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ALCOHOL-ASSOCIATED AGRANULOCYTOSIS, SEPSIS RISK AND CRITICAL ANEMIA: DIAGNOSTIC CHALLENGES OF INFLAMMATORY MARKERS IN CHRONIC ALCOHOL USE

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Background. Chronic alcohol use suppresses hematopoiesis and neutrophil function, and through gastric mucosal injury, malnutrition and malabsorption promotes vitamin B12, folate and other B-vitamin deficiency. In profound neutropenia, conventional inflammatory indicators become unreliable, complicating early recognition of sepsis.

Case presentation. A 33-year-old man with five months of heavy alcohol use, reduced intake for one month and prior opioid substitution treatment was transferred from an addiction clinic with somnolence, abdominal pain, fever, spontaneous ecchymoses and visual hallucinations. On admission he was jaundiced, tachycardic (110/min) and tachypneic (23/min) with SpO2 91%, yet temperature was only 37.2 °C. Laboratory tests showed WBC 0.95×10^9/L with absolute neutrophil count 0.47×10^9/L, hemoglobin 9.6 g/dL, MCV 108.8 fL, platelets 14×10^9/L, CRP 263 mg/L, total bilirubin 78.6 μmol/L, GGT 993 U/L and respiratory alkalosis (pH 7.53). Imaging revealed hepatomegaly with steatosis, bilateral basal consolidation and colonic wall thickening; head CT excluded an acute intracranial process.

Management and course. Hematology consultation confirmed severe neutropenia, thrombocytopenia and macrocytic anemia. Given the high risk of sepsis, empirical ceftriaxone and vancomycin were started with red cell and platelet transfusions, electrolyte correction, thiamine and intensive monitoring. Neutrophils recovered to 3.25×10^9/L by hospital day 3, and by day 4 WBC was 7.78×10^9/L and platelets 112×10^9/L, while CRP fell from 263 to 34 mg/L by day 8. Lipase peaked at 869 U/L despite unremarkable pancreatic imaging. Hectic fever on days 7–8 prompted blood and stool cultures. The patient improved and was discharged after nine days.

Discussion. Leukocyte count and fever, the usual triggers for sepsis evaluation, were unreliable: leukopenia reflected marrow suppression rather than absence of infection, and fever was blunted.

CRP was the most useful marker, but its specificity was limited by concurrent alcohol-related liver injury, colitis and pancreatic enzyme elevation. Macrocytosis, steatosis and rapid neutrophil recovery favored an alcohol-associated mechanism, likely compounded by nutritional deficiency: alcohol-related gastritis impairs intrinsic factor and B12 absorption, and malabsorption and poor intake deplete folate and other B vitamins (levels not measured). Infection and corticosteroids may also have contributed. Home-produced alcohol adds methanol risk, as folate deficiency slows formate clearance; absent metabolic acidosis argued against significant toxicity.

Conclusion. In chronic alcohol users with agranulocytosis, sepsis should be presumed until excluded. Serial CRP trends, interpreted together with clinical findings, imaging and cultures rather than as isolated values, should guide early empirical therapy, nutritional repletion and supportive care.

Keywords: alcohol; agranulocytosis; sepsis; C-reactive protein; critical anemia.