Acute Interstitial Nephritis due to Endosulfan

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Dear Sir,

Acute interstitial nephritis due to drugs present as acute renal failure and may be commoner than is realized. Endosulfan, an organochlorine insecticide has not been previously reported to cause acute interstitial nephritis. We report a case of acute interstitial nephritis and non-oliguric acute renal failure resulting from the ingestion of endosulfan.

A 24-year-old male drank half a glass of beer that had been accidentally contaminated with about 1 ml of endosulfan. Six hours later, he developed nausea, vomiting and dizziness followed by loss of consciousness for 2 h. There were no tremors or convulsions. Physical examination revealed no abnormalities apart from a blood pressure of 180/110 mm Hg. The patient had been diagnosed to have hypertension 4 years previously but had not been compliant with anti-hypertensive therapy. He had not consumed any anti-hypertensive medication or other drugs for 3 months prior to admission. Four days following ingestion of endosulfan, the patient developed facial oedema and loin pain. There was no associated fever, rash or arthralgia. Daily urine output ranged from 1,000 to 1,850 ml. eosinophils. The vessels showed mild hypertensive changes. The patient was managed conservatively. Hypertension was controlled with metoprolol and prazosin. The serum creatinine rose to a peak of 774 $\mu\text{mol/l}$ and subsequently declined to normal level within 2 weeks following ingestion of endosulfan. Eight weeks after discharge, the serum creatinine was 100 $\mu\text{mol/l}$.

Endosulfan is an organochlorine insecticide. Ingestion can cause headaches, nausea, vomiting, restlessness, irritability, vertigo, muscle twitching, confusion, stupor, coma. Investigations showed hemoglobin 14.3 g/dl,

white cell count 9,100/mm³, eosinophils 90/mm³, urea 17.9 mmol/l, creatinine 350 $\mu\text{mol/l}$, uric acid 589 $\mu\text{mol/l}$, and creatine phospho-kinase 40 μl . Urinalysis showed proteinuria of 0.2 g/l, leukocytes 40 $\cdot 10^6/\text{l}$, no red cells, eosinophils, casts, myoglobin and no organisms on culture. Ultrasonography revealed kidneys to be of normal size (10.4 cm) and appearance. Renal biopsy showed an acute interstitial nephritis with normal-looking glomeruli and negative immunofluorescence. The inflammatory infiltrate consists mainly of lymphocytes, plasma cells and occasional and convulsions [1]. Myoglobinuria, acute renal failure and a proximal myopathy following ingestion of 15-30 ml of lindane have been reported [2]. There has, however, been no report of acute interstitial nephritis following ingestion of organochlorine compounds.

This patient had acute interstitial nephritis documented by renal biopsy although he had no associated clinical features such as fever, rash, arthralgia, eosinophilia or eosino-philuria. The serum creatine phosphokinase was normal and there was no myoglobinuria excluding

rhabdomyolysis as a cause for the acute renal failure. Hence, the acute renal failure could be the result of acute interstitial nephritis.

The prognosis for patients who develop drug-induced interstitial nephritis is good. It is important to recognize the condition and discontinue the administration of the suspected drug as failure to do so can lead to progressive renal failure and death [3]. While the place of steroid therapy is unresolved, resolution of the interstitial nephritis and recovery of renal function can occur when steroid treatment is given [4]. This patient improved without steroid therapy.

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