

INTRODUCTION

- Idiopathic hypereosinophilic syndrome (IHES) is a group of myeloproliferative disorders causing multi-organ dysfunction with unknown etiology
- We report a case of IHES in a patient presenting with gastric wall thickening, multiple organ damage including acute coronary syndrome, encephalopathy, and multiple cortical infarcts.

CASE REPORT

- A 47-year-old male patient with past medical history significant for type 2 diabetes mellitus and hypertension presented with nausea, vomiting, abdominal pain, and food intolerance for 7 days. He also complained of intermittent, substernal chest pain for 1-2 days at presentation.
- His labs were significant for elevated troponin, serum creatinine of 2.85mg/dL, and significant leukocytosis (32.80 10^3 /cmm). Absolute eosinophilic count was elevated at 14.70 10^3 /cmm.
- Echocardiogram showed hypokinetic mid inferior and inferolateral walls.
- CT chest/abdomen angiogram was done for concerns of dissection. It showed diffuse gastric wall thickening with upper abdominal retroperitoneal lymphadenopathy and extensive retroperitoneal fat stranding.
- He was started on antiplatelet agent and heparin drip. Ischemic workup was deferred as troponin plateaued and patient had new-onset altered mental status
- MRI brain showed numerous punctate cortical and subcortical foci of restricted diffusion consistent with acute watershed distribution infarcts.

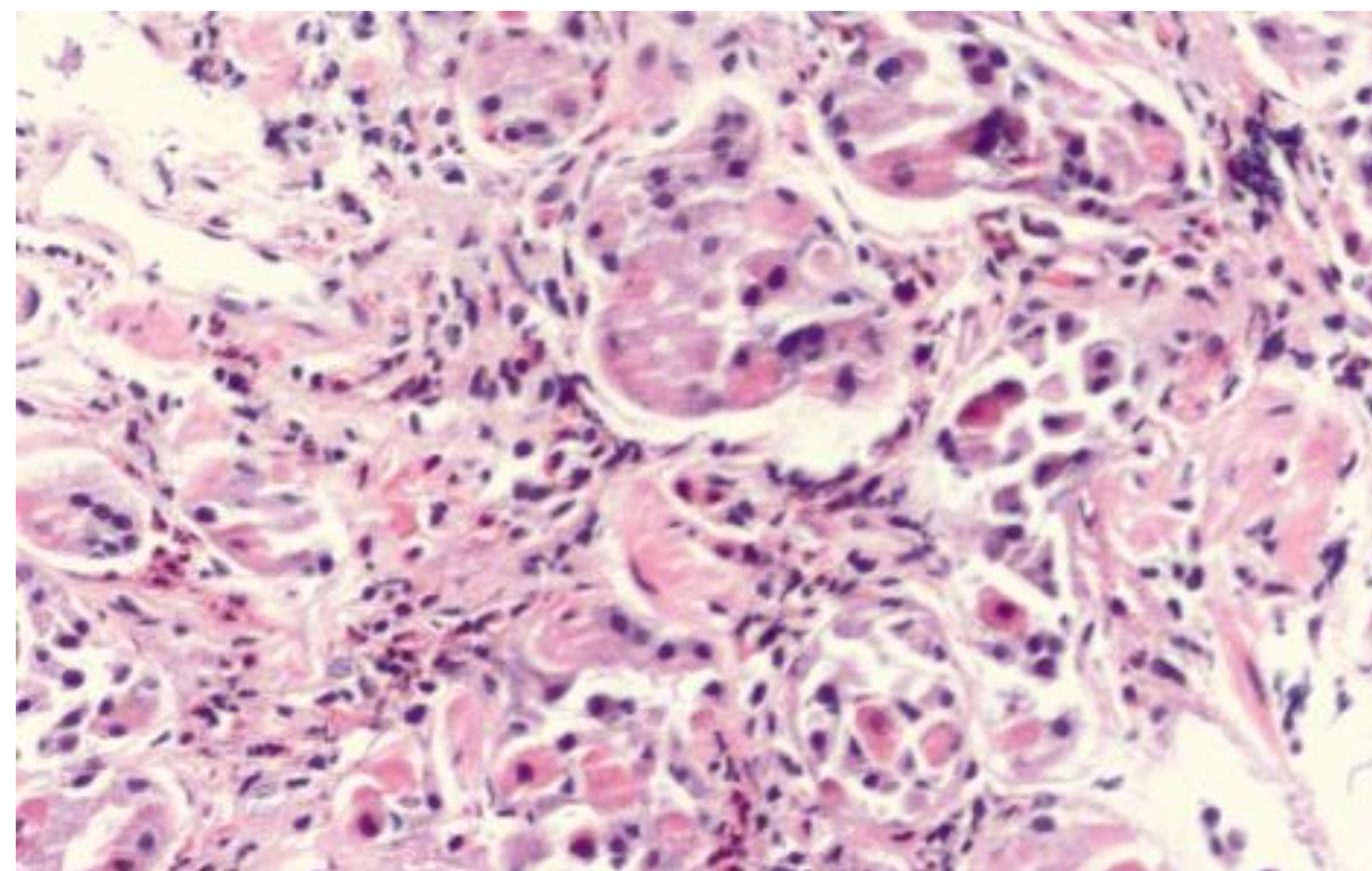
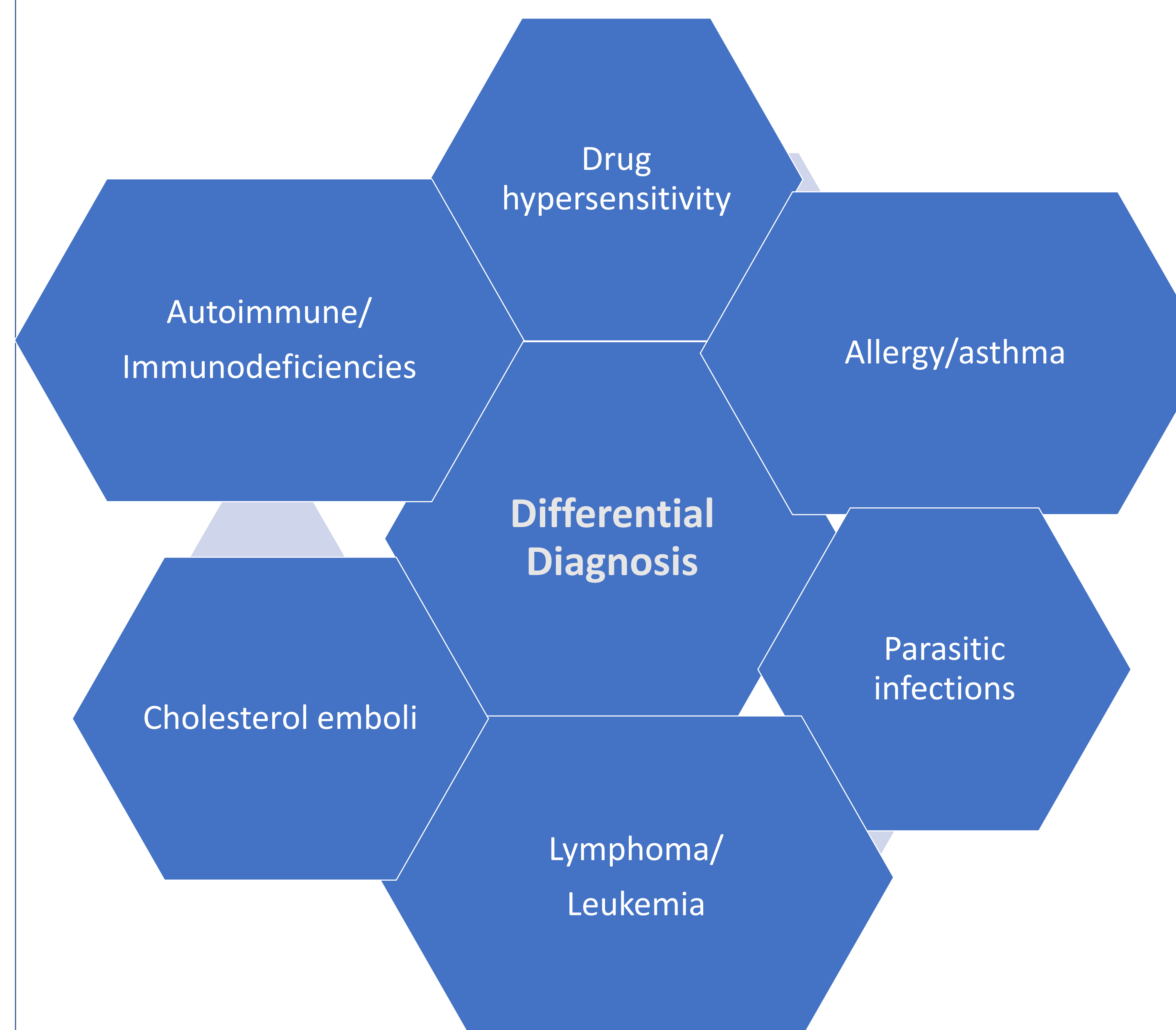


Figure 1- Gastric mucosa with increased eosinophilic infiltration within the lamina propria and focal glandular involvement with eosinophils.



CLINICAL COURSE

- In view of possible need for dual anti-platelet therapy and the high concern for gastric malignancy on CT that required tissue biopsy, gastroenterology was consulted for esophagogastroduodenoscopy (EGD).
- Emergent bedside EGD did not show any gastric mass but diffuse gastritis.
- Biopsy showed mild chronic gastritis and a focal area showing increased eosinophilic infiltration with eosinophilic cryptitis (Figure 1).
- An extensive workup for eosinophilia including infectious etiology returned negative. No secondary causes of hypereosinophilia were identified.

TREATMENT

- Patient was started on 1mg/kg of prednisone with normalization of eosinophils to 0 within 24 hours.
- Steroids were tapered and patient's improved clinically with resolution of altered mental status, abdominal pain, and chest pain.
- Follow-up EGD has been scheduled to re-evaluate eosinophilic deposits.

CONCLUSIONS

- The stomach biopsy showing eosinophilia was the key to diagnosis.
- Resolution of peripheral eosinophilia with steroids confirms the diagnosis in the absence of a secondary cause.
- A repeat EGD with biopsy is scheduled to look for histopathological resolution.

REFERENCES

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