Case Report

Massive post-obstructive diuresis in a patient with Burkitt’s lymphoma

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Introduction

Burkitt’s lymphoma, which originates from B-cells, is commonly a disease of children, although it is also frequent amongst immunodeficient adults. It is the non-endemic form that affects adolescents and young adults, usually presenting with abdominal disease causing constipation, pain, nausea and vomiting [1]. This tumour, one of the very aggressive lymphomas, has the highest growth rate of all of them, and it responds well to chemotherapy.

Acute renal failure occurs frequently in Burkitt’s lymphoma, and uric acid nephropathy is its most common cause.

We report here on a case of Burkitt’s lymphoma with acute obstructive renal failure, the relief of which resulted in massive polyuria, reaching 901 l a day.

Case

A 30-year-old man was admitted to our hospital because of abdominal pain, constipation, anuria, fatigue, loss of weight, excessive sweating and fever. Three weeks previously, an abdomino-pelvic computed tomography (CT) scan, which had been performed at the hospital he was admitted to because of a sudden attack of abdominal distention and severe pain, had revealed multiple enlarged mesenteric and para-aortocaval lymph nodes and marked thickening of the wall of the terminal ileum. Biopsy specimens obtained via colonoscopy had shown atypical lymphoid cell infiltration consistent with a high-grade B-cell lymphoma in the cecum. Because of his progressive, severe abdominal distention and deteriorating general status, the patient had undergone an extended right hemicolectomy, side-to-side ileo-transvers colostomy and omentectomy were performed. The pathologic examination of the resected material had disclosed malignant lymphoma, predominantly of the small cleaved-cell type. A week after the operation, the patient was transferred to our hospital for further treatment.

The patient noted oliguria starting a few days before the aforementioned operation, which progressed to anuria the day before he was admitted to our ward. His physical examination revealed a blood pressure of 220/120 mmHg, trace peripheral oedema, and a generalized distention of both lower abdominal quadrants and a vaguely outlined mass in the right lower quadrant. No signs of chronic hypertension were present. The bladder was not palpable. It was not possible to examine the rectum beyond its distal 4 cm.

His initial laboratory evaluation disclosed the following: a serum creatinine of 7.3 mg/dl, blood urea nitrogen 50 mg/dl, uric acid 15.4 mg/dl, glucose 63 mg/dl, sodium 137 mEq/l, potassium 5.4 mEq/l, chloride 97 mEq/l, phosphorus 7.3 mg/dl, calcium 9.2 mg/dl, total protein 6.5 g/dl, albumin 3.2 g/dl, lactic dehydrogenase (LDH) 715 U/l (normal 240–480 U/l), and liver enzymes within the normal limits. His blood count showed 504 000 platelets per cubic mm and 12 900 white blood cells, with 10 900 granulocytes and 1500 lymphocytes, 9.6 g/dl haemoglobin. A bone marrow biopsy revealed a normocellular marrow with normal maturation.

Arterial blood gases showed metabolic acidosis with an increased anion gap. A renal sonogram showed bilateral grade III hydronephrosis, normal-sized kidneys with normal cortical thickness and increased parenchymal echogenicity.

The patient was anuric, was unresponsive to loop diuretics, and had no urine flow after insertion of a urinary catheter into his bladder. While a drainage
procedure was being considered under the presumptive diagnosis of acute renal failure due to obstructive uropathy, therapy with methylprednisolon, 80 mg a day, and allopurinol, at a dose of 600 mg the first day followed by 300 mg daily, was started. Since the patient’s urinary output increased remarkably with steroid administration, there appeared to be no need for a drainage procedure—it was 4 l on the second day and 7.9 l on the third. Serum creatinine decreased from 7.3 mg/dl to 5.4 mg/dl. The ultrasonography performed on the third day showed marked regression of the hydronephrosis bilaterally. On his fourth day in the hospital, we decided to start chemotherapy with cyclophosphamide, doxorubicin, vincristine and prednisolone at doses, respectively, of 750, 50, 1.4 and 100 mg per square m of body surface (‘CHOP’ regimen). His urinary output, which was 27.501 the first day, increased progressively, and reached its peak of 88.48 l on the third day of the chemotherapy. Alkaline isotonic saline infusion was administered through a large-bore intravenous access. Daily physical examinations and body weight measurements did not show any signs of overhydration. His renal function tests returned to normal within a week (Figure 1). His urinary output, which was at its maximum on the third day of CHOP treatment, declined progressively to 4.6 l on the ninth day. The abdominal ultrasonogram showed no signs of hydronephrosis, and the abdominal CT scan disclosed a remarkable decrease of the size of the enlarged lymph nodes.

After this obstructive episode, the patient underwent eight courses of Hyper-CVAD therapy (fractionated cyclophosphamide, vincristine, doxorubicin and dexamethasone). His renal function 29 months after the acute episode remains stable.

Discussion

Obstruction is encountered in 2–10% of acute renal failure cases. Although the incidence of obstruction declines with age, it is more common in some groups, such as patients with certain types of cancers [2]. Tumours can cause obstruction by compressing the urinary tract from outside, by invading it or the retroperitoneal space, or by causing uric acid nephropathy, which occurs either spontaneously or as a result of chemotherapy—especially if the tumour load is high. Uric acid nephropathy occurs when the kidneys are exposed to increased loads of filtered insoluble crystalline substances [2]. It is the most common cause of renal failure in high-grade lymphomas [3,4].

The bilateral hydronephrosis in our anuric patient and the multiple enlarged lymph nodes displayed on the imaging studies raised the suspicion of an obstruction. There was no evidence of lymphomatous infiltration with enlargement of the renal parenchyma. Involvement of the urinary tract or the infiltration of the kidneys with lymphomas have been reported, but they are rare complications that cause renal failure [5,6]. In a study of 37 patients with Burkitt’s lymphoma, 14 had azotaemia, and evidence of extrinsic compression of the urinary tract was found in three patients with stage III and IV lesions. Also, evidence of obstruction was seen in seven patients who had advanced stage lymphoma but did not have renal failure [4,7]. In another study of 384 patients with lymphomas, obstructive uropathy was found in 7.1% of them [8].

Tumour lysis syndrome was also considered as a possible cause of acute renal failure in our patient, since the blood changes, namely hyperuricaemia, hyperphosphataemia, hyperkalaemia and a high LDH, could be attributed to tumour lysis syndrome in a patient with a highly proliferating tumour. He was unresponsive to diuretic agents, and corticosteroid administration followed by chemotherapy (‘CHOP’ regimen) caused complete relief of the obstruction of the urinary tract with a post-obstructive polyuria reaching 90 l a day. The duresis in our patient was massive. Our PubMed search of the literature in English revealed one case in which the relief of a unilateral obstruction resulted

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**Fig. 1.** Urinary output, fluid intake and creatinine values starting with the first day of steroid treatment.
in an excessive post-obstructive diuresis of more than 58 l per day [9]. The sonogram and CT scan of our patient showed a complete resolution of hydronephrosis after the treatment, which was compatible with the initial diagnosis of obstruction. Since the urinary uric acid/creatinine ratio of our case is not available, it cannot be excluded that uric acid nephropathy contributed to some extent to the acute renal failure.

Release of complete bilateral renal obstruction is usually followed by substantial diuresis. Several factors are presumed to make important contributions to post-obstructive diuresis: the osmotic diuretic effect of retained, and poorly reabsorbable, solutes; the reduction in tubular reabsorbing capacity resulting from tubular damage; the activation of natriuretic factors following extracellular fluid volume expansion; and increases in the production of renal prostaglandins E2 and F2 production are among those factors [2,10,11]. Tubular damage, which impairs the ability to concentrate urine, is not responsive to ADH. Decreased production of ADH-dependent cAMP is thought to be the reason for this disturbance [2]. Urinary output usually returns to normal when extracellular volume and composition return to normal. The recovery of renal function after relief of obstruction depends on the duration and degree of obstruction, whether the obstruction is unilateral or bilateral and whether there is a concomitant infection [10].

In replacing the fluid output in polyuria, care should be taken that the replacement volume does not exceed two-thirds of the daily urinary output, to avoid iatrogenic extracellular volume expansion [12]. The choice of an appropriate fluid therapy must be based on serum and urine electrolyte status; and generally, sodium chloride-containing solutions are the replacement fluids of choice. To avoid hypothermia, fluids should be warmed up to body temperature before infusion. As tumour lysis syndrome was also taken into consideration in our patient, alkaline isotonic saline was used.

In conclusion, obstruction can be a cause of acute renal failure in cases of high-grade lymphomas, and energetic treatment can reverse this potentially life-threatening condition. Relief of urinary tract obstruction may result in massive diuresis, which needs a meticulous management with appropriate fluids and electrolytes, as was done in the case of our patient.

Conflict of interest statement. None declared.

References

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