Case report

A case of severe hemorrhagic cystitis following bone marrow transplantation

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Abstract. Hemorrhagic cystitis (HC) is a common adverse effect caused by the preparative regimens for bone marrow transplantation. Unfortunately, an effective therapy for HC has not yet been established. Here we report a case of severe HC in which the upper as well as the lower urinary tract were severely affected, resulting in right ureteral obstruction and a contracted bladder. Hyperbaric oxygen therapy was effective in suppressing the severe HC, but the cause of HC could not be identified. Five years after the bone marrow transplantation the patient underwent augmentation ileocystoplasty with ileal ureteral replacement.

Key words: Bone marrow transplantation, Hemorrhagic cystitis, Hyperbaric oxygen therapy, Ileocystoplasty

Introduction

Hemorrhagic cystitis (HC) following bone marrow transplantation (BMT) is a common regimen-related toxicity and sometimes life-threatening. Early HC, which occurs within 72 hours of the preparative regimen, is usually related to cyclophosphamide use, while late HC, which occurs more than 72 hours after initiating the preparative regimen, is frequently results from a viral infection such as adenovirus, cytomegalovirus, and BK virus [1, 2]. Although many medical studies have investigated treatments for HC, an effective therapy has not yet been established [1, 2]. Here we report a case of severe HC, in which irreversible organic changes of the urinary tract were induced, and reconstructive surgery using the ileum was performed.

Case report

A 32-year-old male patient presented with gross hematuria, pain on micturition, pollakisuria, and lower abdominal pain 225 days after an allogeneic BMT from his sibling donor, for acute lymphoblastic leukemia. Ultrasonography revealed a blood clot in the bladder and right-sided hydronephrosis. Intravenous pyelography also demonstrated mild to moderate right-sided hydronephrosis (Figure 1A). Extensive hyperemia, bleeding and edema in the bladder mucosa were observed by cystoscopy and histological examination of the bladder biopsy (Figure 3 and 4A). Both urine cytology and bacterial culture were negative. Despite symptomatic treatment, pollakisuria and micturition pain rapidly progressed. To address the pollakisuria, an indwelling bladder catheter was used, while the intractable bladder pain was controlled by epidural anesthesia. A hemostatic was unable to control the gross hematuria, so hyperbaric oxygen therapy at 1.5× atmospheric pressure for 105 minutes per day was administered to the patient on the weekdays. Thereafter, both gross the hematuria and bladder pain gradually improved. After 3 months of hyperbaric oxygen therapy, the patient’s hematuria disappeared. The right-sided hydronephrosis progressed, however, for which a percutaneous nephrostomy was required (Figure 1B) and the bladder contracted to a capacity of 20 ml (Figure 2A). An antegrade pyeloureterogram enabled
us to observe a near complete obstruction of the right lower ureter (Figure 2B). The HC subsided, however, and the patient’s performance status gradually improved from grade 3 to 1. Five years after the BMT the patient underwent an augmentation ileocystoplasty with ileal ureteral replacement according to Studer’s procedure, opening the native bladder up wide in the sagittal plane. After surgery, contrast media flowed easily through the reconstructed ureter (Figure 2C) and the bladder.

Figure 1. (A) Intravenous pyelogram at the time of initial diagnosis. (B) Right hydronephrosis was aggravated during hyperbaric oxygen therapy.

Figure 2. (A) Cystogram just before surgery showing the contracted bladder with bilateral grade 1 vesicoureteral reflux. The bladder capacity was only 20 ml. (B) Antegrade pyeloureterogram just before the reconstructive surgery. The right lower ureter was almost completely obstructed, as shown by the arrow. (C) Contrast media injected from the ureteral catheter flowed smoothly through the reconstructed ureter after surgery.