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Tubulointerstitial Nephritis in a Patient with Ulcerative Colitis

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INTRODUCTION/OBJECTIVES: Ulcerative colitis (UC) is a chronic inflammatory bowel disease that affects rectum or extends to other parts of the colon. It has a relapsing and remitting course, usually extending over years. It also has extra-intestinal manifestations, including renal, like tubulointerstitial nephritis (TIN). TIN is an inflammatory disease that affects renal tubules and the interstitium. Other causes include drugs and toxins. The first line of treatment for UC is 5-aminosalicylic acid (mesalazine). One of the rare complications of mesalazine is also TIN.
CASE PRESENTATION: A 24-year-old male with past medical history of UC was referred to nephrology department because of an elevated serum creatinine level (530 µmol/L, normal range 64-104 µmol/L), with unremarkable urinalysis. UC was diagnosed two years before and since then he has been on oral mesalazine. Renal biopsy showed interstitial fibrosis and tubular atrophy with inflammatory infiltration in 90% of the parenchyma. Possible differential diagnosis was TIN due to mesalazine. Mesalazine has been left out of therapy and has been replaced with methylprednisolone (dose 0.5mg/kg/day). After a few months, levels of creatinine significantly decreased (259 µmol/L). Methylprednisolone was gradually tapered to a dose of 4-8mg/day with stagnant levels of creatininemia.
CONCLUSION: Chronic kidney disease caused by TIN remains a significant finding in patients with UC. It is a diagnostic and therapeutic challenge, since it could be a consequence of mesalazine therapy or an extra-intestinal manifestation of UC. In patients with UC, renal function parameters (serum creatinine and urine) should be monitored regularly.

CR46
Upper GI bleeding in a patient with rare inherited bleeding Disorder – a case report

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Keywords: bleeding, gastrointestinal, Glanzmann's thrombasthenia, thrombocytopathy

INTRODUCTION/OBJECTIVES: Glanzmann thrombasthenia(GT) is a rare inherited thrombocytopathy characterized by insufficient platelet aggregation and normal platelet count. The genetic molecular feature of GT is deficiency or dysfunction of the platelet integrin αIIbβ3(CD41/CD61) receptor for fibrinogen, resulting in bleeding episodes of varying severity. In general, the presence of mucocutaneous bleeding and a normal platelet count raise the suspicion of this disorder. We report a case of gastrointestinal (GI) bleeding in a patient with GT.
CASE PRESENTATION: A 56-year-old woman was admitted due to melena. Her past medical history revealed GT and severe menorrhagia. Her physical examination was unremarkable. Her laboratory results showed normal platelet count (261 x 109/L), iron deficiency anemia (Hb 80 g/L; Fe 6 µmol/L, ferritin 18,4 µg/L), normal fibrinogen (3,3 g/L), PT (0.99) and APTT (21.6 seconds). Flow cytometry revealed reduced surface expression of CD41(0,5%) and CD61 (0.5%). Her upper endoscopy (UE) was unremarkable; colonoscopy showed old traces of blood, while capsule endoscopy revealed some fresh post-bulbar bleeding. Repeated UE showed active arterial duodenal bleeding and the hemostasis was successfully achieved by clipping. Because of anemia red blood cells transfusion and intravenous iron supplementation were administered. Hemostasis was achieved with platelet transfusions, along with tranexamic acid. In addition, lower factor XIII (FXIII) level was detected which was supplemented as well.
CONCLUSION: GT is a rare inherited bleeding disorder characterized by impaired platelet aggregation. Therapy is supportive and essential in acute or chronic bleeding and in preoperative management. GI bleeding in GT patients is challenging and requires a combination of different modalities.