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ABSTRACT N. PO68

WHOLE BLOOD HYPERCOAGULABLE PROFILES IN A PATIENT WITH MARKEDLY ELEVATED LIPOPROTEIN(A) PLASMA LEVELS AND THROMBOTIC COMPLICATIONS: A CASE REPORT.

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Background: Lipoprotein(a) (Lp(a)) is a well-established, genetically determined risk factor for atherosclerotic cardiovascular disease (ASCVD). It is an LDL-like lipoprotein with proatherogenic, proinflammatory, and potentially prothrombotic properties, that contributes to endothelial dysfunction, plaque formation, and thrombosis. Its distinctive component, apolipoprotein(a) is structurally homologous to plasminogen, and may impair fibrinolysis and platelet reactivity. Traditional coagulation tests are unable to identify the prothrombotic phenotype in patients with markedly elevated Lp(a) plasma levels. The possible role of whole blood rotational thromboelastometry and impedance aggregometry to evaluate hemostatic system functions in these patients is unknown.

Case Report: A 64-year-old woman with arterial hypertension and type 2 diabetes complicated by chronic kidney disease since the age of 45 yrs, was referred to our department to study her extensive ASCVD. Her cardiovascular history began in February 2024 with an episode of acute pulmonary edema secondary to a non-ST elevation myocardial infarction, managed with triple coronary artery bypass grafting. Six months later, she developed acute ischemia of the left lower extremity, which required sequential thrombectomy, bypass, and angioplasty. The lipid profile (i.e., LDL-C 102 mg/dL, triglycerides 154 mg/dL, and apolipoprotein B 1.01 g/L) represented the best values achieved to date, considering her statin intolerance and the use of the maximally tolerated lipid-lowering therapy (e.g. atorvastatin 20 mg plus ezetimibe). Notably, plasma Lp(a) levels were markedly elevated (925 nmol/L, reference range <75 nmol/L). Other inherited and acquired thrombophilic conditions were excluded. Whole blood rotational thromboelastometry identified a markedly hypercoagulable profile characterized by a shortened propagation phase of the coagulation and increased clot firmness [Table 1]. Moreover, impedance aggregometry showed a resistance to the antiplatelet therapy taken by the patient (i.e., acetylsalicylic acid 100 mg PO once daily) [Table 1]. Apheresis machine cycles were scheduled but the patient refused to continue due to a vasovagal syncopal episode during the first session. PCSK9 inhibitors were initiated, and acetyl salicylic acid dosage was increased to 165 mg PO once daily. The patient died a week later from an acute myocardial infarction.

Conclusions: This case report highlights the complex interplay between Lp(a) and ASCVD. Emerging evidence suggests that markedly elevated Lp(a) levels may not only accelerate atherogenesis but may also interfere with coagulation, resulting in a prothrombotic phenotype. Whole blood thromboelastometry and impedance aggregometry may help detect hypercoagulability in these patients. Further investigations are needed to confirm our preliminary findings and to clarify their possible clinical implications.

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	Patient's values	Reference values
ROTEM		
Clotting time (CT), sec		
INTEM	193	161-204
EXTEM	65	50-80
Clot formation time (CFT), sec		
INTEM	**48**	62-130
EXTEM	**43**	46-149
Maximum Clot Firmness (MCF), mm		
INTEM	**76**	51-69
EXTEM	**76**	55-72
FIBTEM	**28**	6-21
Impedance aggregometry		
ADP-test	104	48-119
ASPI-test	51	50-112
TRAP-test	121	86-159