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# A Unique Case of Aortic Mural Thrombus in the Non-Atherosclerotic Aorta of Patient with Multiple Hypercoagulable Factors

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**Abstract**: An aortic mural thrombus (AMT) in a non-atherosclerotic wall is a rare but important cause of systemic arterial thromboembolism. We herein report a case of AMT in the thoracic aorta where hypercoagulable factors protein S insufficiency and homocysteinemia were observed and promptly treated with anticoagulation. When we encounter patients with AMT, a precise assessment of a hypercoagulable disease is required since hypercoagulable states can influence the genesis of AMT.

**Keywords**: Protein s Homocysteinemia Mural thrombus Cfa thrombus Mca thrombus Systemic embolism Anticoagulation Thrombhopiliac Hypercoagulant.

**Introduction**: Five percent of systemic embolisms are attributed to aortic thrombosis nevertheless, aortic thrombus is uncommon and seldom identified prior to embolic consequences. Thoracic aortic thrombosis is mostly caused by a history of hematological disorders that produce a condition of hypercoagulability. Aortic pathologies, such as dissection, aneurysm, or vasculitis, are among the other possible causes. Aortic thrombosis can be treated surgically, with interventional therapy, or with medication. When treating aortic thrombosis, prolonged oral anticoagulation, surgical thrombectomy following anticoagulant medication, or both are typically advised. The most recommended course of treatment when it comes to the ascending aorta is surgical thrombus removal, notwithstanding its rarity.

Systemic arterial thromboembolism is a serious potentially fatal condition that can cause limb ischemia, myocardial ischemia, cerebral ischemia, or visceral ischemia. The majority of arterial thromboembolism cases are thought to have cardiac causes, notably sequelae to atrial fibrillation and myocardial infarction [(1).]

Nevertheless, a small number of cases often referred to as "cryptogenic embolisms" occur without such origins [2, 3]. The aorta has been identified as a possible embolic source due to recent advancements in imaging modalities, particularly transesophageal echocardiography (TEE) and contrast-enhanced aortographic studies. When the aorta is suspected as the embolic source, complex atherosclerotic lesions are usually involved [3, 4]; however, mural thrombus on a normal or minimally atherosclerotic aorta has also been regarded as an unusual but possible cause of arterial thromboembolism [2, 5]. Furthermore, some hypercoagulable disorders have frequently been detected in cases of aortic mural thrombus (AMT) [5-9]. As its clinical features are not well-known, we herein report a case of AMT in a patient with multiple hypercoagulable factors like low protein-s activity and homocysteinemia.

## **Case Report:**

A 31-year-old man who was a non-smoker, non-alcoholic with no other comorbidities and no significant past and family history presented to the emergency department due to sudden dysarthria and upper limb and lower limb paralysis on the left side and lower limb pain and swelling on the right side. He had sinus rhythm on his electrocardiogram, a normal left ventricular function, and no abnormal findings in the aortic or mitral valve on transthoracic echocardiography (TTE). contrast-enhanced computed tomography (CT) angiography neck and brain revealed an eccentric thrombus attached to the lateral wall of the posterior part of the aortic arch and thrombotic occlusion of the M1 and M2 segments of the right middle cerebral artery. Magnetic resonance imaging revealed acute cerebral infarction and contrast-enhanced computed tomography (CT) peripheral angiography revealed a thrombosed right common iliac artery 1cm proximal to the origin of the common femoral artery. We considered the lesions on the thoracic aorta, middle cerebral artery, and common femoral artery to be a thrombus and started anticoagulant therapy (ACT) with intravenous unfractionated heparin. A laboratory examination of thrombophilia before ACT demonstrated a low protein s and low protein s activity and increased serum homocysteine.

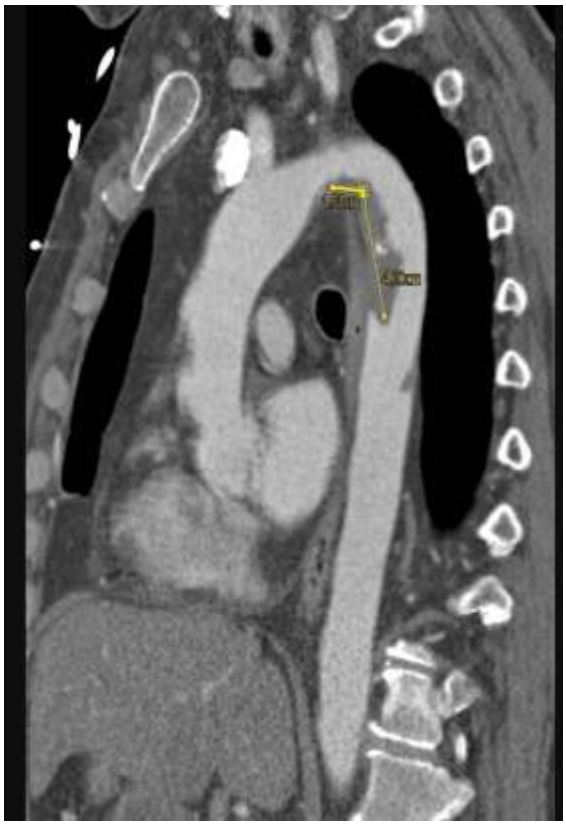

**Fig 1** Aortic arch mural thrombus

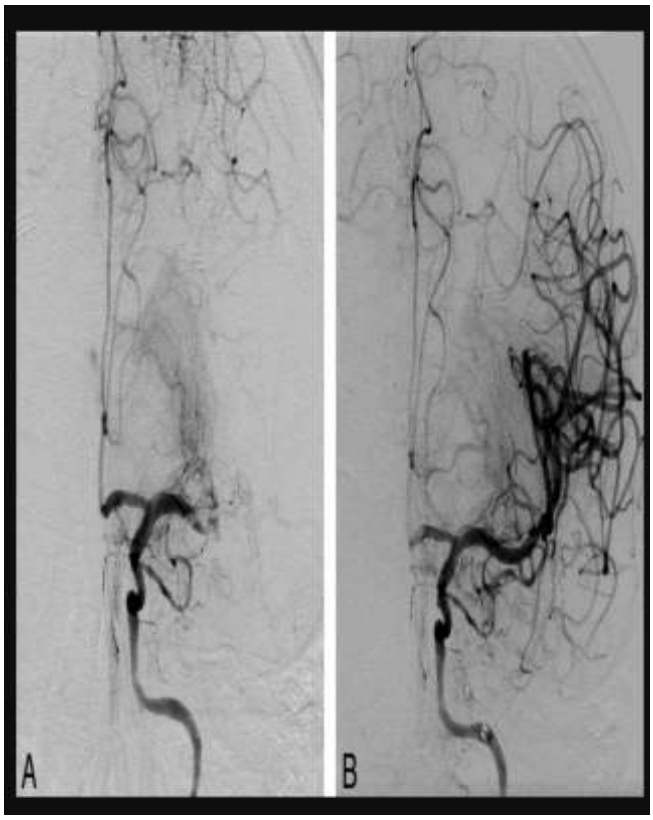


**Fig 2** Right MCA thrombus

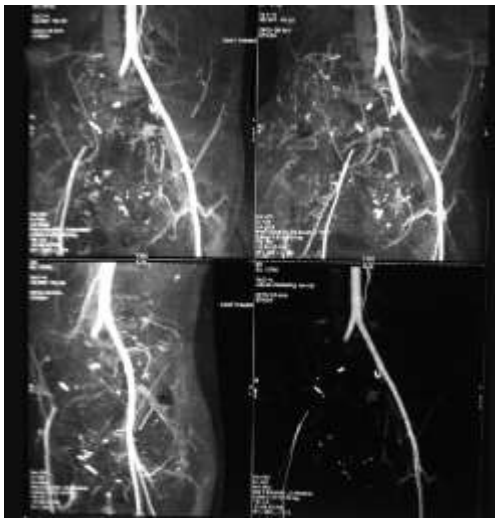

**Fig 3** Right CFA thrombus

**Laboratory Data on Coagulation-fibrinolysis System**

| Variables | Reference | Case |
|---|---|---|
| PT (%) | 70-130 | 108 |
| PT (INR) | 0.9 -1.3 | 1.1 |
| APTT (s) | 25-37 | 32 |
| Platelet (×104/mm3) | 12.0-38.0 | 21.2 |
| D-dimer (μg/mL) | 0-1.0 | 0.6 |
| Antithrombin III (mg/dL) | 23-34 | 28 |
| Antithrombin III (%) | 80-130 | 94 |
| Protein S free antigen (%) | 65-135 | **55** |
| Protein S activity (%) | 60-150 | **40** |
| Protein C antigen (%) | 62-131 | 82 |
| Protein C activity (%) | 64-135 | 101 |
| Antiphospholipid antibody (unit/mL) | ≤10.0 | 6.3 |
| Lupus anticoagulant (normalized ratio) | ≤1.3 | 0.8 |
| Serum homocysteine (μmol/l) | 5-15 | **55** |

PT: prothrombin time, INR: international normalized ratio, APTT: activated partial thromboplastin time

Two weeks after the initiation of ACT and following careful observation, contrast-enhanced CT confirmed the disappearance of the lesion in the aortic arch without any further embolic events. four months later, a follow-up imaging study showed no further embolic findings, and he claimed no further symptoms following oral ACT with warfarin and homocysteine reducing drugs were given and followed

## Discussion

Aortic plaque is an important etiology of systemic embolism from non-cardiac sources and is usually caused by thrombus or cholesterol crystals, both of which arise from severe aortic atherosclerotic plaques [3, 4] . In a non-atherosclerotic wall thrombus, the thrombophilia profile has to be investigated extensively and

multiple factors should be sought as in our case with decreased protein s levels, decreased protein s activity and increased homocysteine levels were found to be associated with the thrombus

## Conclusion

AMT is a rare but important embolic source. In cases of mural thrombosis on a non-atherosclerotic aortic wall, we should consider underlying hypercoagulable disorders and concurrent malignancy and carefully select proper treatment depending on each situation.

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