Acute Renal Failure Following Intravenous Cholangiography*

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SUMMARY

A case of acute renal failure following the diagnostic administration of a tri-iodinated compound is reported. The clinical findings and course are presented, with particular emphasis on the fact that there appeared to be no underlying or associated disorder which may have caused the renal failure, other than possible mild dehydration.


Tri-iodo compounds are widely used, both orally and parenterally, in order to outline the gall bladder, the biliary tree, the renal collecting system, and blood vessels. They have been implicated in the causation of acute renal failure in a number of cases, although, in relation to the number of investigations performed, the incidence of this side-effect is probably low. There have been a number of cases reported in the American literature. It is our purpose to add another case report, in order to emphasize this rare but serious side-effect of a group of drugs in common use today. In addition, some of the postulated pathogenetic mechanisms will be reviewed.

CASE REPORT

Nine days before admission a 34-year-old male suddenly developed rigors and fever, with nausea, vomiting and abdominal pain. Investigations, including a barium meal and intravenous cholangiogram, were done. For the latter he received 20 ml of a 50% solution of methylglucamine iodipamide (Biligrafin Forte, Schering A.G.), and a normal biliary tree was outlined. The barium meal showed 'evidence of gastritis', but no ulcer was demonstrated. The following day the patient complained of severe headache and pain in the back of the neck, and 24 hours later he had an epileptiform seizure.

He had repeated fits at frequent intervals over the next 4 days. During this period he was hypertensive, with blood pressure readings in the region of 185/115 mmHg. He was confused, with dilated pupils, which were not reactive to light. Phenytoin, diazepam and phenobarbital failed to control his seizures. On the ninth day of his illness, before transfer to this hospital, laboratory investigation results were as follows: blood urea 336 mg/100 ml; serum sodium 127 mEq/litre; potassium 7·8 mEq/litre; chloride 77 mEq/litre; CO₂ content 14 mEq/litre. Serum creatinine was 16 mg/100 ml; calcium 4·8 mEq/litre; inorganic phosphate 10·2 mg/100 ml; SGOT 63 units; SGPT 39 units; LDH 550 units; alkaline phosphatase 6·4 King-Armstrong units; and total bilirubin 0·4 mg/100 ml. Peritoneal dialysis was instituted and was continued for 110 hours. There was a progressive improvement in the patient's clinical state. His course, as measured by haemoglobin, blood urea and urinary volume is depicted in Fig. 1. Blood pressure control was achieved initially with intramuscular hydralazine, and subsequently with decreasing amounts of oral a-methyldopa. With the onset of a diuresis, there was a rapid return to normal. At the time of discharge from hospital, the blood urea was 26 mg/100 ml, serum electrolytes were normal, and the serum creatinine was 3·0 mg/100 ml. At a subsequent follow-up, the serum creatinine was 1·3 mg/100 ml.

A needle biopsy of the kidney was performed on the tenth hospital day (i.e. 16 days after the intravenous cholangiogram). The histological features were tubular cell damage, with evidence of tubular necrosis. A mild lymphocytic and polymorphonuclear infiltration was present in the interstitium around areas of tubular damage. Hyaline and haemoglobinuria-type casts were present in tubular lumens (Figs. 2 and 3). There was mild hyaline arteriosclerosis.

DISCUSSION

In 1968 the Council on Drugs of the American Medical Association published a review of adverse reactions to contrast media. Bethyl-glucamine iodipamide was cited as a cause of these in 10 cases. Reactions were acute and explosive, and included acute renal failure, hepatotoxic manifestations, convulsions and anaphylaxis. Other iodinated compounds had previously been reported to produce renal failure, including iopanoic acid and bunamidyl. The latter has since been removed from the market. In 1965 Setter et al. reported a series of 9 patients with renal failure developing after administration of contrast media.

*Date received: 30 April 1971.
Again bunamiodyl was the drug implicated; however, 4 of these patients, of whom 2 died, were also given methylglucamine iodipamide. In our patient the only preparation given was methylglucamine iodipamide, and it is not possible to attribute the clinical course to any other drug.

Other factors may have contributed to the production of acute renal failure in our patient. He had been vomiting and sweating, and was pyrexial before undergoing the intravenous cholangiogram. Although not documented, it is possible that some degree of dehydration had been present. Dehydration has been incriminated as a major factor in the production of renal failure following intravenous pyelography in myelomatosis, rather than a direct nephrotoxic effect of the contrast media used.

Setter et al. suggested that underlying renal disease predisposed to acute renal failure in a large proportion of their 9 patients, but the blood urea had been recorded as being normal in 6 patients before administration of the drug. In the patient reported here, routine investigation, including an intravenous pyelogram done some months before his present illness, showed no evidence of renal disease.

Another factor which has been considered of importance is excessive dosage or multiple doses at short intervals, of the same or different, related compounds. This could not be implicated in our patient. Other than the possibility of dehydration, there was no evidence of underlying disease, including hepatobiliary disease, which may have impaired excretion of the drug. Thus, the renal failure appears to have been related to a direct toxic effect of the compound used.

Collateral evidence for the condition being due to a direct toxic effect of the drug was the clinical presentation, suggesting an encephalopathy, with headache followed by fits shortly after administration of the contrast medium. This could not be attributed to uraemia at that time. Such a presentation corresponds with other reported cases and could be on the basis of a direct toxic or sensitivity reaction to the drug.

Histological changes present were non-specific, but compatible with tubular necrosis from any cause. The histological changes reported in the literature, in patients with renal failure attributed to contrast media, were similarly non-specific.
We wish to thank Dr M. Salmon, Medical Superintendent of the Johannesburg Hospital, for permission to publish this case report; and the Director of the South African Institute for Medical Research, for facilities.

REFERENCES

The Aetiology of Pneumonia Associated with Measles in Bantu Children


SUMMARY
Antemortem and postmortem lung puncture aspiration was performed in Bantu children with pneumonia associated with measles.

The superinfecting organisms were commonly Staphylococcus pyogenes, but from one-third of the patients Gram-negative organisms were cultured. These organisms were rarely sensitive to ampicillin or streptomycin. Antibiotic therapy should be tailored accordingly.


Acute pneumonia in Bantu children often fails to resolve rapidly on treatment. This poor response to therapy is highlighted in the patient with pneumonia associated with measles.

Cultures taken from nose, throat, sputum and trachea do not necessarily reflect the causative bacteria in lower respiratory tract infection. The latter technique (cultures from the trachea) is more reliable than the others, but difficult in children.

A study has been made of organisms present in the lungs of these children by culture of specimens obtained by percutaneous lung aspiration puncture (LAP).

MATERIAL
Twenty-two Bantu children with measles and pneumonia were studied on admission to the Fever Wards at King Edward VIII Hospital, Durban. In some patients a dose of antibiotic had been given before LAP.

A further 5 cases were studied postmortem. The time lapse between death and LAP varied from 10 minutes to 3½ hours. All cases had received antibiotics for some days before death.

The age range of the patients was 5 months to 4 years.

Method of LAP

The lung aspiration was performed with a sterile 5-ml glass syringe and 18-gauge needle. The site of the LAP was selected over the area of maximal clinical signs, taking care to avoid mediastinum, liver and diaphragm.

The skin over the area was prepared with a phenol or iodide solution.

Two millilitres of serum broth was drawn into the syringe. The needle was advanced rapidly into the thorax to a depth of 2-3 cm and withdrawn immediately, negative pressure being applied throughout the procedure. Thus the lung aspirate was drawn directly into the serum broth. Chest radiographs were done after the procedure.

Postmortem specimens were obtained by puncture into each axilla, using the same technique. In addition, 5 ml of heart blood was obtained.

A drop of lung aspirate was placed on two sterile glass slides, another drop in Loewenstein-Jensen media and the remainder of the syringe contents were replaced in the serum-broth culture bottle. The postmortem cardiac blood was placed in glucose broth media.

Bacteriological Methods

The Department of Microbiology, University of Natal, received the specimens without delay and the following procedure was undertaken: