FATAL ACUTE RENAL FAILURE FOLLOWING COPPER SULPHATE NEPHROTOXICITY

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SUMMARY
A case of acute renal failure due to copper sulphate (blue stone) ingestion for treatment of bronchial asthma is presented. This led to fatal renal failure and probably liver failure as well. With the proliferation of traditional herbal and local cures these cases may increase. This case is being highlighted to bring to notice that these cases exist and also for those promoting traditional cures to see the need for research and for public education on the hazards of some of these cures.

Keywords: Fatal acute renal failure, copper sulphate nephrotoxicity.

INTRODUCTION
Copper is a trace metal in humans and is essential to several human enzymes, such as cytochrome C oxidase. Copper sulphate was once used as an emetic in the management of drug poisoning but has been replaced because of its marked toxicity[1].

Acute copper sulphate poisoning is common in India, and copper is commonly employed as a suicidal poison among the people of India[2]. Copper resembles many heavy metals and toxic effects lead to widespread capillary damage, kidney and liver injury and central nervous system damage[3]. Hemolytic anaemias are also described in acute poisoing in man[4]. Circulatory shock and intravascular hemolysis singly or together may lead to renal tubular injury and death in renal failure[5]. Treatment is largely symptomatic. BAL and penicillamine have been used. Exchange transfusion may be of value. The addition of salt-poor albumin to peritoneal dialysis fluid has also been helpful ridding the blood of copper[6]. Haemodialysis has also been used with mixed results[7].

This paper presents a case of copper toxicity which resulted in death after repeated dialysis and toxicological analysis of postmortem samples revealed copper deposition in various tissues of the body.

CASE HISTORY
This 31 year old man who is asthmatic and has a family history of asthma has been on salbutamol and franol tablets at various times for his attacks of asthma. Two months prior to his admission he was introduced to blue stone (copper sulphate) as treatment for asthma. He had taken 3 such solutions uneventfully. Three days before admission he had an attack of asthma and he took unspecified quantity of copper sulphate solution. During the course of the day he became very weak and started vomiting and passing loose stools associated with abdominal pain. He passed dark urine and soon after became drowsy. He was therefore referred to our unit on the third day of his illness on 26/2/93.

On admission he looked ill, febrile T39°C, pale and icteric. The pulse was 120 per minute fair volume. BP 120/70. The heart sounds were present and normal. The respiratory rate was 30 per minute acidotic type (Kussmaul Breathing). The chest was clear. The liver was 4cm palpable below the costal margin and tender. He was drowsy, responsive to deep pain stimuli by clenching his fists. There was no neck stiffness. There were spontaneous movements of all limbs. The reflexes were present and normal. The following investigations were requested. Full blood count, sickling test, blood film for comment, urea, electrolytes, creatinine, liver function tests, blood cultures, urinalysis, G6PD, blood grouping and cross matching.

Management
He was given intravenous frusemide 80mg start and intravenous normal saline was instituted 500mls three hourly. Serial samples of urine were collected and tested for bilirubin, haemoglobin, sugar and casts. He was put on strict intake and output chart. On the fourth day of admission due to severe uremic symptoms he was started on hemodialysis after a double lumen subclavian catheter was inserted into right subclavian vein. He was also transfused because of his initial haemoglobin of 4gm/dl. He continued to be ill whilst on dialysis and four weeks

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after admission he died suddenly on the ward in spite of judicious dialysis. Urine output, and creatinine levels and progression are depicted in figure 1.

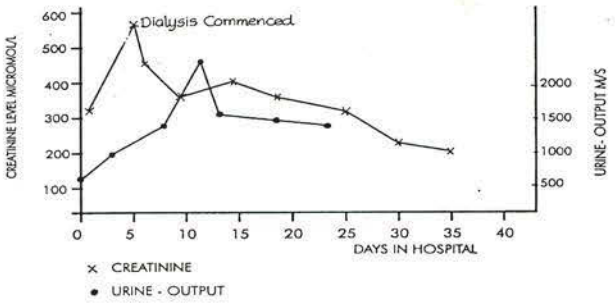

Figure 1 Creatinine and urine output over days

URINALYSIS	SERUM BILIRUMBIN
Haemoglobin+++	Direct 47 umol/L
Protein ++	Indirect 54 umol/L
Ketones trace	Blood culture – No growth

Ultrasound: Normal non-obstructive kidneys

Autopsy report revealed kidneys of normal weight, pale, oedematous with extensive petechial haemorrhages extending to subcapsular surfaces. The bladder showed sandy patches and the right ureter was dilated. The liver was flabby with petechial haemorrhages at the subcapsular areas.

DISCUSSION

This case demonstrates the dangers of persistent use of traditional cures for various ailments in the tropics and particularly Ghana.

Adu et al[9] in 1981 described cases of nephrotoxic renal failure due to African traditional herbal remedies but none of these were of metal aetiology as in this case. Agarwal et al[10] from India reported 19 out of 66 cases of acute poisoning as due to copper sulphate poisoning. The common features in this group were: acute renal failure, intravascular hemolysis, jaundice, hepatocellular toxicity and circulatory collapse. Ahasan et al[11] from Southern region of Bangladesh reported on a number of cases of copper sulphate poisoning with a high mortality 24.9%. Hepatotoxicity, acute renal failure and gastrointestinal bleeding were the main complications.

Our patient presented with uraemia and intravascular hemolysis and needed several blood transfusions for treatment of his illness. He died in spite of repeated dialysis. Postmortem material for toxicological studies revealed copper deposition in the liver and brain at high concentrations. In retrospect one could suggest that a contributory factor to his demise was the liver involvement which was difficult to detect in this clinical setting. Incidentally he had signs of vesical schistosomiasis which was revealed at autopsy. Traditional medical cures have been with us for a long time and new ones are being added and we suggest a national organised approach to this problem to prevent increase cases of nephrotoxic renal failure.

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