Sir Salimullah Medical College Journal

Sir Salimullah Med Coll J 2024; 32: 84-88

Case Report

DOI: https://doi.org/10.3329/ssmcj.v32i2.84715

Tuberculosis and Granulomatosis with Polyangiitis: A Rare Overlap

Ayesha Siddiqua[1], Md. A Hossain[2], Md. M Khair[2]

Article information
Received: 15.08.2025
Accepted: 01.10.2025

Cite this article:
A Siddiqua, Md A Hossain, Md M Khair. Tuberculosis and Granulomatosis with Polyangiitis: A Rare Overlap. Sir Salimullah Med Coll J 2024; 32: 84-88

Key words:
Tuberculosis, Granulomatosis with polyangitis, vasculitis, immunohistochemistry

Abstract:

Bangladesh, a high tuberculosis (TB) burden country, frequently faces diagnostic challenges due to the protean manifestations of TB. Granulomatosis with polyangiitis (GPA), an ANCA-associated vasculitis, can closely mimic TB in clinical and radiological features, and very rarely the two may coexist. We report a 47-year-old male with fever, weight loss, respiratory distress, and mediastinal mass initially treated as TB. Subsequent deterioration with renal involvement, c-ANCA positivity, and hematuria suggested GPA. Immunohistochemistry of the mediastinal biopsy confirmed mycobacterial antigen, supporting concomitant TB. The patient required intensive care with anemia correction, antibiotics, and heart failure management. Following stabilization, he was treated with methylprednisolone and rituximab for GPA alongside a modified anti-TB regimen, which was well tolerated. His renal function and lung status improved significantly, and he was discharged on maintenance immunosuppression and anti-TB therapy. This case highlights the critical importance of considering dual pathology, as timely recognition and combined therapy can be lifesaving.

Introduction:

Bangladesh is one of the 30 countries with the highest TB burden in the world; TB is a multi-systemic disease with myriad presentations and manifestations. On the other hand, vasculitis is a general term for a heterogeneous group of diseases characterized by inflammation and destruction of blood vessel walls.[1] At times the difference between TB and vasculitis can be difficult to determine, because they share similar characteristics, and moreover, both entities can coexist in the same patient very rarely.[2] Granulomatosis with polyangiitis (GPA; formerly known as Wegener's granulomatosis) is a rare necrotizing vasculitis combining inflammation of the vascular wall and peri- and extravascular granulomatosis.[3] According to current nomenclature classification it belongs to the anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) group, alongside microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis (EGPA)[4] The classic form of GPA involves upper airway (ear, nose, throat; ENT), lung, and kidney involvement; however, any organ can be affected. Constitutional symptoms, such as myalgias, loss of appetite, weight loss, fatigue, fever, night sweats, and migratory arthropathy, appear in 50% of patients and may precede weeks to months prior to the onset of clinically apparent organ-involvement.[5]

1. Specialist, Dept of Respiratory Medicine, Bangladesh Specialized Hospital, Dhaka, Bangladesh
2. Consultant, Dept of Respiratory Medicine, Bangladesh Specialized Hospital, Dhaka, Bangladesh

Correspondence: Dr Ayesha Siddiqua, Specialist, Dept of Respiratory Medicine, Bangladesh Specialized Hospital, Dhaka, Bangladesh. E- mail: ayesha.sbmc@gmail.com.

Both TB and GPA, can present with pulmonary lesions, cavitations, fever, arthralgia, and granulomatous inflammation. Thus, conclusive diagnostics (biopsy, ANCA titres, sputum, PCR) are crucial. We report a challenging case of a patient who presented with simultaneous TB and granulomatosis with polyangitis (GPA).

Case report:

A 47-year-old male patient, previously who had history of intermittent low grade fever, dry cough and significant weight loss for 3 months, reported into ICU of Bangladesh Specialized Hospital with sudden severe respiratory distress for 2 days followed by desaturation for 1 day (SpO2 72% in room air at home). Drug history included anti-TB medications (4FDC) (given on the basis of suggestive history, Monteux test 14 mm & non caseous granulomatous inflammation as revealed in the core biopsy from mediastinal mass). However, 2 weeks later, Anti-Koch's was stopped due to generalized weakness, bodyache and pain in upper abdomen. Without any noteworthy past cardiac or lung issue, 1 week after stopping anti-TB medications, he developed gradual onset of breathlessness followed by admission into a tertiary hospital where his anti-Koch's drugs were restarted. Owing to progressive generalized deterioration, he was shifted to ICU in Bangladesh Specialized Hospital.

Thereafter, we found the patient in need of high flow oxygen (30L) by NIV in ICU. He was severely anemic, dyspneic as well as bipedal edema was present. He had bilateral lung crepitations up to mid zones. Patient has no history of hemoptysis, epistaxis or any bleeding manifestation, no small joint pain, pins or needles sensation in hands or feet, headache, vision problem, oral ulcer, rash, photosensitivity, no GI feature.

His investigation profile was consistent with sepsis, anemic heart failure, moderate pericardial effusion & AKI. His Hb was 5.8 g/dl, WBC 20,000/$mm^{3,}$ S. NTProBNP was 3830 pg/ml, S. Creatinine 2.6 mg/dl & Chest x-ray was showed alveolar edema, upper zone vessel predominance, hazy lung field. 5 days later, HRCT chest revealed bilateral with traction bronchiectasis. Hence he was resuscitated with PRBC, diuretics, antibiotics as well as his anti-TB medications were kept hold due to altered LFTs. Meanwhile his immunohistochemistry report (Fig. 3) from mediastinal mass came positive for mycobacterial antigen. Being normal previously, during hospital stay, microscopic hematuria was identified with red cell casts & phase contrast microscopy showed 70% dysmorphic RBC. After 3 days of admission patient was shifted into IPD with 2 liters oxygen maintaining oxygen saturation 90%. His c-ANCA was positive in high titre (62u/ml). After improvement of heart failure and infection, he was given Methylprednisolone & Rituximab induction & maintenance regimen. In addition, anti-TB medications were restarted sparing Pyrazinamide, which he tolerated well enough. Around 2 weeks later, his renal function, lung condition improved, patient was completely free of supplementary oxygen. While on discharge he was planned on maintenance prednisolone & rituximab therapy along with anti-TB drugs. Till date, patient is stable with s. creatinine 1.4 mg/dl, normocytic normochromic anemia with no new pulmonary issue.

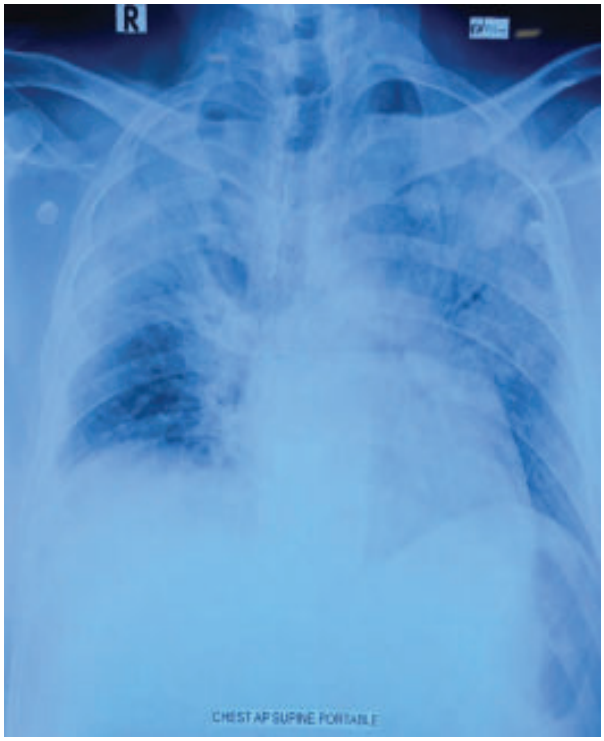


Fig. 1: *Initial chest x-ray showing alveolar edema, upper lobe diversion, haziness of whole lung field.*

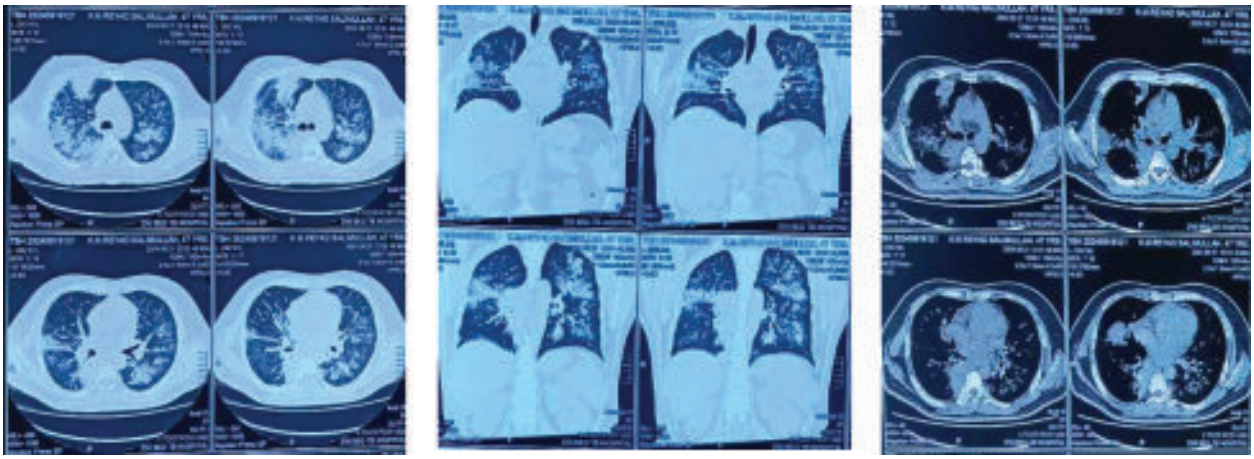
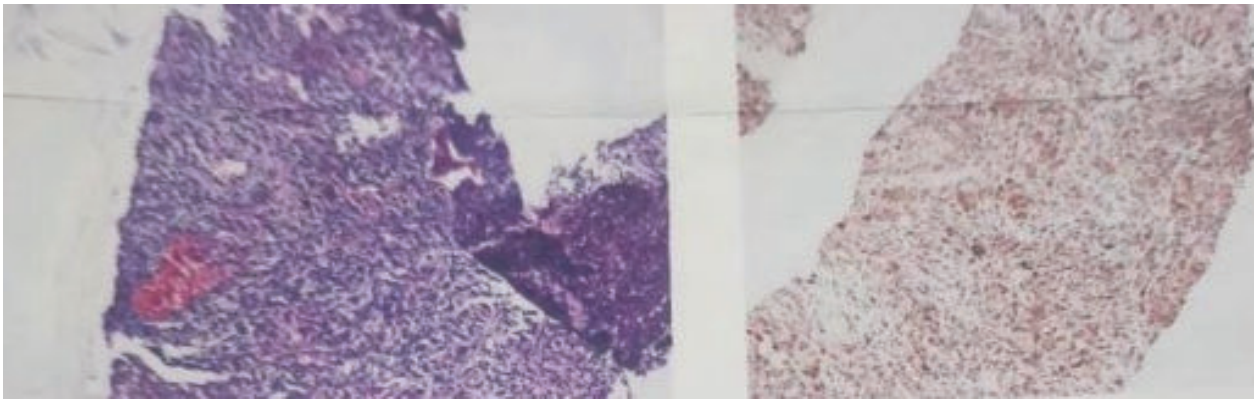


Fig. 2: HRCT chest shows bilateral consolidation with some traction bronchiectasis, 5 days after the acute heart failure state resolved.



H&E: granulomatous inflammation, histologically tuberculous Mycobacterium

Fig. 3: Immunohistochemistry report positive for Mycobacterial antigen

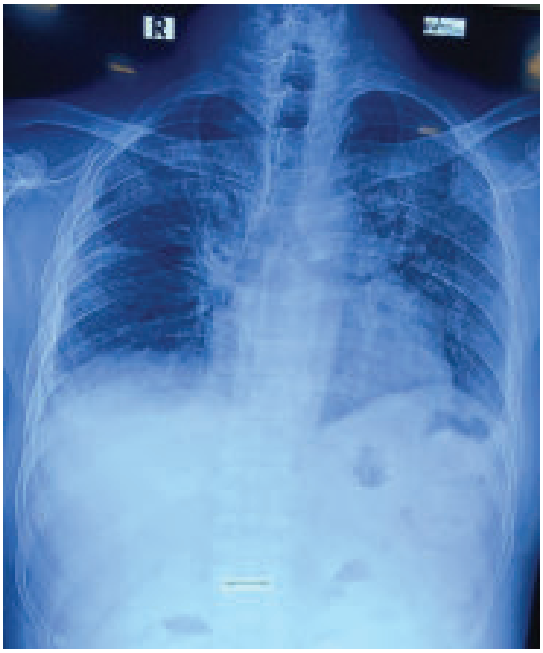


Fig. 4 : During discharge, patient's chest x-ray was improved with some remaining ill defined opacities.

Discussion:

Our patient was diagnosed with simultaneous GPA as well as TB. Any attempt to differentiate GPA from TB can be challenging, since the two conditions share similar clinical and radiological features.[6] Detection of GPA relies on a blend of systemic semiotics indicative of vasculitis, serology positive for c-ANCA and histopathology confirming crescent glomerulonephritis, necrotizing vasculitis and granulomatous inflammation from a biopsy of relevant organ like skin, lung or kidney.[7] According to the American College of Rheumatology two or more of the following criteria are needed to diagnose GPA with a specificity of 92%: nasal/oral inflammation, abnormal urinary sediment, abnormal CXR and suggestive kidney histology. In our patient an abnormal CXR, an abnormal urinary sediment and renal involvement employed to diagnose GPA though taking renal biopsy wasn't feasible.

Immunohistochemistry is a reliable test with high sensitivity as well as high negative predictive value which can be done rapidly for establishing an etiological diagnosis of tuberculosis in histologic specimens.[8] Chronic granulomatous inflammation, which is a classical histological change seen in TB, is considered as the basis for the diagnosis of TB.[9] Considering the positive predictive value of IHC (~40%) finding of MTB antigen, anti TB drugs were thought safer to continue in our patient which actually didn't worsen his condition despite immunosuppressants owing to shape up the diagnosis of TB more strongly.For instance, Iijima et al. described a patient with tuberculous lymphadenitis who developed rapidly progressive renal failure and pleurisy with elevated PR3-ANCA—ultimately diagnosed as GPA during TB treatment—highlighting a true co-occurrence of GPA and TB. Steroid therapy alongside continued anti-TB treatment resulted in clinical improvement.[10]

Conclusion:

Clinicians should always be aware of potential multiple conditions when considering differential diagnoses. Whilst immunosuppressive therapy is relatively contraindicated with active TB, untreated GPA might be life threatening and would result in unfavorable renal outcome in the long term. If an inappropriate diagnosis of GPA is made, the immunosuppression will result in worsening of underlying TB.[11]So, judicious diagnosis is of surmount importance. Combined treatments for both GPA and TB showed positive patient response, according to published case reports[12,13,14]. Keen observation is even more mandatory for such cases where TB itself is a burdensome entity.

Conflict of Interest:

The authors stated that there is no conflict of interest in this study.

Funding:

This research received no external funding.

Acknowledgments:

The authors were grateful to the staffs of the Dept of Respiratory Medicine, Bangladesh Specialized Hospital, Dhaka, Bangladesh.

Authors' contributions:

All authors were involved in the management of the patient and all authors contributed to the conception, writing, and editing of the case report.

References:

1. Elefante E, Tripoli A, Ferro F, Baldini C. One year in review: systemic vasculitis. Clin Exp Rheumatol. 2016;34 Suppl 97(1):S1–6.
2. Molinari L, Melamud JI, Ferrari L, Landi P, Semeniuk G, Quadrelli SA. Granulomatosis de Wegener y tuberculosis. Una mala combinación. Medicina (B Aires). 2009;69(6):640–2.
3. Puechal X. Granulomatosis with polyangiitis (Wegener's). Joint Bone Spine. 2020;87(6):572–8. doi:10.1016/j.jbspin.2020.06.005.
4. Jennette JC, Falk RJ, Bacon PA, Basu N, Cid MC, Ferrario F, et al. 2012 revised international Chapel Hill consensus conference nomenclature of vasculitides. Arthritis Rheum. 2013;65(1):1–11. doi:10.1002/art.37715.
5. Comarmond C, Cacoub P. Granulomatosis with polyangiitis (Wegener): clinical aspects and treatment. Autoimmun Rev. 2014;13(11):1121–5. doi:10.1016/j.autrev.2014.08.017.
6. Kshirsagar AY, Sulhyan SR, Patil MV, Patil YV, Bansode YD. A rare case of coexistence of Wegener's granulomatosis and pulmonary tuberculosis. Lung India. 2012;29(1):90–2. doi:10.4103/0970-2113.92371. Available from: https://www.springermedizin.de/a-rare-case-of-coexistence-of-wegener-s-granulomatosis-and-pulmo/19661640
7. Greco A, Marinelli C, Fusconi M, Macri GF, Gallo A, De Virgilio A, et al. Clinic manifestations in granulomatosis with polyangiitis. Int J Immunopathol Pharmacol. 2016;29(2):151–9. doi:10.1177/0394632015623353.
8. Yalcin F, Kilic A, Turan S, Ozcelik N. Coexistence of granulomatosis with polyangiitis and tuberculosis: a diagnostic dilemma. Tuberk Toraks. 2024;72(3):229–33. doi:10.5578/tt.20241912. Available from: https://thoracrespract.org/Content/files/sayilar/182/TRP_July_2024-33-36.pdf
9. Siddiqui M, Alotaibi M, et al. Granulomatosis with polyangiitis misdiagnosed as tuberculosis: a case report. Cureus. 2024;16(3):e56387. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11363386/
10. Iijima Y, Kobayashi Y, Uchida Y, et al. A case report of granulomatous polyangiitis complicated by tuberculous lymphadenitis. Medicine (Baltimore). 2018;97(43):e12430. doi:10.1097/MD.0000000000012430.
11. Sunderkötter C, Bröcker EB, et al. Concomitant granulomatosis with polyangiitis and tuberculosis: a case

report and review. J Med Case Rep. 2016;10:186. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4954315/

12. Nayak V, Kumar A, et al. Granulomatosis with polyangiitis masquerading as tuberculosis. BMJ Case Rep. 2018;2017:bcr-2017-221948. doi:10.1136/bcr-2017-221948. Available from: https://casereports.bmj.com/content/2018/bcr-2017-221948
13. Liou HH, Huang CC, et al. Extracapillary IgA nephropathy and pulmonary hemorrhage. Clin Pulm Med. 2005;12(5):310–2. Available from: https://journals. lww.com/clinpulm/abstract/2005/09000/extracapillary_iga_nephropathy_and_pulmonary.4.aspx
14. Beaufils H, Morel-Maroger L, et al. Rapidly progressive glomerulonephritis and pulmonary hemorrhage in Wegener's granulomatosis. Nephron. 1991;57(2):251–4. doi:10.1159/000186341. Available from: https://karger.com/nef/article-abstract/57/2/251/216472.