

117. Co-Occurrence of Cerebral Stroke and ST Elevation Myocardial Infarction Associated With Protein S Deficiency

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Body

Background: Venous thrombosis is the usual manifestation of Protein S Deficiency. To our knowledge, there are no case reports on patients with stroke and myocardial infarction occurring simultaneously in the background of Protein S Deficiency.

Case: We report a 51-year-old female, with strong family history of cardiovascular disease and known case of Protein S deficiency since 2013 initially presented with multiple deep vein thromboses of the left arm and recurrent stroke. She was maintained on warfarin and aspirin but was noncompliant. She presented with new onset right homonymous hemianopsia with dizziness and vomiting. On cranial magnetic resonance imaging and angiography, multiple infarcts at different vascular territories were seen. On the second day of hospitalization, she complained of severe chest pain. 12-lead electrocardiogram showed ST elevation in anteroseptal leads. An emergency coronary angiogram showed a total thrombotic occlusion of the proximal left anterior descending artery. Balloon dilation confirmed a heavy thrombus burden hence intracoronary tirofiban 60mcg and 9mcg/min drip was given followed by manual aspiration thrombectomy. After which her symptom resolved, and deferral of stenting was decided. A repeat coronary angiogram 24 hours post tirofiban drip showed lesser thrombus in the proximal left anterior descending artery and mid to distal left circumflex artery but with TIMI flow II; hence drug eluting stents were deployed. She was discharged stable, improved and on aspirin, ticagrelor and apixaban.

Discussion: Protein S Deficiency is a diagnostic challenge because of the immediate need to start and maintain on anticoagulation. Genetic testing is also lacking in a low resource setting. The hypercoagulable state may lead to acute cerebral and coronary thrombosis. Smooth coronary arteries and heavy thrombus burden may be seen and can be treated with intracoronary and intravenous GP IIB/IIIA receptor antagonist followed by stenting. A prolonged triple therapy should be started and maintained indefinitely for prevention.

Table 1. Coagulation profile

<i>Test name</i>	<i>Patient's values</i>	<i>Normal values</i>
<i>Protein S</i>	19%	60-140
<i>Protein C</i>	74.4 %	70-140
<i>Homocysteine</i>	7.21	
<i>Anti thrombin</i>	94.1 %	80-120
<i>Kaolin Clotting time</i>	42.6 seconds	31-45
<i>Dilute Russell Viper Venom Time</i>	38.4 seconds	31-44
<i>Factor VIII</i>	97%	50-150
<i>Anti cardiollipin antibody IgG</i>	1.2 GPL-U/ml	
<i>Anti cardiollipin antibody IgM</i>	1.0 MPL-U/ml	
<i>aPTT</i>	31.9 vs 42.0	



