Polyserositis in Patient with Hypereosinophilic Syndrome

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Introduction:

Hypereosinophilic syndrome encompasses a range of haematological disorders where blood eosinophilia occurs, along with the involvement of at least one organ system, which can be either idiopathic or secondary to autoimmune reactions, neoplasms or infections. The spectrum of presentation of this condition is vast; including cardiomyopathy, gastroenteritis, cutaneous lesions, sinusitis, pneumonitis, neuritis, and vasculitis. With this case, we attempt to understand a few of the many possible manifestations of HES.

Case Report:

Presentation:

A 32 year old male presented to Emergency with acute respiratory distress. He complaint of dyspnea since 2 months, which was NYHA Grade 3 and MMRC Grade 3, and was insidious in onset and was maximum at that time, and continued to be of the same severity for the following 2 weeks, which was reduced in intensity for a while and again worsened one week ago. The dyspnea was not associated with orthopnea, palpitations, cough or chest pain. He also complained of loose stools since 3 days, which were associated with abdominal pain.

On examination: alert, conscious, well-oriented, all vitals were within normal ranges.

Respiratory system: Bilateral normal vesicular breath sounds with bilateral scattered rhonchi were heard on auscultation.

CVS: Loud S1 sound was heard, both S1and S2 heard on auscultation.

Per abdomen: the abdomen was non- distended and diffusely tender on palpation.

CNS: there were no abnormal findings.

Investigations:

Initial blood work up showed eosinophilic predominance (810 /microL absolute count) and inflammatory markers (CRP: 117.39 mg/L) were raised. Troponin(0.043 ng/mL) was elevated, NT Pro BNp (321 pg/ml) was within normal limits and D-dimer(4.5 microg FEU/mL) was slightly raised.

Initial X-ray showed mild pleural effusion, which rapidly worsened. Subsequent X-ray taken 2 days later showed significantly increased effusion.

ECHO: EF: 65%, tachycardia, moderate pericardial effusion on RA/RV side and mild pericardial effusion on LA/LV side. IVC collapse was present. Mild pulmonary hypertension was detected. Myxomatous mitral leaflets with mild regurgitation.

Pleural fluid analysis showed no abnormal cells or parasites; eosinophilic predominance was observed (40%). The fluid was exudative in nature with high protein content.

USG: bilateral gross pleural effusion was detected, along with mild ascites.

CTPA: no evidence of pulmonary embolism, but b/l pleural effusion and pulmonary edema were seen.

Culture reports and HIV testing was negative, ruling out most microbiological etiologies.

Differential diagnosis: Owing to the presence of effusions in both the lungs and the heart, along with a high eosinophil count, fungal infections, malignancies like myelodysplastic syndrome, and drug hypersensitivity were considered as differential diagnosis. In addition, owing to the sudden worsening of the patients dyspnea on admission, acute pneumonitis was also considered.

Diagnosis: Hypereosinophilic syndrome with polyserositis(b/l pericardial and pleural effusions)

Treatment and Results:

In lieu of the elevated markers in the blood workup, Clexane anticoagulant therapy was initiated. The patient was subsequently shifted to ICU in view of increased work of breathing and increase in p/f ratio, where he was placed on NIV. The patient was administered prophylactic antibiotics and diuretics, along with corticosteroids to aid in reducing inflammation.

The patient was discharged once stable with instructions to continue 50 mg Wysolone for 1 month and planned to taper subsequently. The patient was asked to follow-up after 2 weeks.

Discussion:

HES includes a heterogenous and complex group of different subtypes, and though scientific interest in its study has increased in the recent times, health systems are still lacking in terms of pathobiology, phenotyping and personalized treatment, which need to be met with further study.

Conclusion:

Since a wide range of etiologies need to be ruled out before making the diagnosis of HES, which is usually identified as a diagnosis of exclusion, clinicians need to consider this condition as a differential in cases with multisystem involvement or polyserositis in the setting of a high blood eosinophil count.