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Life-threatening Upper Gastrointestinal Bleeding Due to Ruptured Gastroduodenal Artery Aneurysm in a Child

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Abstract: Gastroduodenal artery (GDA) aneurysm is a rare cause of massive life-threatening upper gastrointestinal (UGI) bleeding in children. Prompt resuscitation with fluids, administration of large amount of blood products (massive transfusion), prompt diagnostic evaluation using computed tomography (CT) angiography or digital subtraction angiography (DSA), and therapeutic endovascular or catheter-based interventions are life-saving. In cases with failed endovascular interventions, open surgical approach to ligate aneurysm is required. We report a 10-year-male with life-threatening UGI bleed due to ruptured GDA aneurysm possibly secondary to sepsis requiring resuscitation, massive transfusion, CT angiography and DSA, endovascular intervention, and ultimately surgical management with good outcome.

Key Words: gastroduodenal artery, upper GI bleeding, visceral artery aneurysm

Upper gastrointestinal (UGI) bleeding may present with life-threatening complications in children. Most common causes are gastritis, esophageal varices, coagulopathy, and rarely arterial aneurysmal rupture. Arterial aneurysm can be pseudoaneurysm (lined by a layer of blood clot and fibrin) or true aneurysm (contain all 3 vessel wall layers). True aneurysms develop due to congenital vessel wall abnormalities, collagen disorders, or atherosclerosis. Pseudoaneurysms result from damage to vessel wall secondary to trauma, infections, inflammation, vasculitis, or iatrogenic (post endovascular or surgical procedures).[1]

Visceral arterial aneurysm (VAA) has an incidence of 0.02–0.2% in adult autopsy studies.[2] The common sites of VAA are splenic (32–44%), renal (17–38%), coeliac trunk (8–19%), hepatic (4–16%), superior mesenteric (SMA) (5–7%), and gastroduodenal arteries (GDA) (1–4%).[2,3] We report a case of 10-year-old male with life-threatening UGI bleed due to ruptured GDA aneurysm managed with endovascular procedures and surgery with good clinical outcome.

CASE REPORT

A 10-year-old male presented with papulovesicular skin lesions for 10 days and fever for 4 days. The lesions started over hands and then progressed to involve lower abdomen, perineal areas, and buttocks. One day before admission, he developed altered sensorium and one episode of malena.

At ER, his respiratory rate was 32/min, heart rate 173/min, capillary refill time 4 seconds, blood pressure 90/50 mm Hg, and SpO_2 of 93% in room air. He also had poor peripheral pulses and severe pallor. Crusted pus discharging lesions were noted over both hands (more in web spaces), lower abdomen, perineal area, and buttocks (Figure 1). Neurological examination revealed Glasgow Coma Scale score of E4M4V4, generalized hypotonia, elicitable deep tendon reflexes, flexor plantar reflex, no cranial nerve palsies, and absent meningeal signs. Other systemic examinations were normal.

Initial possibility of infected scabies, septic shock, disseminated intravascular coagulation (DIC), and UGI bleeding was considered. He was resuscitated with normal saline boluses (20 mL/kg, 2 boluses); intravenous infusion of adrenaline (0.3 μg/kg/min), noradrenaline (0.4 μg /kg/min), vasopressin (0.0012 IU/kg/min), dobutamine (20 μg/kg/min), packed red blood cells (PRBCs) transfusion (2 units over 4 hours), intravenous antibiotics (ceftriaxone and cloxacillin), and mechanical ventilation. He was shifted to pediatric intensive care unit (PICU) within 6 hours of admission. He developed acute kidney injury (AKI) on day 2 for which he was started on peritoneal dialysis. He had deranged liver function tests (Table 1). With gradual improvement in clinical status, inotropes were tapered and stopped by day 4 and was extubated on day 6 of PICU stay.

On day 7, he developed severe anemia, hypotensive shock and multiple episodes of massive malena for which he was resuscitated with fluid boluses (40 mL/kg normal saline), PRBCs transfusions, and vasoactive drugs (adrenaline 0.3 μg/kg/min, noradrenaline 0.3 μg/kg/min and vasopressin 0.0001 IU/kg/min). UGI endoscopy revealed a bleeding lesion in the first part of duodenum (Figure 2). Computed tomography angiography (CTA) confirmed GDA pseudoaneurysm (Figure 3A and B). Digital subtraction angiography (DSA) guided coil embolization (Figure 3C and D) transiently controlled bleeding. But, after 12 hours of coiling, he again developed massive episodes of malena requiring PRBC transfusion. Second endovascular cyanoacrylate glue embolization was done in bleeders from tributaries of GDA on day 8. Child continued to have life-threatening UGI bleeding requiring massive PRBC transfusion. He received 190 mL/kg of PRBC, 110 mL/kg of fresh frozen plasma (FFP), and 110 mL/kg platelet concentrates (PCs) as per massive transfusion protocol over 48 hours. His amylase levels, ANA, ANCA, HBsAg, HCV, and HIV reports were normal

On day 9, he underwent laparotomy, duodenostomy, and ligation of GDA aneurysm. UGI bleeding stopped after surgery, inotropes, and respiratory support were tapered, and he was extubated on day 11 of admission. He developed critical illness neuromyopathy and was discharged after 25 days. At 6 months follow-up, he was ambulatory with no recurrence of symptoms.

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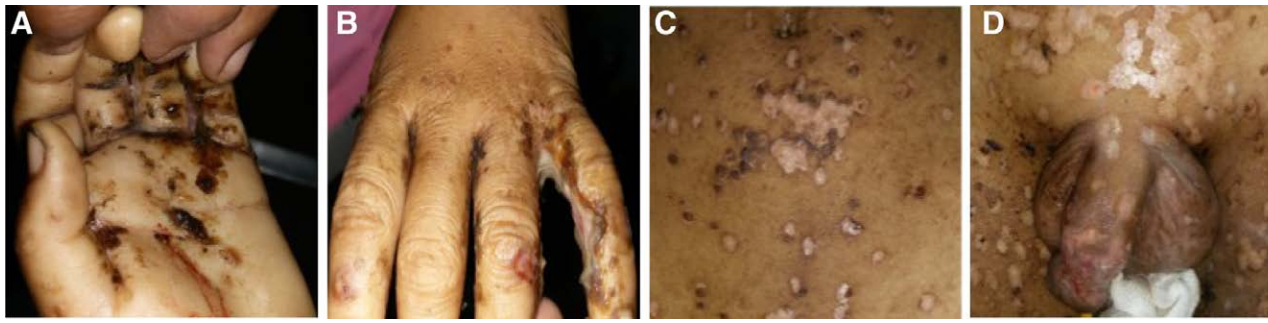

FIGURE 1. Skin lesions over hands (A and B), lower abdomen (C), and perineum (D).

TABLE 1. Hematological and Biochemical Parameters of the Child During Hospital Stay

Analytical Parameters	Day 1	Day 3	Day 7	Day 8	Day 9	Day 10	Day 14
Hb (gm%)	3.9	10	5.4	3.2	5.4	10	9.4
TLC (per cumm)	49400	18560	8130	24000	14150	24000	8290
DLC							
Neutrophils (%)	82	93	90	78	79	90	81
Lymphocytes (%)	12	4	7	12	11	7	11
Platelet (per cumm)	124,000	41,000	99,000	150,000	167,000	160,000	178,000
PT (s)	34.6	15.5	19.6	19	19.6	19	15.6
PTI (%)	40	89	71	73	71	70	90
INR	1.42	1.2	1.36	1.37	1.4	1.4	1.1
aPTT (s)	43	32.2	39.9	32.6	34.9	34	34.4
SGOT (IU/L)	317		59	38			63
SGPT (IU/L)	464		209	86			53
Bilirubin (mg%)	0.57		0.4	0.4			0.7
Total protein (gm%)	5.4		5.2	5.1			4.3
Albumin (gm%)	2.8		2.6	2.5			2.1
Sodium (meq/L)	138		144	138	143	151	148
Potassium (meq/L)	4.2		4.3	3.9	3.2	4.9	4.7
Urea (mg%)	80		49	63	100	88	66
Creatinine (mg%)	0.9		0.4	0.3	1	0.9	0.6

aPTT indicates activated partial thromboplastin time; DLC, differential leukocyte count; Hb, hemoglobin; INR, international normalized ratio; PTI, prothrombin time index; PT, prothrombin time; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; TLC, total leukocyte count.

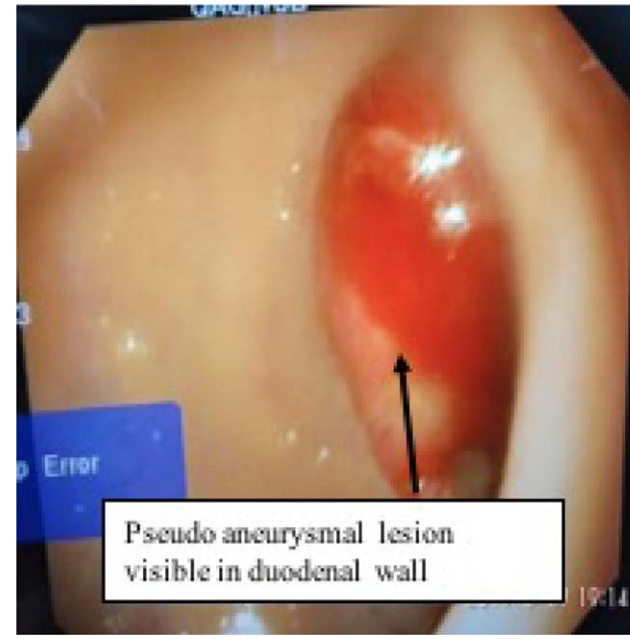

FIGURE 2. Upper gastrointestinal endoscopy showing bleeding pulsatile lesion in the first part of duodenum.

DISCUSSION

GDA aneurysms account for 1–4% of all VAAs.[2,3] GDA aneurysms are usually pseudoaneurysms secondary to chronic pancreatitis, vasculitis, connective tissue disorders, or iatrogenic vascular interventions.[1] Only risk factor in index case was sepsis, which might predispose to mycotic aneurysms. Of all VAA, incidence of aneurysmal rupture is documented up to 76% in pseudoaneurysms and 3% in true aneurysm.[2] Index child developed massive life-threatening UGI bleeding due to rupture of GDA aneurysm.

Habib et al[1] reviewed 74 cases of GDA aneurysms in adults (1956–2011), and common presentations noted were rupture (malena or hematemesis) (52%), abdominal pain (46%), compressive symptoms (vomiting and abdominal mass), or asymptomatic (7.5%). Janik et al[4] reported a 1-year-old child with sepsis and UGI bleeding secondary to GDA aneurysm who underwent successful embolization using silastic balloon. Zarin et al[5] reported an 18-year-old child with GDA aneurysm post Roux-en-Y cystojejunostomy for traumatic pancreatic pseudocyst successfully treated with endovascular embolization using large-sized polyvinyl alcohol particles.

Sensitivity of DSA is 100% in diagnosing VAA.[1] Therapeutic interventions include endovascular procedures like coil embolization, glue embolization using cyanoacrylate, stent-graft, or surgical. Endovascular procedures have lesser risk of complications, lower length of hospital stay, and cost compared to surgical interventions. Failure rate of endovascular procedures is less than 10%.[6] Index patient underwent surgery as endovascular procedures failed to control UGI bleeding.

Mortality ranges from 25% to 70% in ruptured VAA.[7] Massive transfusion along with definitive management of aneurysm helped our patient to survive the catastrophic UGI bleed. Patient required 190 mL/kg of PRBC, 110 mL/kg fresh frozen plasma (FFP), and 110 mL/kg of platelet concentrates (PCs). Balanced transfusion strategy targeting transfusion of 1:1:1 ratio of PRBC, FFP, PC along with coagulation parameters monitoring and limiting crystalloid infusions have been shown to improve mortality in adults with hemorrhagic shock.[8]

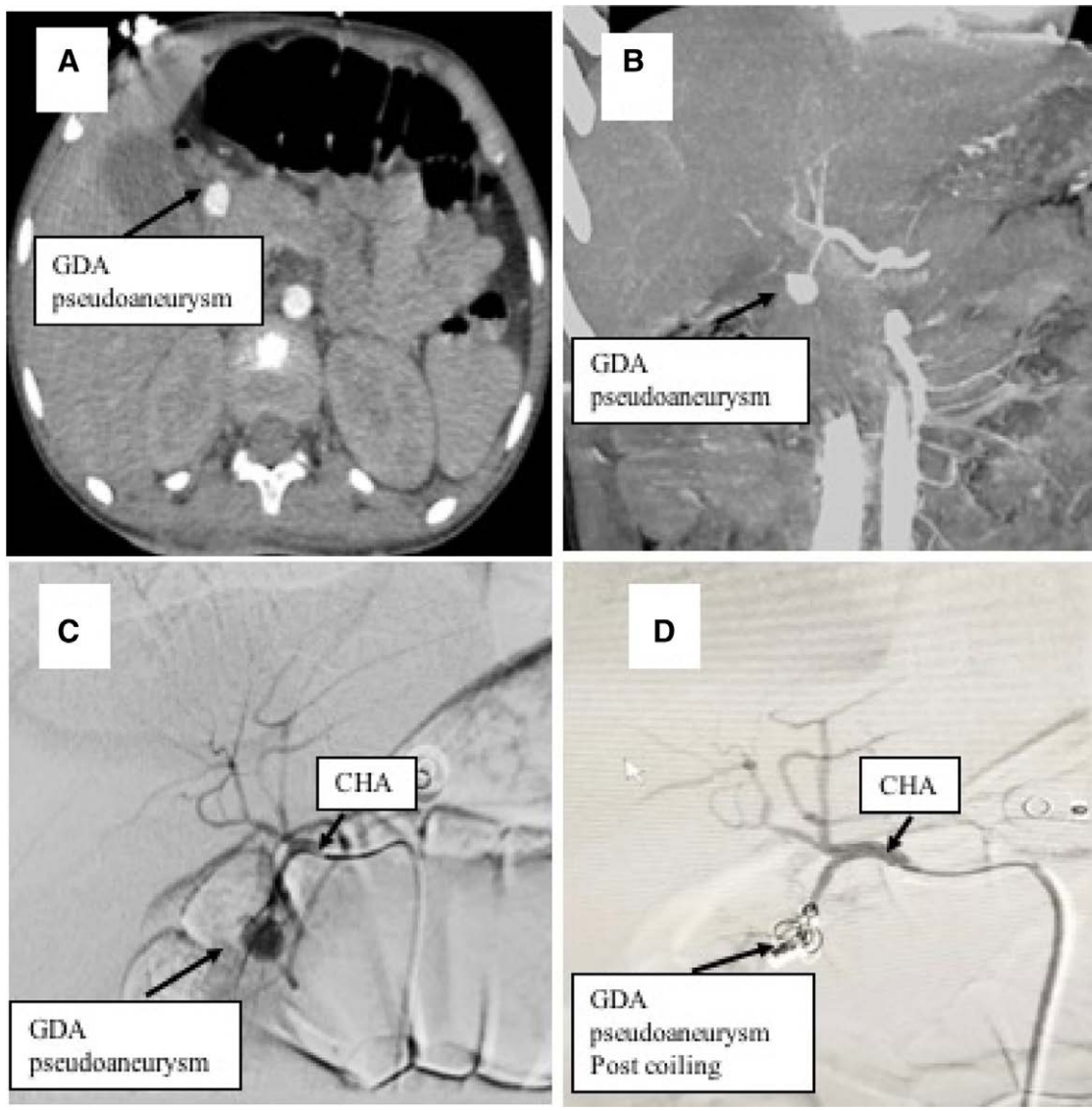

FIGURE 3. Maximal intensity projection CT angiogram showing pseudoaneurysm of gastroduodenal artery in axial (A) and coronal (B) sections. DSA of common hepatic artery showing GDA pseudoaneurysm (C). Post coil embolization DSA of GDA showing coil mass in situ with complete obliteration of pseudoaneurysm (D). GDA, gastroduodenal artery; CHA, common hepatic artery; CTA, CT angiogram; DSA, Digital subtraction angiogram.

Our index child had a life-threatening UGI bleed secondary to GDA aneurysm (possibly secondary to severe sepsis) rupture requiring massive transfusion, high inotropic support, ventilation, endovascular procedures, and laparotomy.

CONCLUSION

GDA aneurysms may result in life-threatening UGI bleeding in children. High index of suspicion of VAA, aggressive initial stabilization, prompt diagnosis by DSA, massive blood transfusion, and early definitive treatment like endovascular procedures and surgery ensues good outcome.

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