A Storm of Polyserositis: Unravelling Multisystem Effusions in Hypereosinophilic Syndrome.

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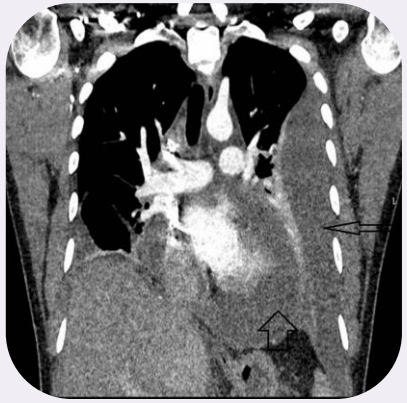
Background

Idiopathic hypereosinophilic syndrome is a rare disorder with persistent eosinophilia and multi-organ involvement. It may mimic common conditions like pneumonia but can progress to life-threatening complications including polyserositis and cardiac tamponade

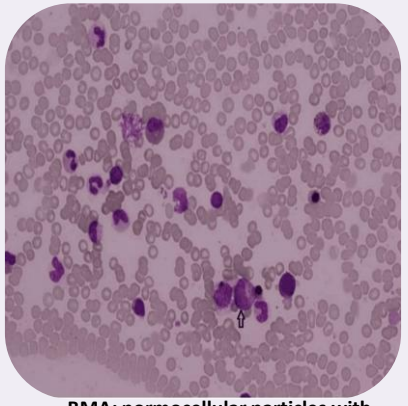
Case Presentation

A 42-year-old previously healthy man presented with fever, pleuritic chest pain, and breathlessness. Initially diagnosed with community-acquired pneumonia, he was treated with antibiotics but showed no clinical improvement. Over the following days, his condition deteriorated, developing bilateral pleural effusions, large pericardial effusion with tamponade, and ascites. Pleural and pericardial fluid analyses revealed eosinophil-rich exudates, with cultures repeatedly negative. Extensive diagnostic evaluation excluded tuberculosis, bacterial infection, parasitic infestations, autoimmune disease, and malignancy. Rising eosinophil counts above 1.5 × 10^9/L, combined with multi-organ involvement and exclusion of secondary causes, supported the diagnosis of idiopathic hypereosinophilic syndrome.

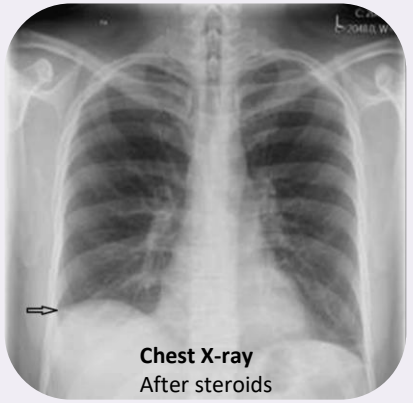
Investigations



large left-sided pleural effusion with large pericardial effusion



BMA: normocellular particles with increased eosinophils.

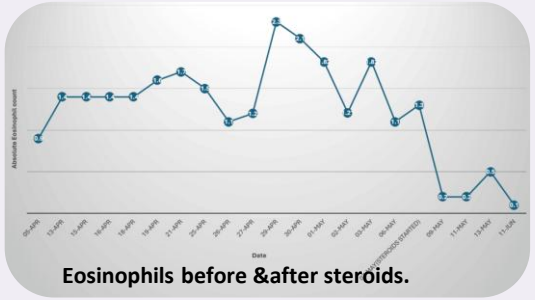


Chest X-ray
After steroids

Test	Result	Comment	Reference range
Vitamin B12	171 ng/L	Normal	160–950 ng/L
IgG	8.9 g/L	Normal	7.0–16.0 g/L
IgA	3.81 g/L	Normal	0.85–4.5 g/L
IgM	0.89 g/L	Normal	0.5–2.0 g/L
IgE	277 KU/L	Elevated	<100 KU/L
Troponin	3.5 ng/L	Normal	<14 ng/L
Plasma BNP	25.0 pg/L	Normal	<100 pg/L
Rheumatoid Factor	2 IU/L	Normal	<14 IU/L
ANCA screen	Negative	Normal	Negative
Parasitology screen	Negative	Normal	Negative
C3	1.91 g/L	Mildly elevated	0.9–1.8 g/L
C4	0.32 g/L	Normal	0.1–0.4 g/L
Hepatitis C antibodies	Negative	Normal	Negative
CMV antibodies	IgG positive, IgM negative	Normal	IgG positive, IgM negative indicates past infection
HIV antibodies	Negative	Normal	Negative
Tryptase	5.5 ng/L	Normal	<11.4 ng/L
Anti-CCP antibodies	2 U/L	Normal	<20 U/L
Anti-dsDNA antibodies	3 IU/L	Normal	<15 IU/L
ENA antibody screen	Negative	Normal	Negative
ANA antibodies	Negative	Normal	Negative
Serum ACE level	17 U/L	Normal	8–52 U/L

TABLE 1: Detailed infectious and autoimmune screen results.

BNP: B-type natriuretic peptide; ANCA: anti-neutrophil cytoplasmic antibodies; EBV: Epstein–Barr virus; CMV: cytomegalovirus; HIV: human immunodeficiency virus; CCP: cyclic citrullinated peptide; ENA: extractable nuclear antigen; ANA: antinuclear antibody; ACE: angiotensin-converting enzyme



Eosinophils before &after steroids.

Investigation	Result
BCR:ABL1, FIP1L1:PDGFRA, PDGFRB, JAK2, FGFR1, and *KIT*	Negative
Bone marrow immunophenotyping	CD34+ myeloid progenitors accounted for 0.4% of the total nucleated cell count with a CD117+, HLADR+, CD33+, and CD13+ phenotype. No clonal B-cells were identified. Negative for the lymphocytic variant of hypereosinophilic syndrome
Bone marrow microscopy	Increased eosinophil count. No evidence of lymphoma or myeloid pathology

Table 2: Bone marrow aspiration showing no malignant cells but revealing increased eosinophils.

Outcome

The patient was started on high-dose corticosteroid therapy following pericardial drainage. He demonstrated dramatic clinical improvement, with resolution of fever, normalization of eosinophil counts, and gradual disappearance of pleural and pericardial effusions. He was discharged on a tapering steroid regimen and remained asymptomatic with no recurrence on follow-up.

Conclusion

This case illustrates the diagnostic challenges of idiopathic hypereosinophilic syndrome, which may mimic common respiratory conditions yet progress to life-threatening complications. Early recognition, exclusion of secondary causes, and timely corticosteroid therapy are crucial. Multidisciplinary management and vigilant follow-up are essential to ensure sustained remission and prevent recurrence.

References

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•Li Z, Chen J, Zeng J, et al.: Application of adenosine deaminase and γ-interferon release assay in pleural fluid for the diagnosis of tuberculous pleural effusion in patients over 40 years old. Infect Drug Resist. 2023, 16:1009-18. 10.2147/IDR.S400838