

## CLINICAL AND LABORATORY FEATURES OF CHANGES IN THE COAGULATION SYSTEM IN PATIENTS WITH CHRONIC PANCREATITIS

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### **Relevance**

Chronic pancreatitis is a long-term inflammatory and degenerative disease of the pancreas accompanied by exocrine and endocrine insufficiency, systemic inflammation, metabolic disorders and microcirculatory disturbances. In prolonged and recurrent disease, toxic-metabolic factors such as alcohol consumption and smoking, as well as hepatobiliary comorbidity, may lead to multidirectional changes in the blood coagulation system. Hemostatic disorders in chronic pancreatitis may manifest either as hypercoagulation or hypocoagulation, increasing the risk of thrombotic and hemorrhagic complications.

### **Aim of the Study**

To study the clinical and laboratory features of changes in the coagulation system in patients with chronic pancreatitis and to identify the main factors associated with hypercoagulation and hypocoagulation.

### **Materials and Methods**

The study included 112 patients with chronic pancreatitis and a control group of 30 healthy individuals. According to coagulation parameters, patients were divided into three groups: Group 1 — hypercoagulation (n=38), Group 2 — hypocoagulation (n=32), and Group 3 — no coagulation disorders (n=42). Complete blood count, platelet count, ESR, biochemical parameters, liver function tests, glucose, HbA1c, APTT, prothrombin time, INR, fibrinogen, D-dimer and soluble fibrin-monomer complexes (SFMC) were assessed.

### **Results**

Chronic pancreatitis was most common in working-age individuals, particularly in the 45-59-year age group, which accounted for 48.2% of cases. Patients with hypocoagulation had a higher mean age ( $52.4 \pm 10.8$  years), indicating an association with longer disease duration and a more severe clinical course. No statistically significant sex differences were found between the groups, although men predominated slightly in all groups (56-62%).

Etiological analysis demonstrated the leading role of toxic-metabolic factors in the development of coagulation disorders. Alcohol consumption was detected in 62.5% of

patients with hypocoagulation, 57.9% with hypercoagulation and 28.6% without coagulation disorders ( $p=0.002$ ). Smoking was most frequent in the hypocoagulation group, reaching 71.9% ( $p<0.001$ ). Smoking more than 10 cigarettes per day significantly increased the risk of coagulopathy ( $p=0.044$ ). The combined effect of alcohol and smoking was observed in 56.3% of patients with hypocoagulation and 42.1% with hypercoagulation, confirming its role as a major risk factor ( $p=0.001$ ).

Peptic ulcer disease was detected in 25.0% of this group, increasing the risk of bleeding. Obesity was identified in 28.9% of patients with hypercoagulation and may contribute to a prothrombotic state through visceral adipose tissue. Disease duration was closely related to coagulation status: in Group 3, duration of up to 5 years was observed in 85.7%, whereas in Group 2, duration of 5-10 years was 43.8% and more than 10 years was 18.7%, indicating gradual decompensation of hemostasis during long-term disease. Clinical manifestations were more pronounced in patients with coagulopathy. Persistent abdominal pain was registered in 71.9% of patients with hypocoagulation, 68.4% with hypercoagulation and 40.5% without coagulation disorders ( $p=0.003$ ). Steatorrhea, reflecting severe exocrine insufficiency, was detected in 62.5%, 55.3% and 31.0% of these groups, respectively ( $p=0.004$ ).

Laboratory findings showed anemia and thrombocytopenia in the hypocoagulation group: hemoglobin was 118.6 g/L, erythrocytes  $3.8 \times 10^{12}/L$  and platelets  $192.8 \times 10^9/L$ . In the hypercoagulation group, reactive thrombocytosis was observed, with platelet count reaching  $285.6 \times 10^9/L$ . Biochemical tests in Group 2 confirmed liver dysfunction: total protein 62.8 g/L, albumin 32.4 g/L, bilirubin 32.4  $\mu\text{mol}/L$ , GGT 124.6 U/L and alkaline phosphatase 242.6 U/L. Carbohydrate metabolism disorders were also more pronounced, with glucose 7.4 mmol/L and HbA1c 6.8%. Coagulation analysis revealed two opposite hemostatic patterns. In Group 1, shortened APTT (26.4 s), increased fibrinogen (4.8 g/L), elevated D-dimer (846.4 ng/mL) and increased SFMC (8.6 mg/100 mL) confirmed a prothrombotic state. In Group 2, prolonged APTT (44.8 s), prolonged prothrombin time (20.6 s), increased INR (1.8) and decreased fibrinogen (2.1 g/L) indicated a high risk of hemorrhagic syndrome. Inflammatory markers were highest in the hypocoagulation group: C-reactive protein 24.8 mg/L, IL-6 16.8 pg/mL and IL-8 36.4 pg/mL, confirming the close relationship between systemic inflammation and coagulation disorders.

### **Conclusion**

Changes in the coagulation system in patients with chronic pancreatitis are multifactorial and are associated with alcohol consumption, smoking, disease duration, recurrence frequency, liver pathology, exocrine insufficiency and systemic

inflammation. Hypercoagulation is characterized by shortened APTT and increased fibrinogen, D-dimer and SFMC levels, indicating thrombotic risk. Hypocoagulation is associated with prolonged APTT and prothrombin time, increased INR, decreased fibrinogen and hepatobiliary dysfunction, which increases the risk of hemorrhagic complications. Comprehensive assessment of coagulation tests, inflammatory markers, liver function parameters and clinical factors is essential for early detection of coagulopathy, risk stratification and development of individualized treatment strategies.

**Keywords**

Chronic pancreatitis, hemostasis, coagulopathy, hypercoagulation, hypocoagulation, APTT, INR, fibrinogen, D-dimer, inflammatory markers.