA	Anti-neutrophil cytoplasmic antibody (cytoplasmic and perinuclear)
ESR	Erythrocyte sedimentation rate

TURP Transurethral resection of the prostate
BOO Bladder outlet obstruction

References

1. Pereira Beceiro J, Rodríguez Alonso A, Bonelli Martín C, Pérez Valcárcel J, Mosquera Seoane T, Cuerpo Pérez M. Prostatitis and acute urinary retention as first manifestations of Wegener's granulomatosis. *Reumatol Clínica*. 2014;10(6):409–412.
2. Alba MA, Moreno-Palacios J, Beça S, Cid MC. Urologic and male genital manifestations of granulomatosis with polyangiitis. *Autoimmun Rev*. 2015;14(10):897–902.
3. Stillwell TJ, Engen DE, Farrow GM. The clinical spectrum of granulomatous prostatitis: a report of 200 cases. *J Urol*. 1987;138(2):320–323.
4. Khattak AQ, Nair M, Haqqani MT, Williamson EP. Wegener's granulomatosis: prostatic involvement and recurrent urinary tract infections. *BJU Int*. 1999;84(4):531–532.
5. Masiak A, Zdrojewski Z, Pęksa R, et al. The usefulness of histopathological examinations of non-renal biopsies in the diagnosis of granulomatosis with polyangiitis. *Reumatologia*. 2017;55(5):230–236.