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Original Research Article

From A Missing Pulse to Malignant Diagnosis: A Thrombotic Masquerade Revealing Gastric Adenocarcinoma

Soumik Dey[1], Madhumita P. Das[2], Tarliboyina Rama Krishna[3], Bhupali Talukdar[3], Rajib Kumar Roy[3]

[1]Post Graduate Trainee, Department of General Medicine, Gauhati Medical College & Hospital, Guwahati, India

[2]Professor & Head of the Department, Department of General Medicine, Gauhati Medical College & Hospital, Guwahati, India

[3]Post Graduate Trainee, Department of General Medicine, Gauhati Medical College & Hospital, Guwahati, India

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Corresponding author: Dr. Soumik Dey

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Abstract

Introduction: Cancer-associated thrombosis is a well-recognized contributor to morbidity and mortality in patients with malignancies. Although venous thromboembolism is common, arterial thrombosis is rare and often overlooked, particularly when it precedes typical cancer-related symptoms. Gastric adenocarcinoma initially presenting with arterial thrombosis is exceedingly uncommon. This report describes an unusual presentation in which upper-limb arterial thrombosis served as the first clinical indication to an underlying gastric malignancy.

Case Report: A 50-year-old woman with well-controlled hypertension presented with headache and dizziness and was noted to have absent peripheral pulses in the right upper limb. She had no history of diabetes, dyslipidemia, smoking, autoimmune disease, or thrombotic events. Vascular imaging revealed thrombosis of the distal right brachial artery extending to the bifurcation, with collateral formation. Brain imaging revealed acute and chronic lacunar infarcts, suggesting recurrent silent ischemic events. However, as the conventional cardiovascular risk factors or an identifiable embolic source were not present, the etiology initially remained unclear.

Further evaluation of secondary causes of thrombosis revealed an acquired hypercoagulable state, with reduced levels of protein C, protein S, and antithrombin III, along with elevated homocysteine levels. Autoimmune and antiphospholipid antibody test results were negative. During hospitalization, the patient developed upper gastrointestinal bleeding, prompting endoscopic evaluation. Upper gastrointestinal endoscopy revealed ulceroproliferative growth involving the gastric antrum and pylorus, and histopathology confirmed poorly differentiated gastric adenocarcinoma with signet ring cell features. Computed Tomography showed diffuse gastric wall thickening with regional and para-aortic lymphadenopathy, consistent with advanced disease, without evidence of additional arterial thrombosis.

Discussion and Conclusion: This case highlights the diagnostic challenges posed by malignancy-associated arterial thrombosis, particularly when it precedes overt oncological symptoms. The combination of arterial ischemic events and acquired deficiencies in natural anticoagulants supports a paraneoplastic hypercoagulable state. Clinicians should maintain a high index of suspicion for occult malignancy in patients presenting with unexplained arterial thrombosis, as early recognition may facilitate timely diagnosis and appropriate management.

Categories: Internal Medicine, Oncology, Gastroenterology.

Keywords: Arterial Thrombosis, Cancer-Associated Thrombosis, Gastric Adenocarcinoma, Hypercoagulable State, Occult Malignancy.

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Introduction

Gastric carcinoma is a malignancy that ranks among the leading causes of cancer-related mortality worldwide [1]. It poses a significant global health burden and is often associated with various systemic complications that can negatively impact patient outcomes. The early stages of gastric adenocarcinoma are mostly asymptomatic, and clinical symptoms such as dysphagia, abdominal pain, weight loss, or gastrointestinal bleeding often develop late, signifying advanced stages of the

disease [2,3]. Thromboembolic events are among the rare yet serious complications in such patients [4]. These events can occur in various ways, such as deep vein thrombosis or pulmonary embolism, resulting in further complications during management [5,6]. Although the vast majority of these events are venous thromboembolic complications, arterial thromboembolisms can also occur. Both are predictors of poor prognosis and increased mortality [4,6]. However, arterial thrombosis as an initial presentation of gastric adenocarcinoma is very rare, mostly unheard of and poses a major diagnostic dilemma.

Cancer patients present with a hypercoagulable state mostly due to the complex interplay between a numbers of underlying biochemical mechanisms. Cancer cells can produce and release fibrinolytic and procoagulant substances as well as inflammatory cytokines. In addition, direct interactions between tumour cells and circulating blood cells, such as monocytes, platelets, and neutrophils, or with vascular endothelial cells can trigger the activation of the coagulation cascade. Nonspecific factors, including the acute-phase response, tumour necrosis, hemodynamic disturbances leading to blood flow stasis, and altered protein metabolism may also lead to thrombus formation [7].

The association between malignancy and thrombosis was first described by Armand Trousseau in 1865, highlighting that recurrent or migratory thrombosis can be a possible clue for occult visceral malignancy [8]. In solid tumours, including gastric adenocarcinoma, arterial thrombosis may also arise in the absence of significant atherosclerosis, especially in hypercoagulable states arising from acquired deficiencies of anticoagulants such as protein C and protein S, which are naturally found in the body [9].

Although several case reports have documented arterial thrombosis in cancer patients [10], there are very few documented cases in which an arterial thrombotic event preceded the diagnosis of gastric cancer itself. This case illustrates an atypical presentation, highlighting that clinicians should maintain a high index of suspicion for underlying malignancy in patients presenting with unusual arterial thrombosis, especially when there is evidence of a prothrombotic state.

Case Presentation

A 50-year-old woman, who is a known hypertensive for one year on oral antihypertensive therapy, presented with headache and dizziness and was initially evaluated at a local health care facility. She was not known to have diabetes and had no history of dyslipidemia. During the initial assessment, she was noted to have hypotension and an absent peripheral pulse in the right upper limb, while blood pressure and peripheral pulses were preserved on the contralateral side.

Vascular imaging was performed in view of the absence of pulses in the right upper limb. Computed tomography angiography (CTA) of the right upper limb demonstrated an abrupt luminal cutoff of the distal right brachial artery at the supracondylar region, extending up to its bifurcation, with the involvement of a short proximal segment of the ulnar artery. The length of the occluded segment was approximately 4.8 cm, with evidence of collateral vessel formation, consistent with arterial thrombosis. Extrinsic compressive lesions or intramuscular collections were not observed.

Neuroimaging (MRI brain) revealed an acute lacunar infarct in the left high parietal lobe, along with chronic lacunar infarcts and areas of encephalomalacia, suggestive of previous silent ischemic events. MR angiography and venography did not reveal any significant large-vessel arterial stenosis or venous sinus thrombosis.

It was at this stage that the patient was referred to Gauhati Medical College and Hospital, a tertiary care center in north-eastern India, for further evaluation and management in view of unexplained arterial thrombosis. Upon further eliciting the history, she did not have any history of diabetes, smoking, or alcohol use. The patient denied any history suggestive of atherosclerotic peripheral vascular disease, including claudication, rest pain, or limb ischemia.

There was no history of thrombotic events, including deep vein thrombosis, pulmonary embolism, or cerebrovascular accidents. The patient had no history of oral contraceptive use, hormone replacement therapy, or prolonged immobilization. There was no history of recent surgery, trauma, or indwelling vascular catheters in the affected limb. There was no history suggestive of any autoimmune disease, such as photosensitivity, oral ulcers, Raynaud's phenomenon, skin rash, digital ulcers, arthralgia, or proximal muscle weakness.

There was no history of jaw claudication, visual disturbance or scalp tenderness. There was no history of fever, night sweats, or symptoms suggestive of infective endocarditis. The patient did not complain of chest pain, palpitations, syncope, or known cardiac disease. There were no complaints of chronic anorexia or unexplained weight loss prior to presentation. The patient's family history was unremarkable.

On examination, pulse was absent in the right upper limb with hypotension, while blood pressure and pulse were normal in the contralateral limb. There was no carotid bruit, and no clinical evidence of peripheral vascular disease was noted elsewhere. Cardiovascular, respiratory and neurological clinical

examinations were within normal limits with no abnormal findings.

Based on the clinical findings and vascular imaging, a diagnosis of right brachial artery thrombosis was established. But the underlying etiology was not initially apparent. There was no prior diagnosis of malignancy. No surgical or endovascular intervention was performed.

Her electrocardiographic findings were normal. Baseline laboratory investigations showed haemoglobin 8.4 g/dL, total leukocyte count of 7.6 × 10^3/µL, platelet count of 160 × 10^3/µL, and prothrombin time of 14 seconds, with an International normalized ratio (INR) of 1.04. Renal and liver function tests were within normal limits except for hypoalbuminemia (3.1 g/dL).

Autoimmune serology showed negative antinuclear antibody (ANA) and antineutrophil cytoplasmic antibody (ANCA) results. Given the unexplained arterial thrombosis, a thrombophilia evaluation was undertaken. The antiphospholipid antibody profile was negative. Thrombophilia screening revealed reduced levels of protein C, protein S, and antithrombin III, along with elevated homocysteine levels, which is consistent with an acquired hypercoagulable state. Tumour marker evaluation revealed an elevated carcinoembryonic antigen (CEA) level of 30.4 ng/mL, with normal CA 19-9 levels. Laboratory findings, including additional haematological and coagulation parameters, such as red cell distribution width (RDW), and dilute Russell viper venom time (DRVVT), are all listed in the following table (Table 1).

Table 1: Laboratory Investigations

Test Name	Result	Unit	Reference Range
WBC	7.6	10^3/µL	4–11
RBC	3.4	10^6/µL	4.5–5.5
Haemoglobin (Hb)	8.4	g/dL	13–16
Haematocrit (HCT)	27.1	%	40–50
MCV	79.1	fL	80–96
MCH	24.5	pg	27–32
MCHC	30.9	g/dL	32–36
Platelet Count	160	10^3/µL	150–450
RDW-SD	72.5	fL	38–46
RDW-CV	25.2	%	11.6–14
Neutrophils	91.6	%	40–80
Lymphocytes	7.1	%	20–40
Monocytes	1.3	%	2–10
Eosinophils	0	%	1–6
Basophils	0	%	0–2
Prothrombin Time (PT)	14	seconds	11–13.5
INR	1.04	—	0.8–1.2
Blood Urea	35.9	mg/dL	19.3–42.8
Serum Creatinine	0.4	mg/dL	0.5–1.2
Total Bilirubin	0.5	mg/dL	0.2–1.3
Unconjugated Bilirubin	0.3	mg/dL	0–1.1
Conjugated Bilirubin	0.2	mg/dL	0–0.3
AST (SGOT)	23	U/L	15–46
ALT (SGPT)	12	U/L	4–45
Alkaline Phosphatase	65	U/L	28–126
GGT	14	U/L	12–58
Total Protein	6.6	g/dL	6.3–8.2
Albumin	3.1	g/dL	3.5–5.0
HBsAg	Negative	—	Negative
Homocysteine	14.92	µmol/L	4.44–13.56
Protein C Activity	31	%	70–130
Protein S Antigen (Free)	22	%	70–134
Antithrombin III Activity	42	%	80–120
Factor VIII Activity	42.5	%	56–191
Lupus Anticoagulant (DRVVT)	Absent	—	Absent
DRVVT Screen Ratio	1.62	Ratio	0–1.20
Beta-2 Glycoprotein IgG	<0.80	U/mL	Negative <7
Beta-2 Glycoprotein IgM	<2.40	U/mL	Negative <7
Anticardiolipin IgG	8.46	U/mL	0–48

Anticardiolipin IgM	3.84	U/mL	0–44
Anti-phospholipid IgG	2.09	AU/mL	Negative <10
Anti-phospholipid IgM	4.49	AU/mL	Negative <10
Factor V Leiden Mutation	Not detected	—	Not detected
ANA (HEp-2, IFA)	Not detected	—	Not detected
P-ANCA	Not detected	—	Not detected
C-ANCA	Not detected	—	Not detected
CEA	30.4	ng/mL	0–3
CA 19-9	1.4	U/mL	0–37

Abbreviations: WBC, white blood cell; RBC, red blood cell; Hb, haemoglobin; HCT, haematocrit; MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RDW-SD, red cell distribution width–standard deviation; RDW-CV, red cell distribution width–coefficient of variation; PT, prothrombin time; INR, international normalized ratio; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; HBsAg, hepatitis B surface antigen; DRVVT, dilute Russell viper venom time; APLA, antiphospholipid antibodies; ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; P-ANCA, perinuclear antineutrophil cytoplasmic antibody; C-ANCA, cytoplasmic antineutrophil cytoplasmic antibody; CEA, carcinoembryonic antigen; CA 19-9, carbohydrate antigen 19-9.

Following confirmation of an acquired hypercoagulable state on thrombophilia screening, characterized by reduced protein C, protein S, and antithrombin III levels with elevated homocysteine, the patient was started on therapeutic low-molecular-weight heparin (LMWH) for management of the documented brachial artery thrombosis. Within 10 days of commencing anticoagulant therapy, the patient developed upper gastrointestinal bleeding manifested by hematemesis and melena. Although the etiology of the bleeding was not immediately apparent, anticoagulation was withheld, and conservative management was instituted. Upper gastrointestinal endoscopy was undertaken, which revealed an ulceroproliferative lesion involving the antrum and pylorus with mucosal edema and luminal narrowing. Biopsies were obtained. Histopathological examination revealed poorly differentiated adenocarcinoma with signet ring cell features, with signet ring cells comprising approximately 30% of the tumour, confirming the diagnosis of gastric adenocarcinoma. The findings are shown in the figures (Figure 1 shows histopathology and figure 2 shows upper gastrointestinal endoscopy findings).

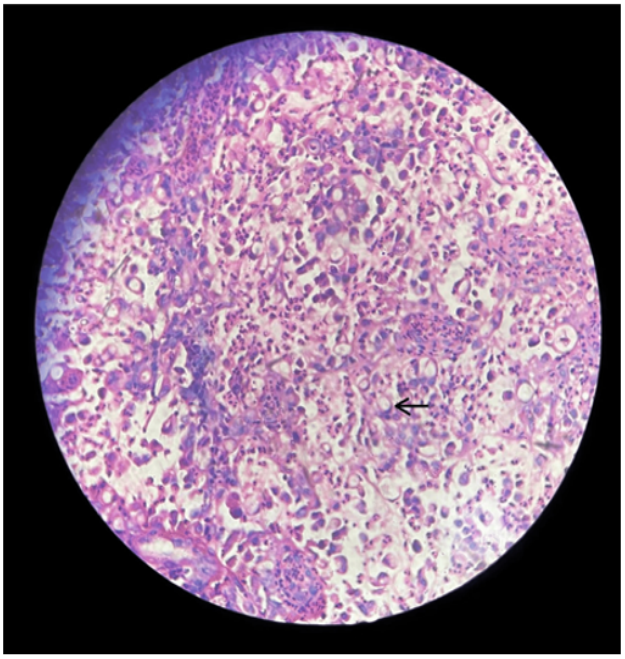

Figure 1: Poorly differentiated gastric adenocarcinoma with signet ring cells. Histopathology shows poorly differentiated adenocarcinoma composed predominantly of signet ring cells characterized by large mucin vacuoles compressing the nuclei to the periphery (arrow). Haematoxylin & Eosin stain, ×40 magnification.

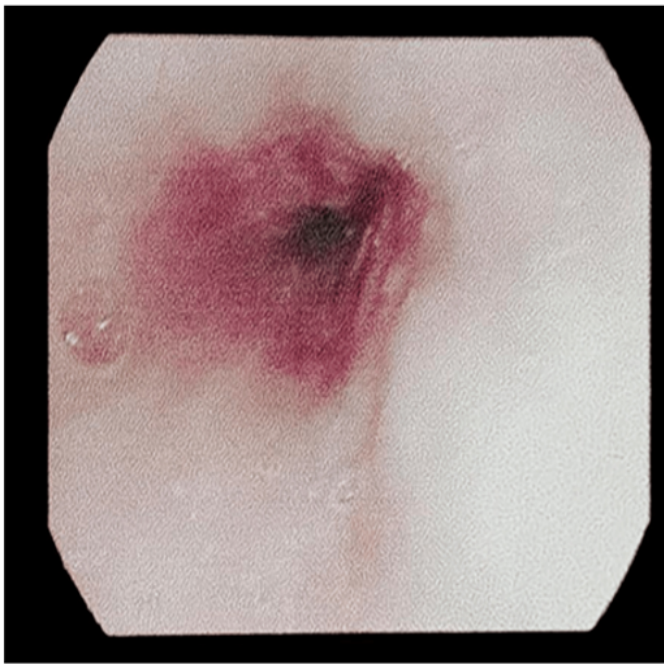

Figure 2: Upper gastrointestinal endoscopy findings showing ulceroproliferative growth in stomach. Endoscopic image showing an ulceroproliferative lesion in the gastric antrum and pylorus with mucosal irregularity, edema, and luminal narrowing, from which biopsies were obtained.

Following the endoscopic diagnosis, contrast-enhanced Computed Tomography (CT) of the chest, abdomen, and pelvis was performed. Imaging revealed diffuse long-segment transmural circumferential thickening of the body and antrum of the stomach (up to 1.7 cm thickness) with luminal narrowing and perilesional fat stranding, consistent with primary gastric malignancy. Enlarged left gastric, celiac axis, superior mesenteric artery, and para-aortic lymph nodes were noted, suggesting metastatic nodal disease. Mild ascites was present. No focal hepatic lesions were identified.

To evaluate for any additional vascular pathology, Computed Tomography (CT) Angiography of the thorax and abdomen was performed. The pulmonary arteries and thoracic aorta were normally outlined, with no evidence of pulmonary embolism, aneurysm, or dissection. The abdominal aorta, celiac axis, superior mesenteric artery, inferior mesenteric artery, and bilateral renal arteries were patent without significant luminal narrowing. Mild atherosclerotic calcification was noted in the abdominal aorta. No arterial thrombosis was identified in the thoracoabdominal vasculature. These findings collectively established the diagnosis of gastric adenocarcinoma associated with arterial thrombosis due to acquired hypercoagulable state.

Discussion

Cancer-associated thrombosis is a well-recognized clinical entity that remains a major contributor to morbidity and mortality in patients with malignancy [11]. While venous thromboembolism is the most frequent manifestation of malignancy-related hypercoagulability [6,12], arterial thrombotic events, particularly those involving the upper limb, are extremely rare and often under-recognized [13, 14].

The present case illustrates an unusual thrombotic masquerade in which upper limb arterial thrombosis preceded the diagnosis of gastric adenocarcinoma, thereby posing a significant diagnostic challenge. Moreover, the absence of diabetes, dyslipidaemia, smoking, significant atherosclerotic disease, cardioembolic sources, autoimmune vasculitis, and antiphospholipid syndrome strengthens the likelihood of a malignancy-driven hypercoagulable state [7].

Previous case reports do highlight some instances of arterial thromboembolism in malignancies in spite of its rarity. Boon and Boon reported arterial thrombosis occurring shortly after initiation of chemotherapy in a patient with metastatic gastric adenocarcinoma, highlighting the prothrombotic milieu associated with advanced malignancy and its treatment [10]. Similarly, Chien et al. described lower-limb arterial occlusion as a manifestation of Trousseau syndrome in advanced gastric cancer, while Bashir et al. reported catastrophic multi-arterial and venous thrombosis in hereditary diffuse gastric cancer [15,16]. Collectively, these reports demonstrate the diverse and infrequent arterial manifestations of gastric malignancy.

In contrast to the previously reported cases, our patient presented with brachial artery thrombosis accompanied by radiological evidence of silent cerebral ischemic events before the diagnosis of

gastric adenocarcinoma was established. The involvement of the upper limb, together with the absence of conventional risk factors, represents an unusual clinical presentation that further expands the spectrum of arterial thrombotic manifestations associated with gastric cancer.

A particularly notable aspect of this case was the coexistence of reduced protein C, protein S, and antithrombin III levels. Although inherited deficiencies of these natural anticoagulants are well-recognized causes of thrombophilia, acquired reductions have also been reported in association with malignancy and may result from increased consumption, inflammatory cytokine-mediated dysregulation, and impaired synthesis [17]. While the precise contribution of these abnormalities cannot be determined in an individual case, their presence alongside an underlying gastric adenocarcinoma suggests a possible role in the hypercoagulable state that may have contributed to the arterial thrombotic presentation.

The present case highlights the importance of maintaining a broad differential diagnosis in patients presenting with unexplained arterial thrombosis. When routine cardiovascular, autoimmune, and thrombophilia causes have been excluded, consideration of an underlying malignancy may facilitate earlier diagnosis and appropriate oncological evaluation, particularly in patients with atypical thrombotic presentations [18,19].

Conclusions

This case highlights the importance of a comprehensive diagnostic approach while evaluating atypical arterial thrombotic events. Early recognition of malignancy-associated hypercoagulability may facilitate prompt diagnosis of the underlying cancer and guide appropriate management strategies, including anticoagulation and oncological evaluations.

This report has several limitations inherent to its nature as a single-patient case study. Although the temporal association between arterial thrombosis and the subsequent diagnosis of gastric adenocarcinoma suggests a possible malignancy-associated hypercoagulable state, a definitive causal relationship cannot be established. The observed reductions in protein C, protein S, and antithrombin III were documented during an acute thrombotic event and may have been influenced by concurrent systemic inflammation, acute-phase responses, or other disease-related factors. Furthermore, mild atherosclerotic changes and elevated homocysteine levels may have contributed to the thrombotic process. An additional limitation is the lack of long-term follow-up data. Following confirmation of the diagnosis, the patient was referred to a specialized oncology center for further evaluation and management; however, subsequent information regarding treatment received, disease progression, recurrence of thrombotic events, and survival outcomes was unavailable. Consequently, the broader clinical implications of this association and its impact on patient outcomes remain uncertain. Further studies involving larger patient cohorts and long-term follow-up are needed to better characterize the relationship between occult gastric malignancy and arterial thrombotic events, as well as the underlying mechanisms contributing to this presentation.

Despite these limitations, the present case highlights an uncommon presentation of occult gastric adenocarcinoma and highlights the importance of considering an underlying malignancy in patients presenting with unexplained arterial thrombotic events after exclusion of more common etiologies.

Author Contributions: All authors contributed equally to this work.

Human Ethics Statement: This study was conducted using anonymized patient data. No identifiable patient information is included in this report.

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