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METABOLIC SYNDROME AND INHERITED THROMBOPHILIA: ARTERIAL THROMBOSIS MASQUERADING AS ACUTE PANCREATITIS

Gastroenterology

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ABSTRACT

This case report presents a 38-year-old woman with metabolic syndrome (poorly controlled type 2 diabetes, HbA1c 13.2%; hypertension) and previously undiagnosed thrombophilia who developed extensive arterial thrombosis mimicking acute pancreatitis. The patient presented with a two-month history of intermittent epigastric pain that acutely worsened, accompanied by severe metabolic acidosis (bicarbonate 10.3 mmol/L), marked leukocytosis (35.8 × 10^3/μL), and moderately elevated pancreatic enzymes (amylase 223 U/L, lipase 324 U/L). Coagulation studies revealed profoundly elevated D-dimer (5,200 ng/mL) and fibrinogen (680 mg/dL), with identified deficiencies in protein C (35%), protein S (42%), and antithrombin III (55%). Contrast-enhanced computed tomography (CT) demonstrated thrombotic occlusion of the suprarenal aorta and celiac axis without pancreatic necrosis. Management included therapeutic anticoagulation (lowmolecular-weight heparin transitioned to warfarin) and intensive metabolic control. This case highlights three key clinical insights: (1) the diagnostic challenge of differentiating vascular thrombosis from pancreatitis in metabolic syndrome patients, (2) the catastrophic thrombotic potential of combined metabolic and hematologic disorders, and (3) the necessity of multidisciplinary management addressing both acute thrombosis and underlying predispositions. The case underscores the importance of comprehensive thrombophilia evaluation in patients with unexplained arterial thrombosis and metabolic risk factors.

KEYWORDS

INTRODUCTION

Acute pancreatitis represents a leading gastrointestinal emergency, with contemporary epidemiological studies reporting an incidence of 13-45 cases per 100,000 population in industrialized nations [1]. Although gallstone disease (40-70%) and alcohol abuse (25-35%) remain the predominant etiologies, metabolic syndrome - particularly hypertriglyceridemia-induced pancreatitis - has emerged as an increasingly recognized third major cause, accounting for 2-10% of cases in recent series [2,3]. The condition carries significant morbidity, with approximately one-quarter of patients developing vascular complications, most commonly splanchnic vein thrombosis (11-23% incidence) [4]. In contrast, arterial thrombosis represents an exceptionally rare complication (<1% of cases), typically occurring in patients with underlying hypercoagulable states [5]. This case report presents the diagnostic and therapeutic challenges encountered in a 38-year-old woman with metabolic syndrome and previously undiagnosed thrombophilia who developed extensive arterial thrombosis mimicking acute pancreatitis. The clinical presentation featured several unusual elements: (a) the concurrence of complete celiac axis occlusion with suprarenal aortic thrombosis, (b) the absence of pancreatic necrosis despite marked enzyme elevation, and (c) the identification of combined protein C, protein S, and antithrombin III deficiencies. These findings provide important insights into the complex interplay between metabolic derangements and inherited thrombophilias in precipitating catastrophic vascular events.

Case Presentation

A 38-year-old woman with poorly controlled type 2 diabetes (HbA1c 13.2%) and hypertension presented with a two-month history of intermittent epigastric pain that acutely worsened over six days. Her presentation with metabolic acidosis (bicarbonate 10.3 mmol/L, lactate 15.3 mmol/L) and markedly elevated inflammatory markers (WBC 35.8 × 10^3/μL) represents a severe manifestation of metabolic syndrome complications [6]. The finding of 4+ urinary ketones alongside elevated pancreatic enzymes (amylase 223 U/L, lipase 324 U/L) initially suggested pancreatitis, though such biochemical changes can also occur in vascular thrombosis [7]. Critical laboratory results included profoundly elevated D-dimer (5,200 ng/mL) and fibrinogen (680 mg/dL), prompting thrombophilia testing that revealed protein C (35%), protein S (42%), and antithrombin III (55%) deficiencies [Table 1]. These coagulation abnormalities represent known risk factors for extensive thrombosis [8]. Contrast-enhanced CT demonstrated extensive arterial thrombosis including suprarenal aortic partial occlusion and complete celiac axis thrombosis [Figure 1,2], findings consistent with severe thrombotic events in hypercoagulable states [5]. The absence of pancreatic necrosis on expert radiology review helped differentiate this from true pancreatitis.

Management followed current guidelines for major arterial thrombosis, beginning with therapeutic low molecular-weight heparin and transitioning to warfarin [9]. The addition of statins for hypertriglyceridemia (176 mg/dL) and intensive glycemic control addressed her metabolic syndrome components [10]. This comprehensive approach led to clinical improvement, with discharge after 14 days on lifelong anticoagulation - a crucial consideration given her thrombophilia [11].

DISCUSSION

This complex case demonstrates the critical interplay between metabolic syndrome and inherited thrombophilia in precipitating catastrophic arterial thrombosis. The patient's presentation with abdominal pain and moderately elevated pancreatic enzymes initially suggested acute pancreatitis, a common diagnostic consideration in patients with uncontrolled diabetes. However, several atypical features prompted further investigation, including the prolonged symptom duration, absence of pancreatic necrosis on advanced imaging, and markedly abnormal coagulation parameters. These findings underscore the diagnostic challenges in differentiating pancreatic inflammation from vascular thrombosis in high-risk patients, particularly when multiple thrombotic risk factors coexist [5]. The pathophysiological mechanisms underlying this patient's extensive arterial thrombosis involve a perfect storm of metabolic and hematologic abnormalities. Severe hyperglycemia (HbA1c 13.2%) likely induced endothelial dysfunction through multiple pathways, including increased oxidative stress and chronic inflammation. This endothelial injury created a prothrombotic milieu that synergized with the patient's profound anticoagulant protein deficiencies (protein C 35%, protein S 42%, antithrombin III 55%). The resulting hypercoagulable state explains the development of extensive arterial thrombosis affecting both the suprarenal aorta and celiac axis - a pattern rarely seen in typical pancreatitis-associated vascular complications [12]. The management of this case required careful consideration of both acute and long-term therapeutic strategies. Immediate anticoagulation with low-molecular-weight heparin followed by warfarin was essential to halt thrombus progression and prevent potentially fatal complications like mesenteric ischemia.

The transition to warfarin rather than direct oral anticoagulants was particularly appropriate given the patient's antithrombin III deficiency, which may reduce the effectiveness of factor Xa inhibitors [8]. Concurrent optimization of metabolic parameters through intensive insulin therapy and statin administration addressed underlying contributors to endothelial dysfunction, highlighting the importance of comprehensive risk factor modification in such complex cases [10].

This case provides several crucial insights for clinical practice. First, it emphasizes the need for thorough thrombophilia evaluation in patients with unexplained arterial thrombosis, particularly those with additional risk factors like metabolic syndrome. Second, it demonstrates that pancreatic enzyme elevation alone should not preclude consideration of alternative diagnoses when clinical or radiographic features are atypical. Finally, it illustrates the critical role of multidisciplinary collaboration in managing complex thrombotic disorders, where hematologic, metabolic, and vascular considerations must be carefully balanced to optimize outcomes.

CONCLUSIONS

This case highlights how metabolic syndrome and inherited thrombophilia can combine to cause life threatening arterial thrombosis that mimics acute pancreatitis. While the patient initially presented with classic pancreatitis symptoms - abdominal pain and elevated pancreatic enzymes - the absence of pancreatic necrosis on imaging and discovery of extensive arterial thrombosis revealed a more complex pathophysiology. This underscores the diagnostic challenge of distinguishing true pancreatitis from vascular events in high-risk patients, particularly when laboratory findings overlap. The case reinforces that optimal management requires both addressing the immediate thrombotic emergency and controlling underlying metabolic dysfunction. The extensive celiac axis and aortic thrombosis demonstrates the catastrophic potential when prothrombotic conditions coexist with metabolic syndrome. Multidisciplinary care proved essential, combining anticoagulation with glycemic and lipid control. For such high-risk patients, lifelong anticoagulation may be necessary given their persistent thrombotic potential, highlighting the need for comprehensive evaluation and personalized long-term management strategies when vascular complications masquerade as pancreatic inflammation.

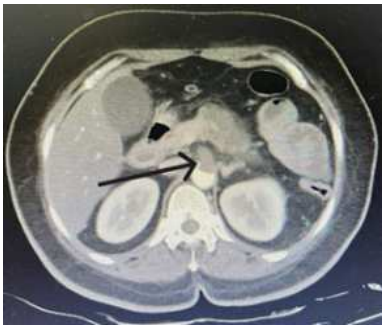

Figure 1 - Contrast-enhanced CT Reveals Complete Thrombotic Occlusion Of The Celiac Artery (Arrow) With Absent Contrast Flow.

Complete celiac artery thrombosis (arrow) on axial CT, explaining the patient's abdominal pain and subsequent splenic infarction in this case of combined metabolic syndrome and thrombophilia.

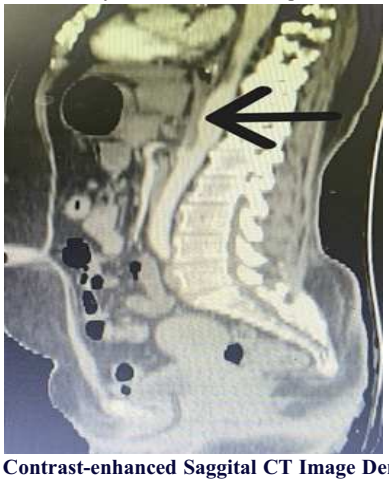

Figure -2 - Contrast-enhanced Saggital CT Image Demonstrates Partial Thrombotic Occlusion Of The Suprarenal Abdominal Aorta (White Arrow), With Visible Intraluminal Filling Defect.

A partially occlusive thrombus in the suprarenal aorta, seen as an intraluminal filling defect.

Table -1 Blood Parameters

Arterial Blood Gas (ABG)			
Parameter	Value	Normal Range	Interpretation
pH	7.31	7.35-7.45	low (acidosis)
Bicarbonate (HCO_3^-)	10.3 mmol/L	22-28 mmol/L	Metabolic acidosis
Serum Lactate	15.3 mmol/L	0.5-2.2 mmol/L	Severe lactic acidosis
Complete Blood Count (CBC)			
Hemoglobin (Hb)	10.9 g/dL	12-16 g/dL	Mild anemia
Total Leukocyte Count (TLC)	35.8 × 10^3/µL	4-11 × 10^3/µL	Marked leukocytosis
Neutrophils (%)	88%	40-75%	Neutrophilia
Platlets (PLT)	526	150-450	Thrombocytosis
Liver Function Tests (LFT)			
Total Bilirubin	0.78 mg/dL	0.2-1.2 mg/dL	Normal
AST (SGOT)	75 U/L	10-40 U/L	Mildly elevated
ALT (SGPT)	93 U/L	7-56 U/L	Mildly elevated
Alkaline Phosphatase (ALP)	143 U/L	44-147 U/L	Normal
Total Protein	5.7 g/dL	6.0-8.3 g/dL	Mildly low
Renal Function Tests (RFT)			
Urea	35 mg/dL	7-20 mg/dL	Elevated (prerenal)
Creatinine	0.86 mg/dL	0.5-1.2 mg/dL	Normal
Electrolytes			
Sodium (Na^+)	142 mmol/L	135-145 mmol/L	Normal
Potassium (K^+)	3.28 mmol/L	3.5-5.1 mmol/L	Mild hypokalemia
Chloride (Cl^-)	100 mmol/L	98-107 mmol/L	Normal
Pancreatic Enzymes			
Amylase	223 U/L	30-110 U/L	Elevated (pancreatitis)
Lipase	324 U/L	7-60 U/L	Elevated (pancreatitis)
Coagulation Profile			
INR	1.10	0.8-1.2	Normal
Protein C	Deficient	70-140%	Thrombophilia
Protein S	Deficient	60-130%	Thrombophilia
Antithrombin III	Deficient	80-120%	Thrombophilia
D-Dimer	3116	<200 ng/ml	Elevated
Fibrinogen	630	<400 mg/dl	Elevated
Additional Workup			
HbA1c	13.2%	<5.7%	Poorly controlled DM
PTH	36.3 pg/mL	15-65 pg/mL	Normal (rules out hyperparathyroidism)
Calcium	8.0 mg/dL	8.5-10.5 mg/dL	Mild hypocalcemia
Triglycerides	176 mg/dL	<150 mg/dL	Mild hypertriglyceridemia
Cholesterol	152	<200 mg/dl	Normal
LDL	108	<100 mg/dl	Elevated
HDL	48	>60 mg/dl	low

Table -1- Laboratory findings demonstrating metabolic acidosis, marked leukocytosis, pancreatic enzyme elevation, thrombophilia profile, and metabolic derangements in a patient with arterial thrombosis mimicking acute pancreatitis. Key abnormalities include severe lactic acidosis (15.3 mmol/L), protein C/S/antithrombin III deficiencies, and poorly controlled diabetes (HbA1c 13.2%).

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