A 20-year-old previously healthy Bengali man presented to our hospital with anuria and features of uremic encephalopathy.
Ten days prior to this presentation, he had experienced severe upper abdominal pain and vomiting, and he had been treated in a primary care facility for having a case of acute pancreatitis.
His initial symptoms improved; however, he gradually became anuric and disoriented.
Then he was transferred to our hospital for further management.
At presentation to our hospital, he was severely agitated, restless, and disoriented.
He was tachypneic with acidotic breath.
Mild pedal edema was present; however, his jugular venous pressure was not raised.
His pulse was 112 beats/minute, his blood pressure was 140/90 mmHg, and his body temperature was 98 °F.
Signs of meningeal irritation were absent, and his plantar response was bilaterally extensor.
His fundus could not be evaluated, and examination of his other systems was unremarkable.
His laboratory parameters showed features of renal dysfunction (serum creatinine 13 mg/dl, serum urea 293 mg/dl), raised pancreatic enzymes (serum amylase 249 U/L [reference up to 100 U/L], serum lipase 227 U/L [reference 13–60 U/L), normal liver function tests (serum bilirubin 0.9 mg/dl, alanine aminotransferase 38 U/L, aspartate aminotransferase 35 U/L, alkaline phosphatase 122 U/L, serum albumin 37 g/L), normal potassium (5.1 mmol/L), normal bicarbonate (19 mmol/L), and normal triglycerides (173 mg/dl).
His serological markers, including antinuclear antibodies, cytoplasmic antineutrophil cytoplasmic antibodies, perinuclear antineutrophil cytoplasmic antibodies, C3, and C4, were within normal limits.
An ultrasonogram of his whole abdomen was unremarkable, but non-contrast-enhanced computed tomography (CT) findings were suggestive of acute pancreatitis (Fig.1).
His kidneys were unremarkable, however.
The patient was managed as having a case of AKI and acute pancreatitis.
Urgent hemodialysis was initiated.
After he had received two sessions of hemodialysis, his level of consciousness improved, but he complained of profound visual loss.
An assessment revealed only perception of light.
A funduscopic examination showed retinal whitening and extensive cotton wool exudates as well as Purtscher’s flecken (Fig.2) compatible with Purtscher’s retinopathy.
High-dose parenteral methylprednisolone (1 g intravenously once daily for 3 days) was administered.
For evaluation of renal dysfunction, a renal biopsy was done; the histopathological findings were compatible with renal cortical necrosis (Fig.3).
The patient denied substance abuse or alcohol ingestion.
He did not consume any nephrotoxic drugs or herbal products in the recent past.
No history suggesting connective tissue diseases was available, nor did he have a history of hypotension throughout the course of his current illness.
The patient was finally diagnosed with acute pancreatitis complicated with renal cortical necrosis leading to AKI and Purtscher’s retinopathy leading to complete bilateral blindness.
He died 16 months after his initial presentation as a result of a recurrent attack of acute pancreatitis.
Before that, he had been undergoing maintenance hemodialysis; however, his renal function did not recover, though his vision was improved to finger-counting at 2 feet.