

## Introduction

- **Malaria** is a parasitic infection that often presents as a systemic febrile illness.
- The ***P. falciparum*** species causes the most severe form of disease.
  - **Clinical Features:**
    - **Systemic:** fevers, chills, myalgias, nausea, vomiting
    - **Chemistries:** hypoalbuminemia, transaminitis, acute kidney injury, proteinuria
    - **Hematologic:** normocytic anemia, thrombocytopenia, leukocytosis/leukopenia, coagulopathy (labs often positive for disseminated intravascular coagulation)
    - **Gastrointestinal:** diarrhea, GI bleeds, splenic rupture, subacute intestinal obstruction
- We report a severe case of *P. falciparum* malaria complicated by a GI bleed

## Clinical Course

- During the next 12 hours, the patient rapidly deteriorated with large coffee ground emesis and melena
- Hemoglobin dropped 12.1 to 8.6; platelets dropped 135 to 15

- Hemolysis labs were positive: haptoglobin < 30, LDH 607, reticulocyte 14.6%, fibrinogen 112
- Patient became encephalopathic; LP with mildly elevated protein otherwise normal, MRI Head nonremarkable, EEG negative for focal slowing, epileptiform discharges or seizures

- Abdominal ultrasound and CT showed no acute intra-abdominal pathologies; Chest X-Ray nonremarkable
- Upper Endoscopy showed Grade-C esophagitis, an oozing duodenal ulcer and hematin all throughout the stomach wall

- With new ongoing hematemesis and melena, he went into progressive septic vs hemorrhagic shock and required vasopressors
- Stress-dose steroids were administered

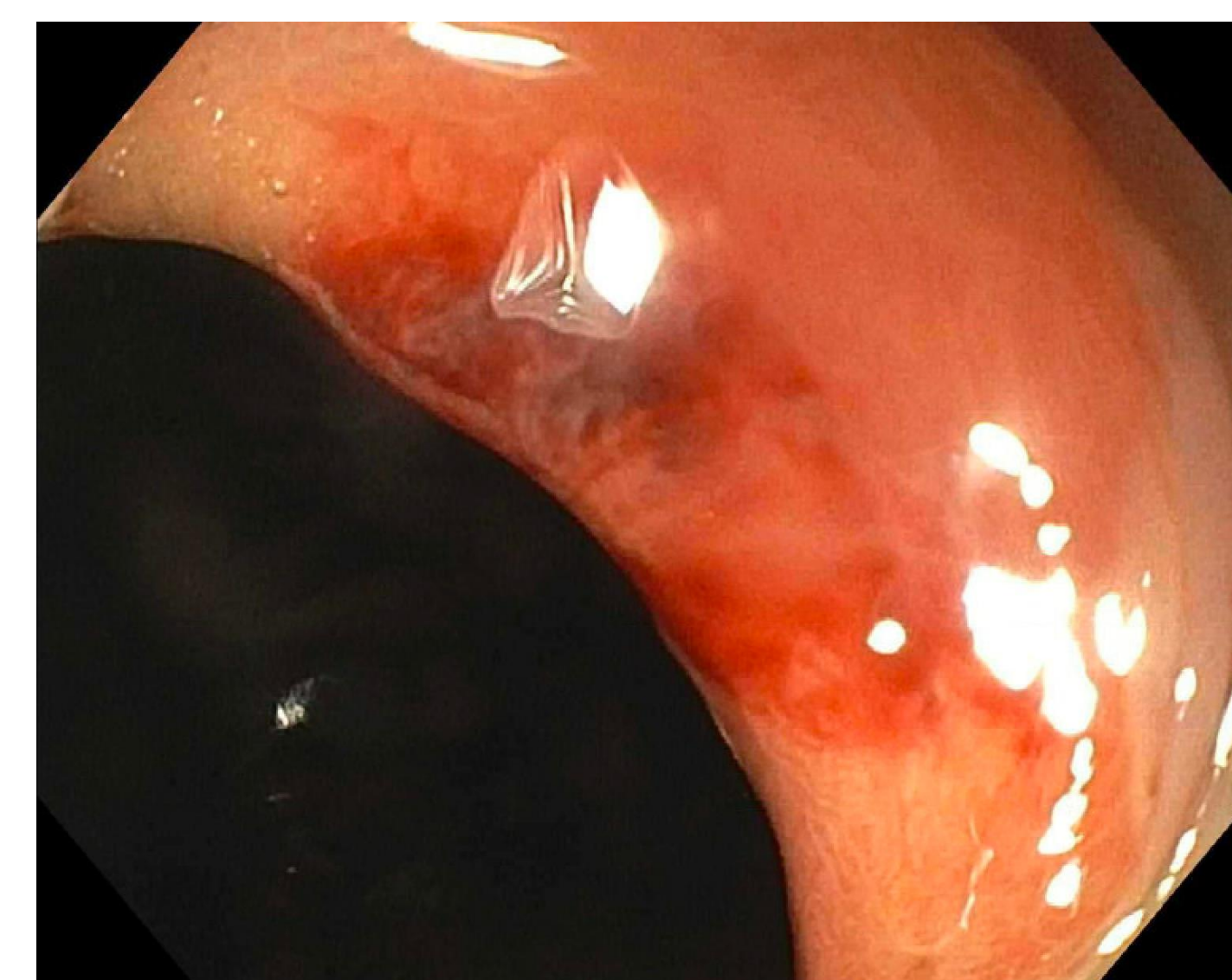
## Discussion

- Despite aggressive measures, our patient ultimately developed shock and died
- The hematologic manifestations often seen in malaria rarely lead to massive GI bleeds, and there is still much to be understood about the pathogenesis of this disease
- Our patient did not use NSAID medications, he was not tested for H. Pylori, his baseline hemoglobin was unknown and despite the stress of his acute severe illness, he had no other risk factors for duodenal ulceration and a GI bleed
- High levels of free oxygen radicals and tumor necrosis factor may have played a role in the etiology of his peptic ulcer formation
  - Malarial hemolysis results from the release of cytokines from schizont parasites and macrophage recruitment.
  - These processes cause a cytokine storm and lead to the activation of Endothelial Adhesion Molecule Type-1 and E-Selectin
  - This enhances the cytoadherence of parasitized cells, mediating lactic acidemia, shock, diffuse gut mucosal damage, increased permeability and eventually disseminated intravascular coagulation

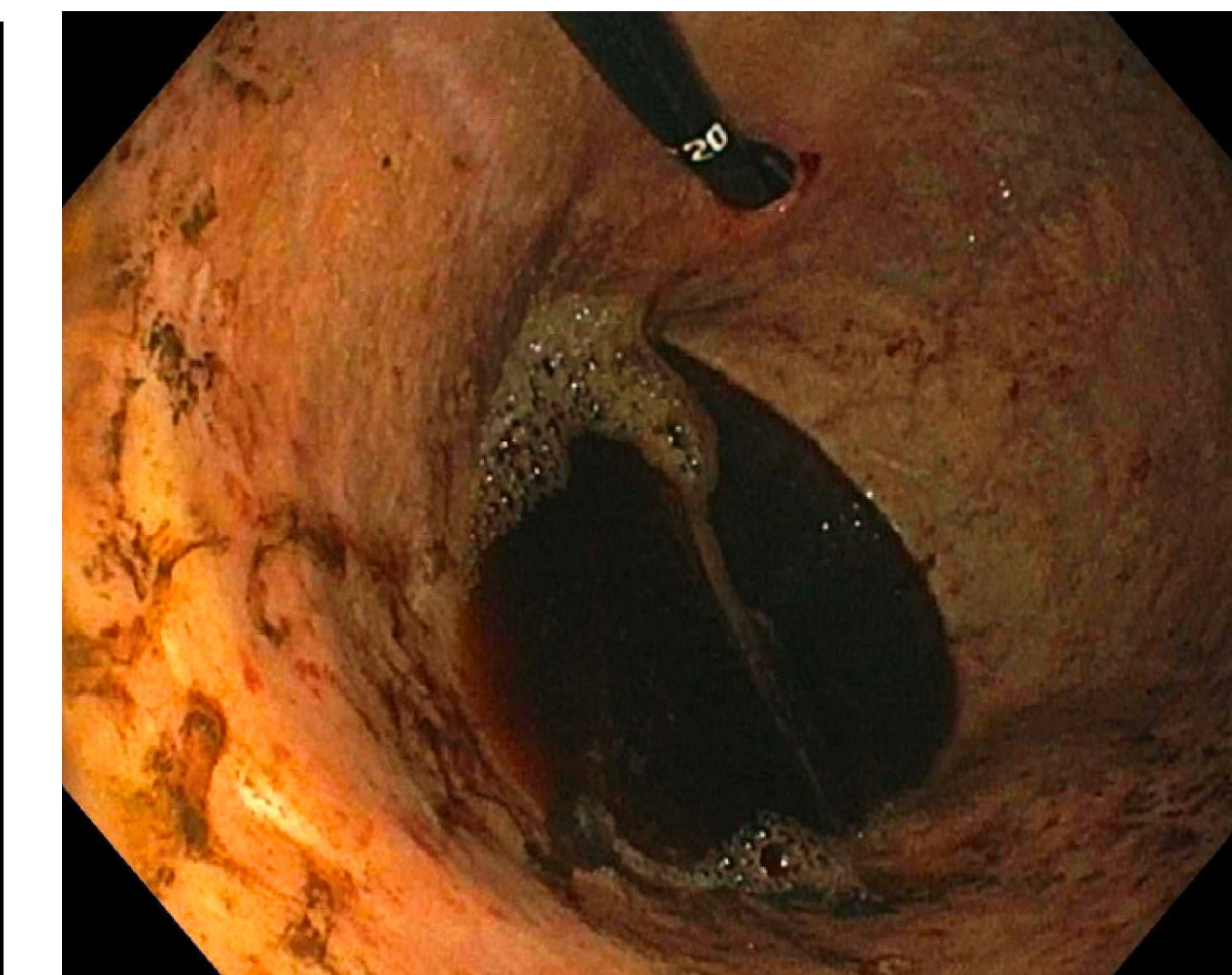
## Patient Presentation

- 66-year-old male with hypertension and recent travel to Nigeria presented for altered mental status, abdominal pain and 25 pound weight loss in the last month.
- Soon after arrival to the ER, our patient spiked a fever to 102F.
- Infectious work-up:
  - LP positive for many RBCs, glucose 81 and protein 47; CSF was negative for meningitis/encephalitis
  - Did not test for EBV, CMV or HIV
  - Blood and fungal cultures were negative
- Due to his recent travel history, our patient had a 5-thin and 2-thick malarial smear which showed ***P. falciparum* malaria ring forms**
- Started on empiric Cefepime, Vancomycin, Metronidazole, Atovaquone and Proguanil

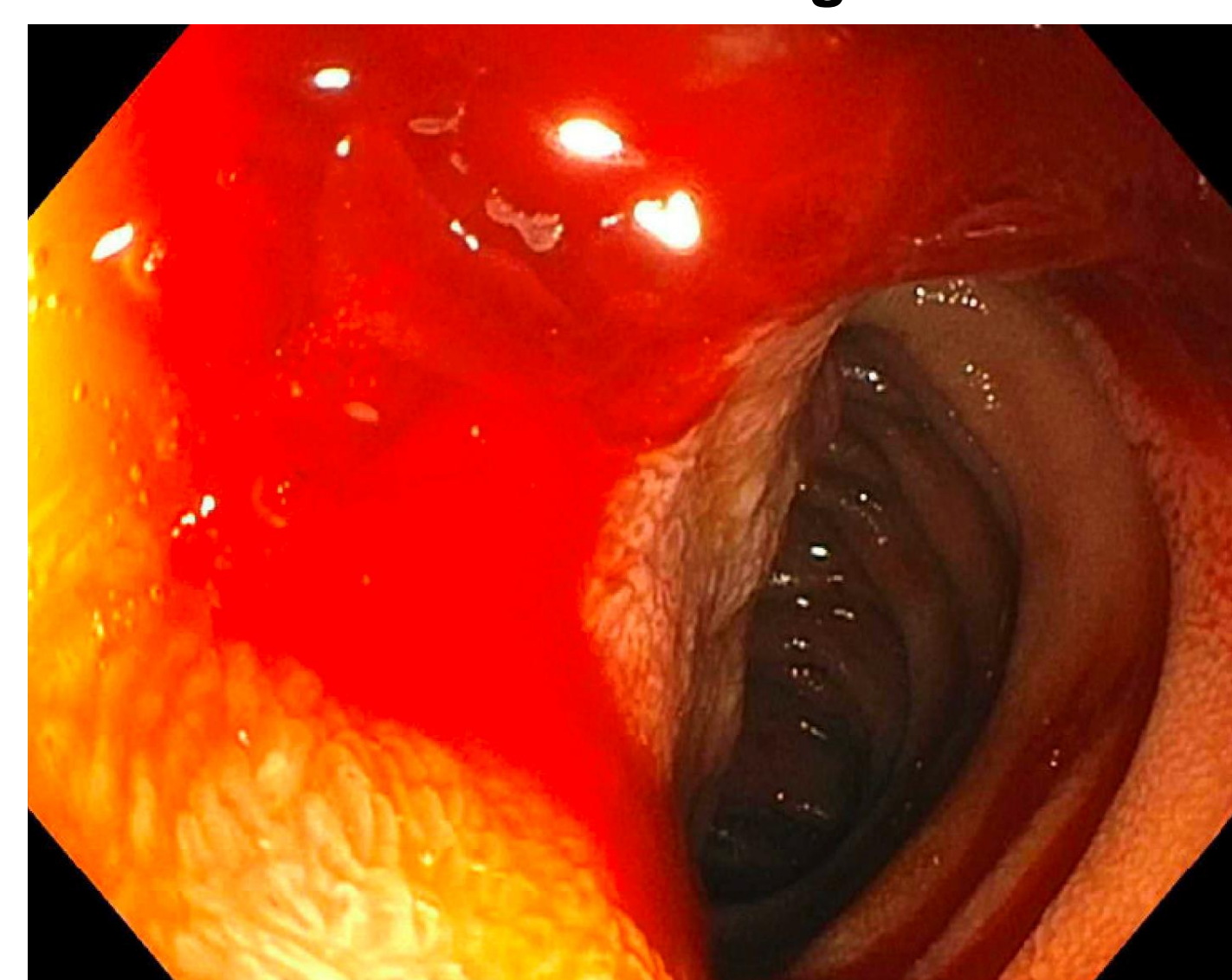
## Upper Endoscopy Imaging



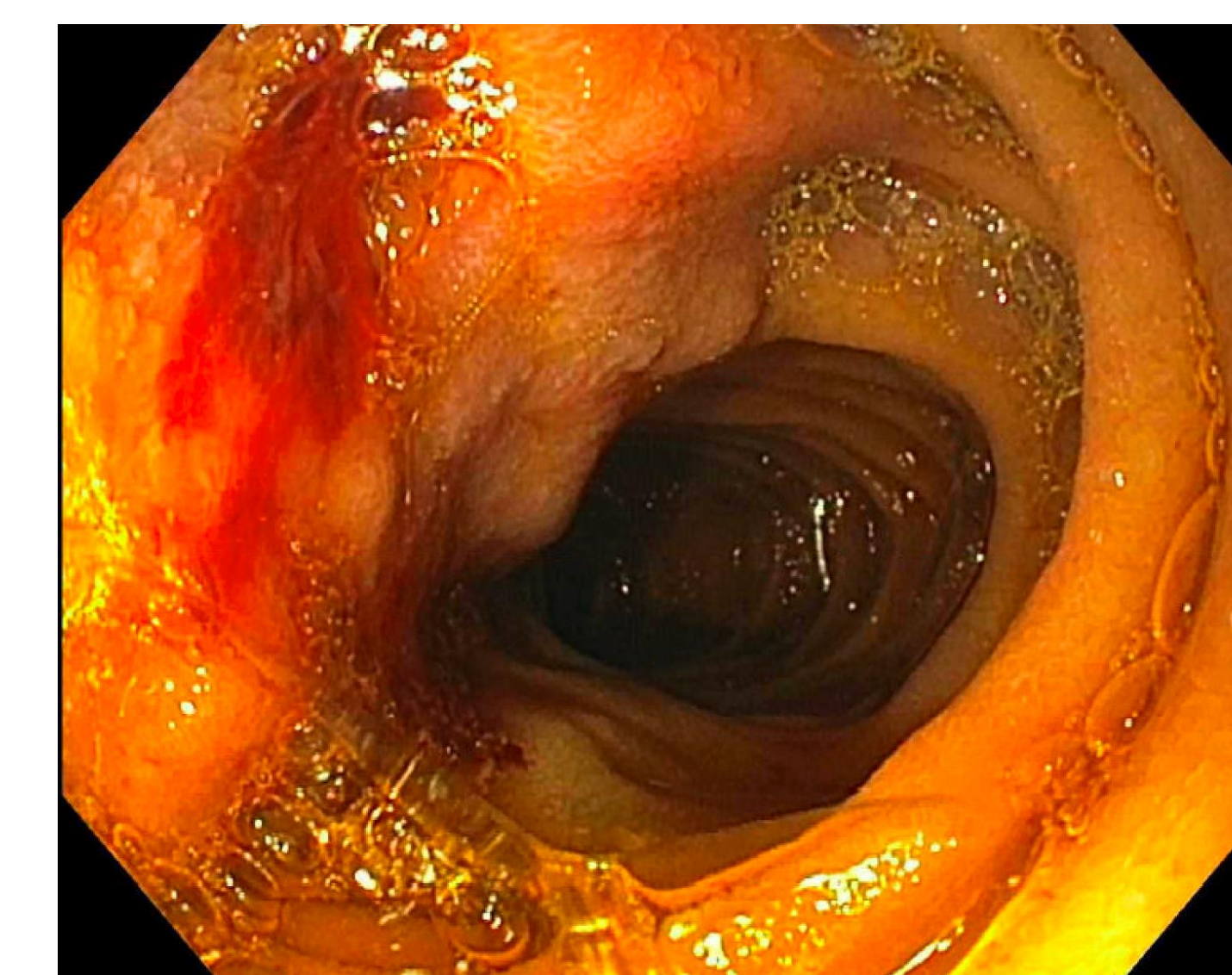
Grade-C Esophagitis in distal esophagus with evidence of recent bleeding



Hematin (altered blood/coffee-ground-like material) in the entire stomach



Oozing duodenal ulcer with oozing hemorrhage (Forrest Class Ib)



## Conclusion

- The hematologic manifestations often seen in malaria rarely lead to massive GI bleeds, and there is still much to be understood about the pathogenesis of this disease

## References

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