CASE REPORTS

Massive Lower Gastrointestinal Bleeding from Cecal Lymphoid Polyp in Renal Transplant Recipient

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Abstract

A 25-year-old renal transplant recipient receiving anticoagulant therapy for renal vein thrombosis, presented with massive lower gastrointestinal bleeding. Superior mesenteric angiogram revealed active bleeding in the cecum. Colonoscopy revealed a sessile ulcerated polyp in the cecum with satellite polyps. The polyps were fulgurated with Nd:YAG laser. Colonoscopy later revealed a remnant of the polyp, which was excised. The histopathology revealed a lymphoid polyp.


Keywords: Immunosuppression, angiography, laser, anticoagulants.

Introduction

Lower gastrointestinal (GI) bleeding is an infrequent but life-threatening complication in renal transplant recipients.1-2 Its common causes include infections, pseudomembranous colitis, ischemic colitis, uremic colitis, idiopathic ulcers and neoplasms.3-4 Post-transplant lymphoproliferative disorders (PTLD) occur in the setting of exogenous immunosuppression following organ transplantation.5 Their early recognition has important therapeutic implications.6

We report a renal transplant recipient on immunosuppression and anticoagulant therapy for renal vein thrombosis, who presented with massive lower GI bleeding from a lymphoid polyp in the cecum.

Case Report

A 25-year-old male renal transplant recipient on immunosuppression (azathioprine, prednisolone and cyclosporine) had developed renal vein thrombosis confirmed by venography and was on anticoagulation therapy (heparin followed by warfarin). One month following the initiation of anticoagulant therapy, he was admitted to the hospital with massive lower GI bleeding. Hemoglobin was 8 g/dL and prothrombin time was more than one minute. Hemodynamic stabilization was achieved with packed red blood cells and intravenous fluids. Emergency superior mesenteric artery (SMA) angiogram, showed normal vascular anatomy and active extravasation of the contrast from a branch of the ileocolic artery (Fig). Selective arterial vasopressin infusion was started at the rate of 0.2 units per minute for 20 minutes. The bleeding persisted and failed to subside despite further infusion of vasopressin at 0.4 units per minute for the next 20 minutes. At this stage the patient was advised surgery. The patient refused and conservative treatment was continued. Twenty-four hours later SMA angiogram was repeated with superselective cannulation of the feeding vessel for embolization. The bleeding appeared to have ceased and hence embolization was not done. The patient continued to have melena. Upper GI endoscopy was normal.

On the seventh day after the onset of bleeding, colonoscopy was performed. A 10 to 12 cm sized polyp covered with blood clots and three small satellite polyps were seen in the cecum just below the ileocecal valve. After obtaining biopsy, all four polyps were coagulated with 0.2 sec pulses of 50 watt Nd:YAG laser (M88 Medilas II, Munich). Colonoscopy was repeated two weeks later after prothrombin time had become normal. The larger polyp had regressed to 5 mm in size and was removed with a hot biopsy forceps. The satellite polyps had

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resolved completely. Histopathological examination revealed submucosal lymphoid polyp. IgM antibodies for CMV were present in the serum.

The patient remained well without anticoagulation and on immunosuppression therapy for two months, when he developed left sided hemiplegia. Computed tomography scan revealed a small, enhancing space-occupying lesion suggestive of tubercular granuloma in the right parietal cortex. He was treated empirically with etambutol, rifampicin, isoniazid and pyrazinamide. Two months later the patient developed graft rejection which was confirmed by renal biopsy. He died of renal failure eight months after renal transplantation.

Discussion
A review of English medical literature by Stylianou et al had revealed 38 (0.9%) patients with lower GI bleed among 4066 renal transplant recipients. The mortality rate of 55% to 88% in this review confirms the serious nature of this complication. A majority of bleeding episodes occurred within the first four months after transplantation, when the incidence of rejection episodes is highest and renal function is still recovering. Immunosuppressive therapy during this period is the most intense. For these reasons, lower GI bleeding in renal transplant recipients must not be viewed in the same manner as that in the previously healthy subjects.

The usual causes of lower GI bleeding following renal transplantation include colitis from opportunistic organisms, ischemic colitis, uremic colitis and pseudomembranous colitis. Bleeding from idiopathic colon ulcers, diverticular disease and angiodysplasias has also been reported. Though primary colon lymphoma is rare, renal transplant recipients are at a higher risk of developing it. A large proportion of patients with renal failure and many renal transplant recipients exhibit coagulation abnormalities which often contribute to GI bleeding.

The unusual feature in this patient was the presence of a lymphoid polyp while he was on intensive immunosuppressive therapy. Lymphoid depletion rather than lymphoid proliferation is usually seen in animal studies and in renal and hepatic allograft recipients. Use of cyclosporin immunosuppressive therapy for graft preservation has been associated with PTLD. PTLD can involve the GI tract as a common extranodal site. Atypical lymphoid proliferation appearing in tissues of transplant recipients should raise the possibility of lymphoma. Epstein-Barr virus and cytomegalovirus have been implicated in the causation of B cell proliferation in the lymphoid tissues of immunosuppressed transplant recipients.

This probably is the first report of a cecal lymphoid polyp in an immunosuppressed patient, presenting with colonic bleeding as a result of excessive anticoagulation.

References