Intraduodenal Hematoma after Therapeutic Upper Endoscopy Causing Acute Pancreatitis

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Case

A 65-year-old female presented to the emergency department (ED) for right-sided conjunctival hemorrhage, weakness and fatigue. She had a history of large, infiltrating ductal carcinoma of the breast diagnosed in 2011, when she underwent a right mastectomy with 6 of 9 positive lymph nodes. A right-sided tissue expander was placed, but reconstructive surgery was never performed. No chemotherapy was performed, as the patient sought out an alternative medicine doctor, who treated her with intravenous zinc and other unconventional therapies. Years later, she was diagnosed with metastasis to the liver, bone, spine, and lungs. Other pertinent past medical history included chronic renal insufficiency.

The patient had an allergy to penicillin. She denied any current medications except for the occasional over-the-counter ibuprofen or naproxen, which she took for varied aches and pains. She denied a history of smoking or alcohol intake.

Initial laboratory values performed in the ED found the patient to be anemic with a hemoglobin (Hgb) of 6.6 g/dL. Additional laboratory values include a white blood count (WBC) of 4.9 K/uL, mean corpuscular volume (MCV) 103.7, platelet count (Plt) 51 K/uL, AST 41 U/L, alkaline phosphatase 159 U/L (nml 35-105), INR 1.1, PTT 46 seconds (nml 25-35) and a reticulocyte count of 4.5% (nml 0.5 to 1.5). History was negative for melena or bright red blood per rectum. Fecal occult blood was positive in the ED. The patient’s hemoglobin responded appropriately to 9.7 g/dL, after transfusion of two units of packed red blood cells (PRBC).

Gastroenterology was consulted, and an esophagogastro-duodenoscopy (EGD) and colonoscopy were performed on hospital day #2. EGD showed mild gastritis and was otherwise normal. Cold biopsies were taken of the antrum, duodenal bulb, and the second and third portions of the duodenum. Colonoscopy was negative for bleeding, polyps, diverticula, or any mucosal abnormalities. No biopsies were taken during the colonoscopy. The gastric biopsies later returned negative for Helicobacter pylori. The duodenal biopsies were negative for blunting of villi and for intraepithelial inflammatory cell infiltrates. On hospital day #3, that patient was discharged home in stable condition.

One day after discharge, the patient returned to the ED with nausea, vomiting, and epigastric abdominal pain. She denied any overt bleeding such as melena or bright red blood per rectum. She did report several episodes of coffee ground emesis. Lab values were as follows: Hgb of 6.6 g/dL. WBC 6.6 K/uL, Plt 51 K/uL, creatinine (Cr) 1.3 mg/dL, lipase 64 U/L, bilirubin 0.7 mg/dL.

A second EGD was performed on hospital day #2. Oozing of blood was noted coming from the previous cold biopsy sites at the fundus, body, and duodenum (Figure 1). Nearly a liter of dark, yellow fluid was suctioned from the stomach. Per the endoscopy report, duodenal edema with possible duodenal obstruction was noted. Within the duodenum, several large clots were removed with the endoscope. Active oozing was seen along a biopsy site. The biopsy site was 1 to 2 cm downstream from the papilla, along the inferior, posterior border of the 2nd portion of the duodenum. Four mL of 1:10,000 epinephrine was injected at the bleeding biopsy sites, followed by bipolar electrocautery. Given the amount of blood loss during the endoscopy, 2 units of PRBC and 2 units of platelet were transfused.

On hospital day #3, the patient continued to have epigastric pain, nausea and vomiting. A lipase level that morning was greater than 600 U/L. Aggressive fluid resuscitation with lactated ringers at 200 mL/hour was initiated. Computed tomography scan (CT) of the abdomen and pelvis was performed on an urgent basis. No intravenous contrast was given, as the patient’s glomerular filtration rate was 47 (Normal >59). The CT scan (Figure 2) showed an 8cm hemorrhage, within wall of the second and third portion of the duodenum. No free intraperitoneal air was seen, yet there was concern of a small perforation given a small amount of extraluminal hemorrhage visualized on the scan. Numerous hepatic metastases and extensive metastatic osseous disease were noted. Incidental cholelithiasis was noted as well. Given the CT scan findings, the patient was made NPO, and intravenous levofloxacin and metronidazole were initiated.

A hematology consult contributed the anemia to the intraduodenal hematoma, malignant bone marrow infiltration, and increased consumption of red blood cells (RBC). It was thought the thrombocytopenia and increased RBC consumption were from low-grade disseminated intravascular coagulopathy (DIC). Three doses of desmopressin were given as well as blood product support to keep the platelets above 100K/uL, and to
keep the hemoglobin above 8 g/dL. Over the next 8 hospital
days, an additional 4 units of platelets, and 3 units of PRBCs
were given to meet these parameters.

The patient became progressively more jaundiced, and the
bilirubin continued to rise by approximately 1-2 mg/dL a day.
On hospital day #6, labs were as follows: Total bilirubin 9.4,
direct bilirubin 7.3, AST 106, ALT 47, alkaline phosphatase
232, lipase 117, Hgb 6.6. Magnetic resonance cholangio-
pancreatography could not be performed secondary to the metal
in the previously placed breast tissue expander. Endoscopic
ultrasound was not available at our institution. Transabdominal
ultrasound of the right upper quadrant showed multiple masses
in the liver consistent with metastatic disease. No biliary ductal
dilation was noted. There were small gallstones and sludge
noted in the gall bladder, but no evidence of cholecystitis.

On hospital day #11, the patient developed numbness along the
left side of her face, as well as a left-sided facial droop. A non-
contrast CT scan of the head showed no evidence of acute
hemorrhage or focal mass. A heterogeneous lytic appearance of
the skull, consistent with metastasis, was demonstrated. Labs
were as follows: Bilirubin 19.8, AST 187, ALT 54, alkaline
phosphatase 513.

Treatment options were discussed in detail with the patient and
her family. Given her advanced cancer and poor prognosis, the
patient decided not to proceed with any further intervention or
treatment. The patient was discharged on home hospice.

Discussion

Intraduodenal Hematoma (IDH) were first described in the
medical literature in 1838.1 IDH are most common in the
pediatric population, and are most commonly secondary to
blunt abdominal trauma.2 The 2nd and 3rd portion of the duodenum has a relatively fixed retroperitoneal position, and is
adjacent to the vertebral spine. This makes the 2nd and 3rd portion of the duodenum more prone to shear injury, and to
the development of hematomas after abdominal trauma.2,4 A rich
submucosal vascular plexus, and the lack of a well-developed
serosal layer in the retroperitoneum, likely increase the risk of
hematomas as well.2,4 Of note, spontaneous, non-traumatic
IDH's have been reported in patients with coagulopathies and
in patients on anticoagulation.5

IDH following various endoscopic procedures have also been
described. Cold biopsies of the duodenum taken during upper
endoscopy have been reported as a cause of IDH in several
reports.6,7 IDH after endoscopic retrograde cholangiopan-
creatography (ERCP) has been reported.8 This therapeutic
ERCP included a sphincterotomy, balloon dilation and extrac-
tion of gallstones. Also, IDH has been reported after submu-
cosal injection of epinephrine into the base of a duodenal ulcer.9-12 Risk factors for IDH after endoscopic procedures
include underlying coagulopathies, recent use of anticoagu-
ulants, thrombocytopenia, and liver cirrhosis.13

IDH should be suspected in the differential diagnosis of upper
abdominal pain, nausea, and vomiting occurring after
endoscopy. Occasionally, a drop in hemoglobin is noted post
procedure. CT scan is usually the best method of diagnosis,
and it also can be used to rule out the dreaded complication of
intestinal perforation.

Complications of IDH can lead to perforation, intraluminal
bleeding, as well as pancreatitis.14-16 Pancreatitis is thought to
occur from ampullary obstruction and compression from the
hematoma.10 Mass effect from the hematoma can lead to gastric
outlet obstruction and proximal duodenal obstruction.

Treatment is typically conservative with nasogastric tube
placement and bowel rest. The hematoma typically resolves
within a period of weeks. Total parenteral nutrition has also
been recommended when patients are NPO over prolonged
periods of time. Surgical drainage has been described in more
urgent cases of ongoing bleeding and instability.17 Drainage
under ultrasound or CT guidance are also potential options.
There is one case reporting successful intraluminal endoscopic
drainage of the hematoma.18

The true incidence IDH after endoscopic procedures is un-
known, given it is a rare complication. In a pediatric population
at a single institution, approximately 2500 upper endoscopies
with duodenal biopsies were performed over a 5-year time
period. Two cases of IDH were documented in their population.
The authors thus estimated the incidence of IDH as possibly as
high as 1 in 1250.6

Zinelis et al19 suggested that extending the forceps greater than
3cm beyond the tip of the endoscope to grasp mucosa could
cause tenting of the mucosa, possibly tearing submucosal
vessels. As a way to decrease the chance of causing IDH, these
authors suggested not extending the biopsy forceps >2-3 cm
past the tip during biopsies. Authors have also recommended
taking cold biopsies well away from the major ampulla, in order
to decrease the risk of pancreatitis after IDH.

Conclusion

Intraduodenal hematomas secondary to endoscopic procedures
are a known, yet rare, complication following endoscopy.
Physicians need to be cognizant of this rare, yet potentially fatal
complication.
Figure 1. (Left picture shows oozing/bleeding in the 2nd portion of the duodenum. Middle picture shows previous biopsy site after blood clots removed. Right picture shows biopsy site after injection and cautery)

REFERENCES


