A Bloody Mystery: When Subarachnoid Hemorrhage Masquesrades as a Gi Bleed

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***Abstract*—Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening condition typically associated with sudden-onset severe headache, neurological deficits, and altered consciousness. However, atypical presentations, including gastrointestinal (GI) bleeding, are less commonly reported but can complicate the clinical course.**

We present a case of a patient with aSAH who initially presented with an upper GI bleed, demonstrating the challenges in diagnosing this condition in the absence of classic neurological symptoms.

The patient's presentation included hematemesis, which were initially attributed to a primary gastrointestinal etiology. However, subsequent imaging and neurological assessment revealed the presence of an aneurysmal subarachnoid hemorrhage, highlighting the importance of maintaining a broad differential diagnosis when faced with unusual combinations of symptoms.

This case underscores the need for careful evaluation of both neurological and gastrointestinal symptoms in patients with unexplained upper GI bleeding, as early recognition and intervention are crucial for improving outcomes. Furthermore, it emphasizes the complex interplay between the central nervous system and gastrointestinal tract, particularly in the setting of vascular events such as aSAH.

***Index Terms*—aneursymal, hemorrhage, vomiting, headache, gastroenterologist, coiling**

I. INTRODUCTION

47-year-old female was brought to emergency room with history of hemetemesis 4 episodes since an hour, dark colour blood around 50ml in each episode of vomiting.

No history of pain abdomen,no abdominal distention,no jaundice,no drug intake, no history of chronic liver disease, no habits

No previous comorbidities, not on any blood thinners

Primary survey
Airway-patent
Breathing-RR of 24 cpm
Spo2-98% Room air
Circulation
Bp of 110/80 mmhg
Pulse-88 /min
All peripheral pulses well felt

DISABILITY:
GCS 15
Pupils 3mm B/L reacting to light
GRBS-119mg/dl
Exposure: -temp of 98F

ADJUNCTS
USG abdomen
no organomegaly
fatty liver grade 1
no features suggestive of chronic liver disease

venous blood gas-hb-11.2mg dl, lac-4, serum electrolytes normal,ph=7.36, screat=1.0

UGI SCOPY –no bleeding varices or blood or ulcers
COLONOSCOPY-no hemorroids .no fresh or altered blood seen
CT ABDOMEN- was also done which was normal to look for other causes
CT BRAIN -anuersymal sah

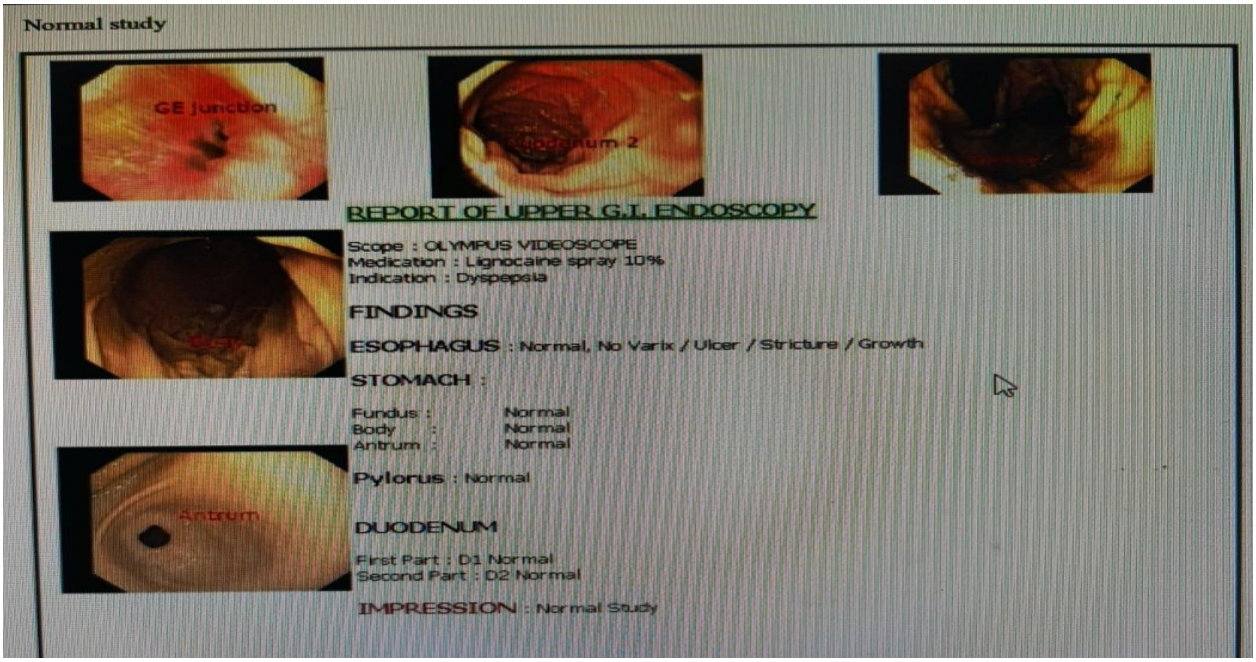
II. SECONDARY SURVEY

Per abdomen-abdomen soft,no abdomen distention,or spider naevi
Per rectal-no blood seen

Cns-patient consious oriented moving all 4 limbs.

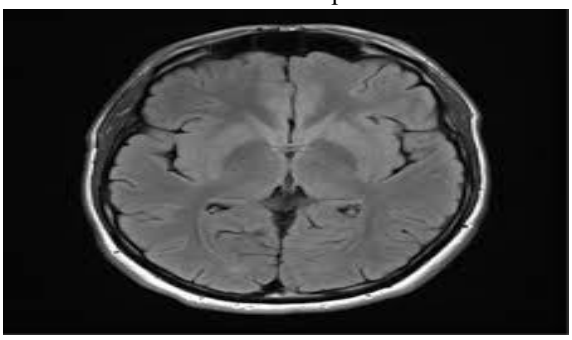
Pupils 3mm bilateral reacting to light
GPE: -no clubbing,no flaps, no tremors
Cardiovascular and Respiratory examination was within normal limits

Evaluation; -pt was conservatively managed in the ED with fluids, kept nil by mouth, started on injection pantaprazole after bolus dose, inj ocreotide, antiemetics, gastroenterology opinion was taken, antibiotics, arranged blood and planned for OGD scopy and was shifted to ICU
OGD scopy turned out to be normal.



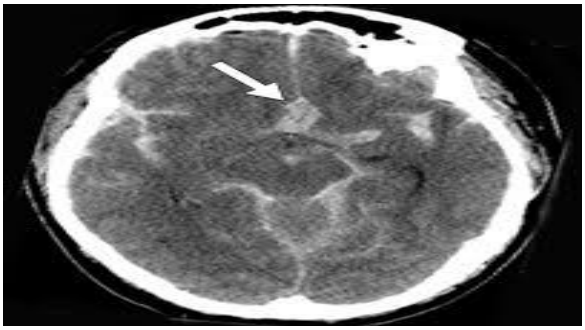
Colonoscopy was done which also was normal.
CT abdomen was done to look for other causes of hemetemesis which dint reveal any findings

Patient complianed of headache 2 hours after presentation,which was thought due to dehydration so inj paracetomol was given and the headache subsided.
after few hours patient compliants of left lower limb numbness along with headache,
power of 5/5 in all 4 limbs .no blurring of vision.
Mri brain was done and was reported normal.



Patient continued to have headche and this was severe with neck stiffness and projectile vomiting with blurring of vision, blood pressure of 140/80 mmhg

Fundus examination revealed vitreous hemorrahge
CT brain was done and diagnosed to have anuersymal sah



Tab nimodipine 60 mg was started and neurosurgery consultation was given following which inj 3%saline @10ml/hr was started. patient was taken to OT and coiling was done

Mean arterial pressure of 110 mmhg was maintained

III. CONCLUSION

SAH has a clinical presentation that varies from a simple headache to obvious neurological deficits. In addition, it is a rare condition which many emergency and primary care physicians may encounter infrequently even over many years of practice

Many such instances where CT brain done within 6 hours after symptom onset failed in which one patient with only neck pain who had an inconclusive CT; was diagnosed to have bleeding from a cervical arteriovenous malformation

Despite improvements in diagnostic imaging, CT may be less sensitive in SAH patients presenting with so-called “minor leaks” or normal neurological status

SAH is an essential emergent diagnosis with high morbidity and mortality requiring prompt recognition. Diagnosis is made by CT and, if non-diagnostic and/or more than 6 h have passed, lumbar puncture to evaluate for xanthochromia if there is a high enough clinical suspicion

Once a diagnosis of SAH is made, elucidating the cause will guide management. Traumatic SAHs can generally be monitored with or without seizure prophylaxis, but in a non-traumatic SAH, evaluation should include CT angiography to evaluate for intracranial aneurysms. The cornerstone of management for all SAHs is blood pressure control, which has shown to prevent re-bleeding and ischemic damage and improve mortality

Early consultation with neurosurgical services for definitive aneurysm management should occur in the emergency department.

As always, high-quality supportive care should be initiated, which includes avoiding hypoxia and hyperglycemia, reversal of and/or withholding anticoagulants, and airway management if mental status continues to deteriorate.

If patients are able to tolerate oral medications, nimodipine has been shown to improve neurologic outcomes.

In atypical presentations such as this one, it is important for clinicians to take a step back and consider the case as a whole as well as their own cognitive processes

Limitations of this case are, of course, the solitary patient and inability to draw conclusions regarding the epidemiology of SAH presentation. However, as this case illustrates, the diagnosis of SAH is not nearly as cut-and-dry as a maximal onset thunderclap headache. Clinicians should be careful to judge the history of present illness, risk factors, and perform thorough, detailed, and repeated physical exams in an unclear picture.

Asking the simple question “what else might this be?” is an important part of identifying unusual presentations of disease, particularly those with catastrophic courses.

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