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Short Communication

Significance of Primary Fibrinogenolysis

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Dear editor,

Primary fibrinogenolysis is a circumstance where the fibrinogen is enzymatically decomposed due to plasmin activity. This process indicates that fibrinogen is pathologically degraded by plasmin. Primary fibrinogenolysis in its pure form is rare. However, it can occur if dynamic plasmin is excessively released intravenously once the clotting pathway is malfunctioning. Shock, trauma, surgical interventions, acute leukemia, heatstroke, and advanced liver diseases have all been linked to primary fibrinogenolysis. It can emerge in patients experiencing breast cancer, lung cancer, prostate cancer, and renal cell carcinoma. Many plasminogen activators are released into the bloodstream beyond the inhibitors' capability. They may be produced by bodily reserves (mostly endothelial cells) [1, 2]. In this correspondence, we will explore the hemostatic alterations that have been reported and whether they are relevant to primary fibrinogenolysis.

Substantial bleeding is mediated by fibrinogen depletion (split by plasmin) and the release of fibrin split products from fibrinogen [1]. Patients generally do not experience severe bleeding but are at substantial risk for hemorrhage resulting from hypofibrinogenemia. Pronounced thrombocytopenia is expected to raise suspicions of disseminated intravascular coagulation (DIC) [2].

Prothrombin time and partial thromboplastin time are almost always both prolonged. Platelet count is normal with an absence of microcirculatory thrombosis. One of the essential laboratory tools to discriminate between primary fibrinogenolysis and DIC is platelet count, which stays normal (>150 $\times$ 10^9/l) in primary fibrinogenolysis while reduced in DIC. Secondly, antithrombin concentration is low in DIC, but normal in primary fibrinogenolysis. Thirdly, euglobulin clot lysis time will be markedly reduced in primary fibrinogenolysis while normal or slightly shortened in DIC. Finally, the absence of a high concentration of D-dimer in primary fibrinogenolysis versus its elevated concentration in DIC [1]. Once active bleeding develops, it is tough to distinguish between the two

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entities since fibrin is obtained as an action of thrombin activation and the lysis of fibrin produces D-dimers [3].

The best approach in patients with primary fibrinogenolysis secondary to malignant disease is often an aggressive treatment of the underlying malignant condition. A study by Kulić *et al*. described bleeding as a presenting sign of primary fibrinogenolysis in a 64-year-old patient with prostatic cancer [4]. Furthermore, a study performed by Li *et al*. reported significant gingival bleeding as a presenting finding of primary fibrinogenolysis [2]. Crissman *et al*. highlighted an amniotic fluid embolism due to obstetric abnormalities in a 29-year-old woman diagnosed with primary fibrinogenolysis rather than DIC [5].

In contrast to DIC, anti-fibrinolytic medications such as aminocaproic acid or Tranexamic acid are favored remedies for primary fibrinogenolysis [3]. Transfusion support with cryoprecipitate can also be provided for severe hypofibrinogenemia. If DIC has developed, anti-fibrinolytic agents without systemic anticoagulation (like heparin) are contraindicated due to the potential risk of increased microvascular thrombosis [1]. Caution should be exercised to balance chemotherapy-related bone marrow suppression with bleeding complications due to fibrinogenolysis.

In conclusion, the significantly reduced fibrinogen and increased fibrin/fibrinogen split products (FSP) and D-dimer levels are diagnostically significant. Coagulopathy should always be taken into account in diagnostic practice. With the rising frequency of cancer, primary fibrinogenolysis secondary to some malignancies may likely emerge in clinical studies. Finally, precise diagnosis is critical in patients with primary fibrinogenolysis and clinical presentation for perfect management.

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