

# Hypercoagulable State with Elevated Levels of Lipoprotein(a)

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#### Abstract

It is well described that hyperlipoproteinemia(a) is a risk factor for cardiac events as well as venous thromboembolism and cerebrovascular accidents. While there are reports of such clinical presentations individually in association with elevated lipoprotein(a), but there is no reported case of diffusely hypercoagulable presentation consisting of myocardial infarction, venous thromboembolism and cerebrovascular accidents. Here, we report the first case of such constellation of findings and discuss diagnostic and therapeutic modalities.

*Keywords:* Lipoprotein(a); Hyperlipoproteinemia(a); Venous Thromboembolism; Myocardial Infarction; Cerebrovascular Accidents; Apheresis; Plasmapheresis; Cardiac Computed Tomography Angiogram

#### Case Report

#### Case

A 52-year-old man with no past medical history presented to our hospital with left sided hemiparesis and an NIH stroke scale of 19. Immediate code stroke was called and subsequent stat computed tomography (CT) arteriogram of the brain with intravenous contrast revealed an occluded proximal right middle cerebral artery (MCA) approximately 1.5 cm distal to its origin. Based on this patient's clinical history and presenting symptoms, he was given tPA. Subsequent magnetic resonance imaging (MRI) of the brain correlated with his CT findings, demonstrating moderately extensive acute right MCA infarcts as well as moderately extensive scattered chronic ischemic deep white matter disease (Figure 1A, 1B).

Although, patient denied any chest pain, an elevated troponin level of 0.48 and abnormal electrocardiogram with signs of lateral ischemia and anteroseptal infarction raised concern of significant coronary disease. Immediate cardiac catheterization was deferred in setting of recent tPA administration, and patient elected for non-invasive evaluations. An echocardiogram revealed an ejection fraction of 25% with focal wall motion abnormalities involving the inferior and lateral walls consistent with ischemic cardiomyopathy. Coronary CT angiography showed total occlusion of the left anterior descending (LAD) artery, subtotal to total occlusion of the proximal right coronary artery (RCA), as well as extensive bilateral pulmonary emboli (Figure 2A, 2B and 2C). Few days later, cardiac catheterization confirmed coronary CT angiography findings, including layered thrombus in the distal RCA (Figure 3A, 3B, 3C, and 3D).

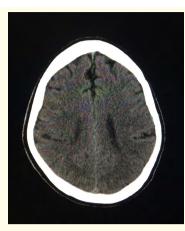
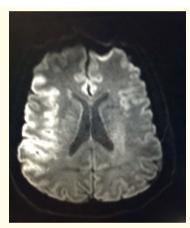


Figure 1A: Computed Tomography (CT) of head without contrast- extensive acute infarct of right middle cerebral artery territory.



*Figure 1B:* Magnetic Resonance Imaging (MRI) of brain- confirming CT finding of extensive acute infarct of right middle cerebral artery territory.



Figure 2A: Coronary CT Angiogram: showing total occlusion of LAD and flow limiting lesion in a diagonal branch.

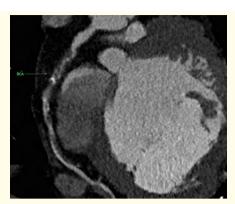


Figure 2B: Coronary CT Angiogram: subtotal to total occlusion of RCA, suspicious for layered thrombus.



Figure 2C: Coronary CT Angiogram: extensive bilateral pulmonary embolism.



Figure 3A: Coronary Angiogram: Left Lateral view-layered thrombus in RCA.



Figure 3B: Coronary Angiogram (LAO view)- flow limiting lesion in RCA.

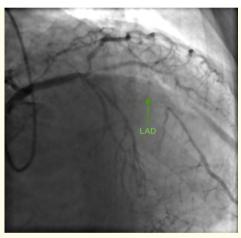


Figure 3C: Coronary Angiogram (RAO Cranial view)- flow limiting lesion in LAD.

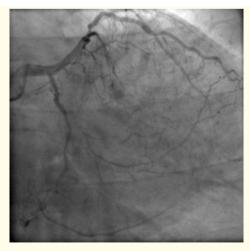


Figure 3D: Coronary Angiogram (RAO Caudal view)- flow limiting lesion in LAD.

A hypercoagulable work up was performed to explain the embolic CVA, bilateral pulmonary emboli, and RCA thrombus: He was found to have a severely elevated level of lipoprotein(a): 492 nmol/L (desirable levels < 35 nmol/L). Remaining hypercoagulable work up was unrevealing.

To prepare for coronary artery bypass graft (CABG) surgery, the patient was started on plasmapheresis, which lowered his lipoprotein(a) levels to 92 nmol/L. CABG was performed without complication. Post-operatively, the patient continued to receive plasmapheresis for one week along with warfarin and oral niacin. The patient did well and was discharged home in good condition with markedly improved left hemiparesis and only minimal residual left hand weakness one month after the initial admission.

#### Discussion

To our knowledge, this is the first case report of elevated lipoprotein(a) associated with simultaneous embolic CVA, bilateral PE and thrombotic non-ST elevation myocardial infarction. These hypercoagulable presentations are reportedly separately in relation to elevated lipoprotein, but never collectively; there are clear individual association between elevated lipoprotein with coronary disease and myocardial infarction [1-3] as well as venous thromboembolism [4,5], and embolic cerebrovascular accident [6,7]. Coronary CT angiogram is an essential non-invasive diagnostic modality, especially in cases when immediate cardiac catheterization is at high risk. For our case, its findings of obstructive coronary disease as well as bilateral pulmonary embolism were instrumental in making the diagnosis of hyperlipoproteinemia(a), which led to an appropriate treatment regimen.

Plasmapheresis was recommended for this patient prior to his scheduled CABG, due to the fact that hyperlipoproteinemia is a known risk factor for pulmonary embolism in patients undergoing CABG [8]. There are new observational data, which validates the efficacy of lipoprotein apheresis in reducing future cardiovascular events in setting of hyperlipoproteinemia [9]. However, this treatment and preventive option is highly contentious and there needs to be a further randomized study investigating not only the efficacy of lipoprotein apheresis [10], but also the cost effectiveness of this invasive option compared to alternative options.

## Conclusion

Patients with hyperlipoproteinemia are at an increased risk of thrombotic events including CVA, MI, and PE. Plasmapheresis is one of the available treatment options in setting of hyperlipoproteinemia, and should be considered.

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