Oxalate Creatinine Ratio in the Peritoneal Fluid of Patients with Primary Hyperoxaluria

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Dear Sir,

Primary hyperoxaluria or oxalosis is a hereditary disorder with an autosomal recessive mode of inheritance and constitutes a rare but important cause of chronic renal failure (CRF) in children. Its diagnosis is important not only for treatment and genetic counselling, but also for kidney transplantation as the disease can recur in the renal allograft [1]. Affected children usually present with nephrocalcinosis and nephrolithiasis, and the diagnosis is confirmed by high oxalate excretion in urine [2]. With declining renal function oxalate excretion decreases, and its urine levels are of little or no diagnostic value in patients with advanced renal failure. In such patients, diagnosis is confirmed by demonstrating birefringent oxalate crystals in kidney, liver or bone marrow biopsy specimens [3, 4].

Over a period of 3 years, we received 4 patients with oxalosis, all presenting with acute or CRF. Plain X-ray of the abdomen in patients 1 and 2, who were twins, was normal and the renal ultrasound examination revealed hyperechogenic kidneys. Percutaneous kidney biopsies in both sisters revealed abundant oxalate deposits. The other 2 patients were older, and abdominal X-ray in both revealed severe nephrolithiasis. Oxalosis was confirmed by kidney biopsies. All 4 patients needed acute dialysis on admission which was followed by continuous ambulatory peritoneal dialysis (CAPD).

Gilboa et al. [5] in 1984 reported a high oxalate concentration in the peritoneal fluid of a 4-month-old infant with oxalosis whereas none was detected in the control. This prompted us to study the oxalate level in the peritoneal fluid of our patients and compare the results with age-matched controls who

were also on dialysis but did not suffer from oxalosis. Oxalate levels were measured by high performance liquid chromatography [6] and in order to accommodate possible individual variations in peritoneal membrane permeability, its level was interpreted in terms of milligrams per gram of creatinine. The mean level in patients was 512.25 mg/g creatinine (range 265-638) as compared to 130.0 mg/g of creatinine (range 64-191) in controls (table 1). The difference was significant by Students' unpaired t test ($p < 0.0003$).

In conclusion, oxalate concentration in peritoneal fluid provides a simple method of diagnosing oxalosis in patients presenting with terminal renal failure. More patients need to be studied before a reference range can be established.

Acknowledgement

We would like to thank Dr. William G. Robertson, Chairman of Biological and Medical Research, King Faisal Specialist Hospital, for helping us with the oxalate measurement in our samples.

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0028-2766/92/

0604-0496\$2.75/0

Table 1

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