Acute Renal Failure from *Callilepsis laureola**

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SUMMARY

This article describes the clinical course and management of a patient who developed hyperkalaemic acute renal failure due to a herbal medicine, Callilepsis laureola.

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Acute renal failure due to herbal medicine among Bantu patients is not uncommon. In many of the patients a history of herbal ingestion is not forthcoming except on direct questioning; even if a history is obtained it is difficult to ascertain whether the herbal medicine is responsible as the patient could have taken herbal medicine for symptoms of uraemia once acute renal failure had developed. This report describes a patient who presented with features of hyperkalaemia due to acute renal failure.

*Date received: 31 March 1971.

A Bantu male, aged 42 years, presented with symptoms of pain in both loins and the epigastrium and passage of diarrhoea with blood and pus. He was tentatively diagnosed as a case of amoebic dysentery with amoebic liver abscess and the possibility of pyelitis was entertained; because of the likelihood of pyelitis he was inadvertently treated by the resident medical officer with tetracycline 250 mg *q.i.d.* and potassium citrate 15 ml *t.i.d.* for 4 days (84 mEq potassium daily). Four days after admission he was found to be completely paralysed with no sensory loss; the extremities were cold and clammy and there was bradycardia with a poor pulse volume. He was anuric with a urine volume of less than 100 ml in 24 hours. An electro-

cardiogram showed gross widening of the QRS complex, tall T waves, no P waves and bradycardia (Fig. 1), which were features of hyperkalaemia. He was immediately treated with 20 ml of 10% calcium gluconate intravenously, 40 ml of 4% sodium bicarbonate and 20 units soluble insulin with 50 g dextrose intravenously. This was followed with peritoneal dialysis over 48 hours, as the patient had a serum potassium of 8 mEq/litre and blood urea of 310 mg/100 ml. Twenty-four hours later the serum potassium was 5 mEq/litre and the electrocardiogram was normal (Fig. 1).

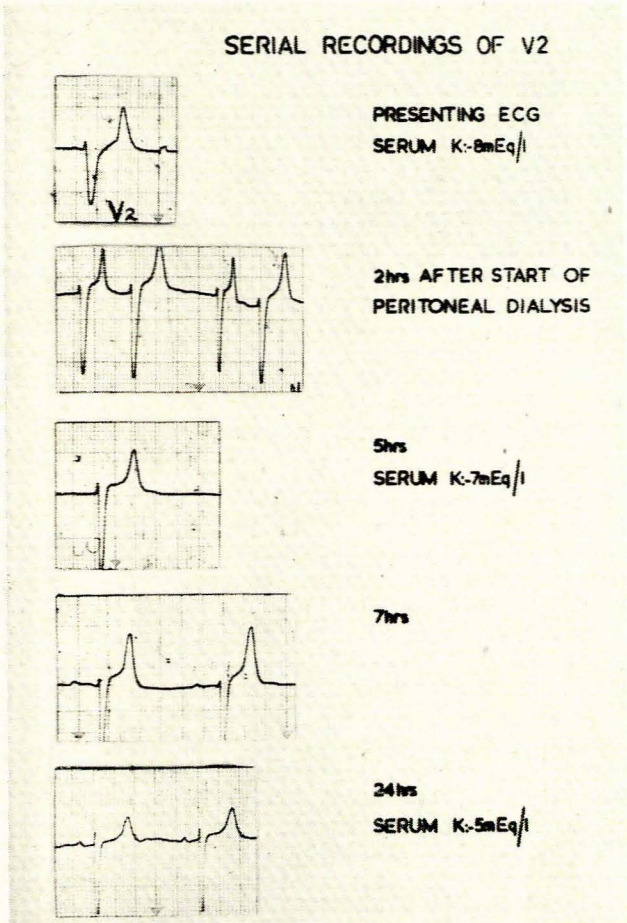

Fig. 1. Serial electrocardiograms of the patient showing changes reverting from hyperkalaemia to a normal state.

On recovery, direct questioning gave a history of his having visited a witch-doctor for impotence; he was prescribed a herbal medicine 'impila', which he took orally after it was boiled in water. The patient did not follow the instructions of the witch-doctor in preparing the herbal medicine. He placed the infusion into one bottle instead of diluting it to fill 8, and thus the dose was at least 8 times greater than prescribed.

The urine contained albumin 2, sugar 1, pus cells 1 and amorphous debris 1. The blood count showed haemoglobin 16·5 g/100 ml, white cell count 11 000/mm³. Erythrocyte sedimentation rate was 4 mm/hour; serum cholesterol was 220 mg/100 ml; the amoebic gel diffusion test was negative; retrograde pyelography excluded postrenal obstruction and showed the kidneys to be of normal size. Urine urea was 56 mg/100 ml, urinary sodium 62 mEq/litre, and urinary potassium 100 mEq/litre; blood sugar 118 mg/100 ml, serum bilirubin 0·7 mg/100 ml, direct Van den Bergh negative, total protein 7·1 g/100 ml, serum albumin 2·1 g/100 ml, serum globulin 5·0 g/100 ml, serum alkaline phosphatase 12 KA units; serum glutamic oxaloacetic transaminase 46 units/ml (normal 9 - 40 units/ml), serum glutamic pyruvate transaminase 5 units/ml (normal 5 - 35 units/ml) and serum lactic dehydrogenase 587/per ml (normal 100 - 500 units/ml). An ammonium chloride assay according to the short test of Wrong and Davies[1] showed that the acidification power of the kidney was normal in that the urinary pH was 4·65 when the pH of the blood was 7·3.

DISCUSSION

Herbal medicine is not an uncommon cause of acute renal failure in the Bantu and this should be considered in all cases presenting with acute renal failure. This case report raises some interesting facts for discussion.

Callilepsis laureola ('impila'), the cause of the acute renal failure in our patient, is a known poison. Zulus take a decoction of the root as a vermifuge and the infusion as a purgative. A paste of the root is used to kill maggots in cattle.[2] Maberley[3] records that the root gathered in winter is a cough remedy and that he has found that the active principle, which is a resin, is useful in the treatment of whooping-cough. No reference could be found by us of the value of 'impila' in the treatment of impotence as occurred in our patient.

The management of hyperkalaemia in acute renal failure is a medical emergency as it is a common cause of sudden death. Because of the electrocardiographic features of hyperkalaemia and the clinical picture of muscular paralysis from hyperkalaemia, our patient was tided over the immediate period with calcium gluconate, sodium bicarbonate intravenously and soluble insulin with intravenous dextrose until the effect of peritoneal dialysis in lowering the serum potassium was obtained. This patient was inadvertently given potassium citrate before it was realized that he was oliguric. This strongly illustrates the point that sodium citrate is a very much safer (and just as effective) urinary alkinizer. It is likely that in our patient the inadvertent use of potassium citrate precipitated the hyperkalaemic manifestations. The use of tetracycline as was done in our case is to be condemned in oliguric states, as tetracycline has an anti-anabolic factor[4] and may have a direct toxic effect on an already vulnerable kidney.[5]

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REFERENCES

1. Wrong, O., and Davies, H. E. F. (1959): Quart. J. Med., **28**, 259.
2. Watt, J. M. and Breyer-Brandwijk, M. J. (1932): *The Medicinal and Poisonous Plants of Southern Africa*, 1st ed., p. 194. Edinburgh: E. & S. Livingstone.
3. Maberley, J. (1905): Report of a Joint Meeting of the British and South African Associations for the Advancement of Science, **3**, 340.
4. Shils, M. E. (1963): Ann. Intern. Med., **58**, 389.
5. Edwards, O. M., Huskisson, E. C. and Taylor, R. T. (1970): Brit. Med. J., **1**, 26.