Acute Renal Failure Associated with Rhinosinuso-Orbital Mucormycosis Infection in a Patient with Diabetic Nephropathy

C. Utaş
K. Ünlühizarcı
T. Ökten
Y. Ünlü
M. Doğanay
F. Keleştimur

Department of Nephrology, Erciyes University Medical School Hospital, Kayseri, Turkey

Dr. Cengiz Utaş, Erciyes Üniversitesi Tip Fakültesi, Nefroloji Bilim Dah, TR-38039 Kayseri (Turkey)

Dear Sir,

Mucormycosis is an acute and often fatal infection caused by fungi of the family Mucoraceae and can present in different clinical forms. The infection is rare and typically occurs with an increasing frequency among patients exposed to cytotoxic and immuno-suppressive treatments. Debilitating diseases, most commonly poorly controlled diabetes mellitus, are the other predisposing factors [1-3]. Conditions associated with metabolic acidosis also enhance fungal growth and favour infection by Mucor Sp. which is usually a saprophytic, non-pathogenic fungus [1, 2]. Although in patients with renal failure mucormycosis develop [1-4, 5], to our knowledge mucormycosis leading to acute renal failure is not described elsewhere.

The patient with diabetic nephropathy presented herein developed oliguric acute renal failure associated with rhinosinuso-orbital mucormycosis and was treated successfully. A 60-year-old woman with a history of right-sided orbital headaches during the last week was admitted to the hospital. She had noted a decrease in urine output and nausea 2 days before admission. She had type 2 diabetes of 13 years’ duration. Her only regular medication was NPH insulin which had been started 6 months earlier when diabetic nephropathy was diagnosed. At examination the patient appeared ill and she had a blood pressure of 100/80 mm Hg. The temperature was 37 °C, the pulse was 104/min. Stupor and acidotic respiration were observed. Because of metabolic acidosis and hyperkalaemia due to oliguric acute renal failure, peritoneal dialysis was performed. Urine analysis revealed urinary osmolality of 334 mosm/l and 200 mg/day protein with normal microscopy without cellular casts. Fractional excretion of filtered sodium was 4%. Haematocrit was 28%, white blood cell count was $5.6 \times 10^9$/l with 65% neutrophils, 30% lymphocytes, 2% eosinophils and 3% monocytes. The platelet count was $412 \times 10^9$/l. The erythrocyte sedimentation rate was 35 mm/h. The blood urea nitrogen was 25.3 mmol/l (normal range 2.9-8.9), the creatinine 389 µmol/l (normal range 53-133), the glucose 10.5 mmol/l and the HbA1c 11% (normal < 6%).
Total serum protein was 76 g/l (normal range 60-80) and serum albumin was 42 g/l (normal range 38-48). Serum calcium was 2.2 mmol/l (normal range 2.1-2.6), phosphorus 2.3 mmol/l (normal range 0.84-1.45) and the uric acid 392 µmol/l (normal range 137-393). Sodium was 135 mmol/l, potassium 7.2 mmol/l and chloride 101 mmol/l. The liver enzymes and bilirubin were within normal limits. Funduscopic examination revealed bilaterally proliferative diabetic retinopathy. The Waters graphy for the right-sided orbital headaches, oedema, erythema and proptosis of 23 mm showed right maxillary sinusitis and the computed tomographic scan demonstrated right maxillary sinusitis, right orbital proptosis and retro-orbital soft tissue infiltration involving the musculus rectus medialis. Internal and external ethmoidectomy and surgical débridement were performed. All infected tissue, necrotic and polypoid material were excised. The cultures of the surgical specimens were negative for bacteria and there was a growth of mold on fungal cultures, but specific identification could not be made. The histopathologic examination revealed inflammation, thrombosis and invasion by branching, non-septate hyphae, specific for the diagnosis of mucormycosis. Intravenous administration of amphotericin B (0.25 mg/kg/day) was initiated. After 2 months of therapy, the patient showed both clinical and radiological improvement with proptosis of 18 mm.

Our patient had a 13-year history of type 2 diabetes, and clinical diabetic nephropathy with persistent proteinuria and normal serum creatinine had been diagnosed 6 months earlier. She has developed acute oliguric renal failure which is superimposed on pre-existing renal insufficiency. No known cause of acute renal failure was found in the patient. Therefore it can be postulated that mucormycosis infection might be the trigger mechanism of acute renal failure in this patient with diabetic nephropathy and poorly controlled diabetes. The metabolic acidosis due to acute renal failure might be the reason for further growth of fungi and marked enhancement of the clinical impairment.

In systemically ill diabetic patients such as those with acute renal failure, a high degree of clinical suspicion is required for prompt diagnosis of the mucormycosis, to begin early aggressive medical and surgical therapy.

References