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ACUTE UPPER GASTROINTESTINAL HAEMORRHAGE CAUSED BY NON-CIRRHOTIC GASTRIC VARICES BLEEDING IN A PATIENT WITH ANTIPHOSPHOLIPID SYNDROME : A CASE REPORT

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ABSTRACT

Background: Gastric varices are intricate vascular connections linking the portosplenic venous circulation with systemic abdominal and thoracic veins. Gastric varices tend to bleed more massively and are more difficult to control endoscopically than oesophageal varices. They have a higher rebleeding and mortality risk. The management is also more complex, often requiring combined approaches such as endoscopic therapy, radiologic interventions, vasoactive agents, and sometimes shunt procedures. The hemodynamic instability in acute bleeding, coagulopathy, and portal hypertension could also complicate resuscitation and therapeutic options. In this report we describe a case of a woman, previously diagnosed with Antiphospholipid Syndrome (APS), presented with acute upper gastrointestinal haemorrhage caused by non-cirrhotic gastric varices bleeding. **Case presentation:** a 37-year-old female patient was admitted to the Emergency Department with the primary complaint of hematemesis. She has a history of Antiphospholipid Syndrome 3 years prior and was given anticoagulant therapy but she didn't continue the therapy. The contrast CT of the abdomen showed that there was a thrombus within the main portal vein. She was given packed red cells transfusion and later underwent esophagogastroduodenoscopy (EGD). The EGD result showed that she had oesophagus varices grade 1 and gastric varices GOV 2. The gastric varices GOV 2 was treated with cyanoacrylate glue injection to stop the bleeding. After she was stabilized, she was given warfarin to continue the anticoagulant therapy. **Conclusion:** Antiphospholipid syndrome can cause a hypercoagulable state and portal vein thrombosis which later present as gastric varices bleeding and needs a careful treatment approach

INTRODUCTION

Acute upper gastrointestinal haemorrhage is defined as haemorrhage that originates at the Treitz ligament and involves the oesophagus, stomach, or duodenum. The overall mortality rate associated with gastrointestinal bleeding in hospitalized patients is 14%, with a rate of 11% observed in emergency admissions and 3% among in-patients[(1)]. A thorough population-based audit conducted in Western countries has established that the incidence of acute upper gastrointestinal bleeding is 103 cases per 100,000 individuals annually. Only 4% of these cases were caused by variceal haemorrhage[(2)]. Bleeding from gastric varices tends to be more severe, with a 3-year incidence ranging from 16% to 45%, and is linked to increased mortality. Comparable rates of bleeding and mortality have been observed in patients with non-cirrhotic portal hypertension and those with cirrhosis.[(3)].

Antiphospholipid syndrome (APS) is a systemic autoimmune disorder marked by the presence of arterial, venous, or microvascular thrombosis,

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pregnancy complications, or non-thrombotic manifestations in individuals with persistent antiphospholipid antibodies (aPL). Portal vein thrombosis (PVT) refers to the formation of a thrombus in the portal vein, superior mesenteric vein, splenic vein, or inferior mesenteric vein. APS has been identified as one of the acquired prothrombotic conditions related to PVT[4]. Hirohata et al. described a case of PVT in an APS patient after ruling out more prevalent causes of PVT, implying that antiphospholipid antibodies could be the culprit in cryptogenic PVT[5]. We present a case involving acute gastrointestinal bleeding in a patient suffering from APS-related portal vein thrombosis, emphasizing the diagnostic difficulties and management considerations in the case of venous thrombosis at unusual sites.

Case Reports

A 37-year-old woman arrived at the Emergency Room, reporting episodes of vomiting bright red blood along with the presence of black stools. The patient had a history of Antiphospholipid Syndrome, diagnosed three years prior, with a mild positive Lupus Anticoagulant (LA) profile. She was checked for ANA test with the result of 9.3 unit and anti ds DNA level was 21.5 IU/mL. She had four children and had never had a miscarriage. She was receiving anticoagulant therapy at the time of her diagnosis three years ago, but stopped taking her anticoagulant and doing her check-ups. This is her first episode of gastrointestinal bleeding since being diagnosed with antiphospholipid syndrome three years ago.

Upon arrival at the emergency room, a physical examination revealed a pale appearance. Laboratory results showed severe anaemia and mild thrombocytopenia as follow.

Table 1. Complete blood count

Parameters	Results	Normal value	Denomination
Hemoglobin	6.5	11.7-15.5	g/dL
Hematocrit	22.2	32-62	%
Erythrocyte	2.65	4.4-5.9	10^6/uL
MCH	24.5	27-32	pg
MCV	83.8	76-96	fL
MCHC	29.3	29-36	g/dL
Leukocyte	5.1	3.6-11	10^3u/L
Thrombocyte	106	150-400	10^3u/L
RDW	17.6	11.6-14.8	%

The viral marker HBsAg was nonreactive and anti-HCV was nonreactive. Liver stiffness from the transient elastography examination showed compliance with the F0-F1 metavir scale. The quantitative D-dimer was elevated by 1,640 µg/L. The contrast CT examination of the abdomen revealed a thrombus in the main portal vein.

Table 2. Immunology parameters result

Parameters	Results	Normal value	Denomination
Lupus anticoagulant (LA) 1	45.8	31.0 – 44.0	seconds
Lupus anticoagulant (LA) 1 control	37.6		
Lupus anticoagulant (LA) 2	35.8	30.0 – 38.0	seconds
Lupus anticoagulant (LA) 2 control	32.9		
LA 1 : LA 2 ratio	1.28	< 1.2 negative 1.2 – 1.5 positive mild 1.5 – 2.0 positive moderate >2.0 positive severe	
ANA	9.3	Negative <20 Equivocal 20-60 Positive >60	unit
Anti ds DNA	21.5	Negative 0-200 Equivocal 201-300 Positive >300	IU/mL

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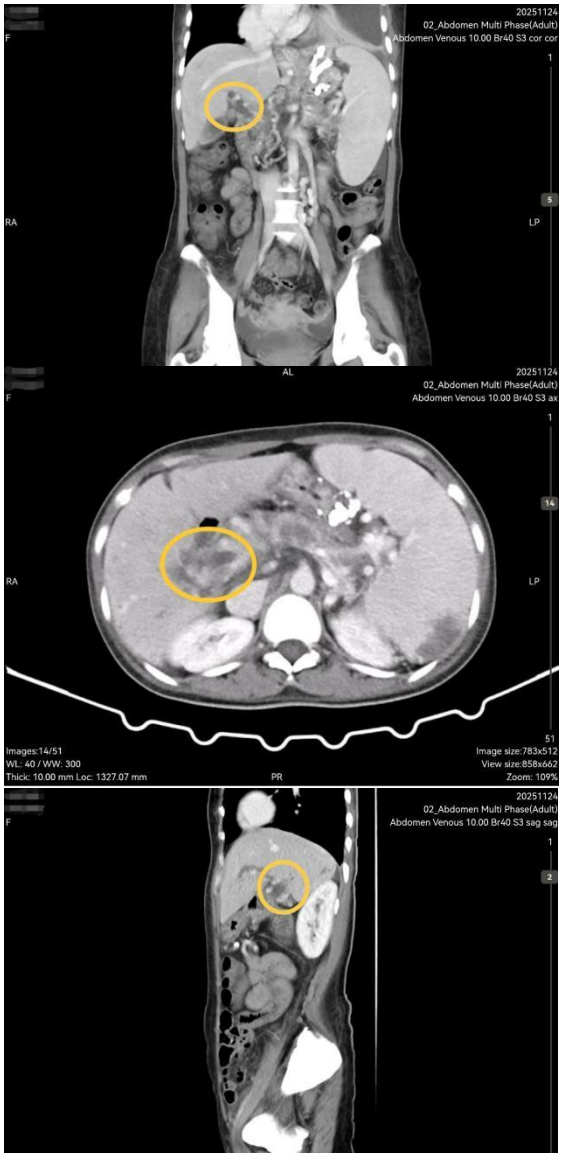

Figure 1. Contrast CT of the abdomen showed thrombus within the main portal vein

The patient received treatment with packed red blood cell transfusions and octreotide as a vasoactive agent. After the patient was stabilized, an endoscopy was performed, revealing grade II oesophageal varices and bleeding from gastric varices GOV 2 with signs of portal hypertensive gastropathy (PHG). Cyanoacrylate glue was injected to treat the gastric variceal bleeding.

After the bleeding stopped, the patient was scheduled to begin anticoagulant therapy with the vitamin K antagonist (VKA), warfarin. Her baseline INR was checked, and her result was 1.07. Her baseline prothrombin time (PT) was 12.2 seconds with a control of 10.3 seconds, and her activated partial thromboplastin time (aPTT) was 28.8 seconds with a control of 23.0 seconds. VKA treatment was initiated, and the patient was monitored for recurrent bleeding episodes. The patient was also monitored for the routine haematology test after the endoscopy procedure and the result was Hb 9.3 g/dL, leukocytes 5,600/µL and platelets 122,000/ µL.

DISCUSSION

Portal Vein Thrombosis (PVT) frequently represents the initial thrombotic manifestation in Antiphospholipid Syndrome (APS), characterized by its gradual onset and atypical symptoms, which may result in misdiagnosis. It is imperative for clinicians to exercise heightened vigilance in patients presenting with both APS and PVT. The presence of double antiphospholipid antibodies (aPLs) positivity, thrombocytopenia, and inflammation may be associated with PVT in individuals with APS. Prompt diagnosis and the administration of anticoagulant therapy can facilitate thrombus re-canalization, significantly enhancing prognosis and reducing mortality rates.[4]. In this case, the patient was previously diagnosed with APS and was treated with anticoagulant therapy but lost to follow-up. She came to the hospital with gastrointestinal bleeding and from the results of the endoscopic examination, bleeding was found from the gastric varices GOV 2. This presentation highlights the potentially serious outcomes of insufficient follow-up in APS, where PVT that is not treated or inadequately managed can quietly progress into portal hypertension, leading to devastating gastrointestinal bleeding. This underscores the importance of careful long-term care, consistent use of anticoagulation, and timely assessment of new clinical symptoms to reduce preventable health issues.

Non-cirrhotic portal hypertension (NCPH) refers to high portal pressure caused by illnesses other than cirrhosis. NCPH is a rare illness with significant morbidity and fatality rates. NCPH is difficult to diagnose due to the many causes of portal hypertension. The clinical presentation of NCPH is the presence of portal hypertension and accompanying consequences, but no indication of

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liver impairment. Splenomegaly (left upper quadrant mass) occurs in 26-97% of patients with portosinusoidal vascular disease (PSVD), with an average size of 11 cm (moderate to large). The liver is usually normal or slightly enlarged or smaller[6]. The liver size in this patient was within normal limits, according to the contrast CT of the abdomen, but splenomegaly was discovered to be 16.3 cm. Cirrhosis-related peripheral stigmata are missing. Jaundice and hepatic encephalopathy are uncommon in PSVD, but they might develop following bleeding or shunt operation. Approximately 27-87% of NCPH had hematologic abnormalities related to hypersplenism, with anaemia being the most frequent, followed by thrombocytopenia and leukopenia[6]. Upon arrival, the patient was discovered to have anaemia with Hb 6.5 g/dL, primarily due to bleeding from the stomach varices. The patient also showed mild thrombocytopenia (106,000/µL), but leucocyte count was still within normal limits. Variceal bleeding, ascites, and oedema have been recorded in 32-84%, 10-34%, and 4-18% cases, respectively[6]. This patient had no oedema or ascites, according to her physical examination. An endoscopy indicated grade II oesophageal varices and bleeding from gastric varices GOV 2. Overall, the combination of preserved hepatic architecture, significant splenomegaly, hypersplenism-related cytopenias, and clinically significant varices is characteristic of NCPH. This case underscores how PSVD can present with severe portal hypertensive complications even when liver function appears relatively normal, emphasizing the importance of thoroughly evaluating non-cirrhotic causes in similar cases.

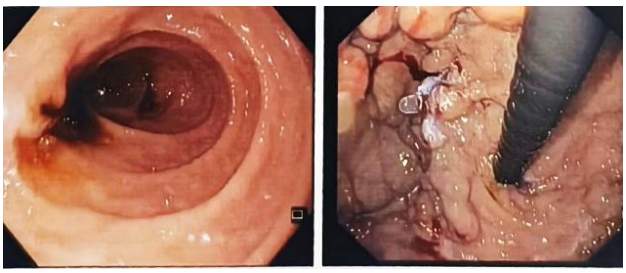

Figure 2. Gastric Varices Bleeding

The primary approach to manage NCPH involves addressing related conditions and reducing elevated portal pressure. Variceal bleeding is among the most frequent complications associated with NCPH. The cornerstone of NCPH treatment includes managing acute variceal bleeding and implementing both primary and secondary prophylaxis. According to Baveno VII, the management should mirror that of portal hypertension in patients with cirrhosis[7]. Initial treatment for acute variceal bleeding in NCPH involves stabilizing hemodynamic, securing the airway, and the administration of vasoactive medications, including terlipressin, somatostatin, or octreotide. These treatments are followed by endoscopic interventions. For acute oesophageal variceal bleeding, endoscopic variceal ligation (EVL) or endoscopic sclerotherapy (EST) should be conducted every three weeks to obliterate the varices[8]. Octreotide was administered to the patient to help lower portal venous pressure. Furthermore, the patient underwent a transfusion of packed red blood cells to stabilize hemoglobin levels. During the endoscopic procedure, cyanoacrylate glue was injected to treat the bleeding from gastric varices. Taken together, the hemodynamic stabilization, vasoactive infusion, transfusion support, and endoscopic cyanoacrylate injection provided for this patient reflect the comprehensive strategy recommended for NCPH-related variceal hemorrhage. This reinforces the need for prompt multidisciplinary management to minimize early mortality and prevent rebleeding.

The prognosis of portal vein thrombosis (PVT) may differ among patients and is contingent upon prompt anticoagulation and the sustained recanalization of the portal and mesenteric veins. Current guidelines suggest that patients with antiphospholipid syndrome (APS) experiencing venous thromboembolism should be managed with standard-intensity oral anticoagulation, targeting an international normalized ratio (INR) of 2.0 to 3.0.[9]. After the patient was free from the bleeding episodes, anticoagulant treatment was given using warfarin. The baseline INR was 1.07 and patient was discharged and monitored for her INR until it reaches the target INR of 2.0 – 3.0. The prompt reinitiation of anticoagulation therapy, accompanied by meticulous monitoring of the International Normalized Ratio

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(INR), is essential for enhancing prognosis and preventing subsequent thrombotic events in cases of antiphospholipid syndrome (APS)-associated portal vein thrombosis (PVT).

CONCLUSION

Antiphospholipid syndrome can lead to a hypercoagulable condition and portal vein thrombosis, which subsequently manifests as bleeding from gastric varices. This situation requires a meticulous treatment strategy to manage the bleeding while also administering anticoagulants to facilitate recanalization and reduce portal vein pressure.

ETHICAL APPROVAL

There wasn't any ethical issue in this manuscript. Written informed consent was obtained from the patient for the publication of the case details. All identifying information has been excluded to maintain confidentiality.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest associated with this publication.

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