

HEREDITARY FACTOR X DEFICIENCY UNMASKED BY NECROTIZING HEMORRHAGIC PANCREATITIS: A CASE REPORT

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Background. Factor X (FX) deficiency is one of the rarest coagulation disorders, typically manifesting as an autosomal recessive trait. While clinicians often suspect an acquired deficit in the presence of severe systemic inflammation or major necrotic events, such as necrotizing hemorrhagic pancreatitis (NHP), the possibility of an underlying genetic condition must be considered when the coagulopathy appears disproportionate to the clinical state. NHP creates a high-risk environment for both massive hemorrhage and thrombosis. This case report discusses the diagnostic journey of a patient whose critical illness led to the discovery of a familial coagulation defect, fundamentally changing the therapeutic approach.

Case Report. A 72-year-old male (born 11/11/1953) was transferred from the Gastroenterology department following severe NHP. His condition was complicated by a Resuscitative Cardiopulmonary (RCP) event, resulting in a vast peripancreatic hematoma and splenic vein thrombosis. Laboratory tests showed a PT ratio of 1.24 and an aPTT ratio of 1.53. Specific assays revealed a Factor X activity of 26%. Initially, an acquired deficiency was strongly suspected due to potential factor consumption within the hematoma or proteolytic degradation by pancreatic enzymes. However, to clarify the etiology, a familial screening was conducted. Surprisingly,

both of the patient's daughters were found to have a similar Factor X deficiency, shifting the diagnosis from acquired to hereditary Factor X deficiency. To identify the causative mutation, genetic analysis via sequencing of the F10 gene is currently ongoing. Despite the hemorrhagic risk posed by the vast hematoma and the genetic deficit, the patient required anticoagulation for the splenic vein thrombosis. He was started on Fondaparinux. Currently, the patient is stabilized and awaiting a follow-up abdominal CT scan to monitor the hematoma's evolution and the efficacy of the anticoagulation in the context of this newly identified genetic risk factor.

Conclusions. This case demonstrates that while necrotizing hemorrhagic pancreatitis can exacerbate coagulopathy, an unexpectedly low Factor X level (26%) should prompt an investigation into the patient's genetic background. The discovery of the same deficit in both daughters confirms a hereditary rather than a purely acquired etiology. Genetic testing, which is currently underway to identify the specific F10 gene mutation, and family screening are essential in these rare scenarios to provide accurate diagnosis, genetic counseling, and tailored hematological management for both the patient and their relatives. Frequent radiological monitoring remains the gold standard for navigating this complex clinical equilibrium.