CASE REPORT

Loeffler's endocarditis with biventricular mural thrombi

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SUMMARY

A 42-year-old man presented to clinic with 1-week history of cough, malaise, loss of appetite and fever. Symptoms started acutely with cough and generalised weakness followed by fever, chills, nausea, vomiting and diarrhoea. He has been healthy before with no chronic illnesses. Further workup disclosed, leucocytosis with 87% eosinophilia, bilateral lung infiltrate, deep venous thrombosis, pulmonary embolism and both right and left biventricular mural thrombi. Extensive workup was done and was consistent with hypereosinophilic syndrome, which explained all his clinical findings. The patient was started on high-dose steroids and responded very well with a complete resolution of symptoms over the course of a few weeks.

BACKGROUND

Hypereosinophilic syndrome (HES) is characterised by eosinophilia that infiltrates peripheral tissues leading to multiorgan failure. HES presentation can be very diverse which makes the diagnosis very challenging. In this case, HES presented as a thrombotic disease involving, the lower extremities, lungs, heart and liver. What makes this case unique is the unusual combination of biventricular mural thrombus with Loeffler's syndrome and multiorgan failure requiring extensive workup to discover the underlying disease. The patient had excellent response to steroids with full recovery in a few months.

CASE PRESENTATION

A 42-year-old man presented to the clinic with a 1-week history of cough, malaise, loss of appetite and fever. The symptoms started acutely with a cough and generalised weakness followed by fever, chills, nausea, vomiting and diarrhoea. He had been healthy before with no chronic illnesses. He denied recent contact with the sick or travel. He also denied any shortness of breath, chest pain or night sweats. Clinically he was febrile with a temperature of 100.8 F, a heart rate of 87 bpm, blood pressure of 120/85 mm Hg, a respiratory rate of 25/min and oxygen saturation of 90% on 2 L oxygen. Giving his acute illness he was admitted to the hospital for a further work-up.

INVESTIGATIONS

Blood work was significant for white blood count of 23×10^3 cells/mm³ (4.5–11.0) with eosinophilia 87% (0.0–6.6%), creatine 2.1 mg/dL, aspartate aminotransferase 140 U/L and alanine transaminase 180 U/L. A chest X-ray showed upper right and left

lung infiltrates. A lung ventilation/perfusion scan showed a high probability of a pulmonary embolism (PE) and a chest CT without contrast showed multifocal lung infiltrates bilaterally (figure 1). Giving the above findings he was started on vancomycin and piperacillin/tazobactam for presumed pneumonia. A Doppler ultrasound of his lower extremities showed extensive bilateral acute deep vein thrombosis. Anticoagulation was initiated using unfractionated heparin. Further workup for eosinophilia including serum HIV antibody, thyroid stimulating hormone, antineutrophil cytoplasmic antibody, tryptase, toxocara and strongyloides antibodies, stool for ova and parasites came back negative. A CT scan of the abdomen and pelvis was unremarkable as well. Bone marrow aspiration and biopsy were performed and showed abundant eosinophil infiltrates with no findings suggesting myeloid maturation or myeloproliferative disorder. Further evaluation of eosinophilia including FIP1-like 1/platelet-derived growth factor receptor α (FIP1L1-PDGFR α) fusion gene test was negative. An echocardiogram showed a mobile mass at the apex of the right and left ventricles. A cardiac MR (CMR) showed a mildly dilated right ventricle with normal left and right ventricular function and preserved ejection fraction (55%). It also showed ill-defined masses in the right as well as left ventricular apices most consistent with thrombus with peripheral fibrosis layering along the endocardial

Figure 1 CT scan of the chest without contrast showing multiple bilateral air space consolidations throughout all lobes of the lungs but more confluent in the bilateral lower lobes.

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Figure 2 Cardiac MRI showing ill-defined masses in right as well as left ventricular apices these findings are consistent with thrombus with peripheral fibrosis layering along the endocardial surface.

surface (figure 2). These findings most likely represent Loeffler's endocarditis. Furthermore, because of gastrointestinal (GI) symptoms and elevated liver enzymes, a liver vascular ultrasound was performed and showed right hepatic vein thrombosis. Based on the extensive eosinophilia and multiorgan involvement, the diagnosis of HES was entertained and confirmed by ruling out other possible diseases.

TREATMENT

All severe cases of eosinophilia with symptomatic leucostasis should be managed emergently with high doses of glucocorticoids; an initial dose of 1 mg/kg/day for a total duration of 1–2 weeks, then tapered down to the lowest effective dose that maintains remission.^{1–3} Patients with positive FIP1L1/PDGFR α fusion gene should be treated more aggressively with imatinib mesylate regardless of symptoms status.^{2 4 5} Our patient was started on high-dose steroids and a few days later his O₂ requirement decreased, and serum eosinophil dropped dramatically to 10%.^{4 6} Kidney and liver function returned to normal.

OUTCOME AND FOLLOW-UP

Eventually, he was started and discharged on oral steroids and warfarin. Three months later, eosinophilia on peripheral blood reduced to 1.1%.

DISCUSSION

HES is a group of disorders that share the presence of eosinophilia that may infiltrate peripheral tissues leading to end-organ damage.^{4 5} HES is seen in young to middle-age patients. Cases with positive FIP1L1-PDGFR α fusion gene are seen predominantly in men, whereas cases with negative FIP1L1-PDGFR α

fusion gene are seen equally in men and women. It can affect multiple systems including the heart, skin, lungs, GI, kidney, blood and the central nervous system.^{6 7} Severity of symptoms varies from slowly progressive to more life-threatening symptoms including cardiac thrombus, PE and stroke. Cardiac involvement is the main cause of morbidity and mortality in patients with HES, with more common occurrence in patients with positive FIP1L1/PDGFR α fusion gene.^{3 7}

Learning points

- ▶ Hypereosinophilic syndrome (HES) is characterised by persistent eosinophilia with multiorgan damage causing diverse clinical presentation.
- ▶ Loeffler's syndrome is a transient respiratory illness associated with blood eosinophilia and radiographic shadowing due pulmonary infiltrates with eosinophilia.
- ▶ Loeffler's endocarditis is a restrictive cardiomyopathy which cause impaired ventricular filling with normal or decreased diastolic volume of either or both ventricles.
- ▶ HES is associated with hypercoagulability with thrombotic events, mechanism of which is unknown.
- ▶ Secondary causes of hyper eosinophilia should be ruled out with extensive work up before assuming a diagnosis of idiopathic HES.

Contributors All authors have seen and approved the manuscript, contributed significantly to the work, and also that the manuscript has not been previously published nor is not being considered for publication elsewhere.

Competing interests None.

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