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POTASSIUM DICHROMATE AS A WITCHDOCTOR'S REMEDY

FIVE CASES OF POISONING

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Chromic acid and its alkaline salts have many industrial uses particularly in tanning, dyeing and printing[1]. Workers in these occupations are exposed to a fine dust, or mist, of chromates which, before the days of adequate protection, produced ulcers of the nasal septum ('chrome holes') or dermatitis of the exposed skin. There is also evidence that chrome workers are more susceptible to carcinoma of the lung than the general population[2]. In contrast to their local action on skin or mucous membranes, chromates act as virulent poisons when they are absorbed into the body. Since Wilson[3] described the first case in 1844, many reports of accidental or suicidal chromate poisoning have appeared in the literature.

In this paper it will be shown that chromate poisoning is also seen in South Africa and that it occurs in an unusual manner: amongst Africans potassium dichromate is regarded as an effective remedy for many diseases. It is not only dispensed by witchdoctors throughout the country, but it is freely used by laymen as an emetic and a purgative. It is often taken in massive dosage and then rapidly causes death, but where less of the substance is absorbed it produces an illness whose true nature may escape recognition, since the patient forgets or withholds the fact of having taken the poison.

CASE REPORTS

Case 1: G.M., an African male aged 20 years, had consulted a European doctor about a week before admission for a bout of vomiting. He returned 4 days later, this time jaundiced, and he was then admitted to hospital.

Examination revealed a very ill young man. He was stuporose, answered questions in a confused manner and grimaced when examined. He hiccoughed continuously. He was deeply jaundiced, his sclerotics being a deep golden colour. The abdomen was distended; the lower edge of the liver could not be felt and its upper border was not displaced. The tongue and skin were dry. Temperature 99°F, pulse rate 100 per min., blood pressure 140/60 mm. Hg. There were no burns in the mouth or throat. The urine contained 3 red blood corpuscles per high-power field, with bilirubin and an excess of urobilin present; specific gravity 1010.

On the 2nd day the patient's breathing became fast and hissing (Kussmaul in type), and a fine urea frost appeared on his brow. For the next 7 days there was little change in his clinical state. The temperature, jaundice, mental clouding, hiccough, hissing breathing, urea frost and tympanites persisted; from time to time he shivered violently. On the 4th day he passed a pasty stool; on the 5th day he developed oedema of the feet and an erythematous rash appeared on his arms and thighs.

On his 8th day in hospital he showed clinical improvement; the mental clouding lifted, he hiccoughed less frequently, his breathing became normal and his jaundice less evident. By the 16th day he was quite well; his skin was desquamating in the distribution of the rash.

Laboratory Studies. Traces of chromium were found in the urine and gastric contents on admission, but none could be detected 3 weeks later. The urinary output was 0·5 l. on the first day and 0·5—1·3 l. daily for the next 10 days. At the 30th and 40th day the specific gravity after 18 hours of fluid restriction was 1023 and 1027 respectively. On admission the blood-urea level was 348 mg. % (Table I) and this fell to normal limits in 2 weeks; the CO_2-combining power of the blood ranged between 39 and 29 c.c. for the first 14 days and became normal 4 weeks after admission. During the first week blood-potassium levels were low but other electrolytes were not disturbed. The level of alkaline phosphatase was high on admission; the liver-function tests though abnormal did not change significantly with clinical recovery. Aspiration biopsy of the liver performed on the 14th day revealed moderate haemosiderosis but no other significant pathological change. Malaria, yellow fever and Weil's disease were excluded by appropriate blood tests.

Treatment For the first week what little nourishment the patient took by mouth consisted of milk and a solution of 1/6 molar lactate. He was maintained on intravenous fluids, 5% dextrose-water, 5% dextrose-saline and 1/6 molar lactate, the quantity restricted to 1 l. in excess of the daily urinary output. In addition he received vitamin K, aureomycin, choline, methionine and BAL.

Summary A young man presenting with the picture of hepato-renal failure was found to have traces of chromium in the urine and gastric contents. He recovered and was apparently left with no permanent renal or hepatic damage. The patient and his relatives steadfastly denied that he had taken potassium dichromate or any Native medicine.

Case 2: J.T., an African male aged 33 years, had received *ndonya* (potassium dichromate crystals) from a witchdoctor 6 days

previously and shortly after drinking a solution of the crystals had vomited violently. He had passed no urine for 48 hours before admission.

On examination he showed similar clinical features to case 1, namely hiccough, Kussmaul breathing, and abdominal distention. He differed only in being less jaundiced and mentally confused. His bladder contained about 150 c.c. of urine. Chromium was detected in both gastric contents and urine. He was uraemic and acidotic, but his blood-potassium level—unlike that of case 1—lay within normal limits (Table I). He deteriorated rapidly. Within 3 days he became drowsy and more deeply jaundiced; he remained persistently anuric, urea frost appeared about his face and he died on the 4th day after admission. As in case 1, there were no burns about the mouth or throat.

From the 1st day he was fed with 1 l. of Bull's emulsion[4] through an intragastric drip and was treated with vitamin K, aureomycin, choline, methionine and BAL. On the third day, ACTH, 20 units 6-hourly, was administered by intravenous drip and this was continued until his death.

Post mortem Examination (*Dr. V. Kemp*). The positive macroscopic findings consisted of a few petechial haemorrhages in the stomach, bladder and renal pelves. There were no burns in the mouth or oesophagus and there was no erosion of the gastric mucosa.

Histological Examination (*Dr. B.J.P. Becker*). (1) Sections of the kidney showed the presence of acute tubular necrosis affecting both upper and lower nephrons. The lumen of the tubules was occupied by intensely-staining eosinophilic granular casts. (2) Sections of the liver showed the presence of focal necrosis with many bile thrombi and bile casts; the necrosis was massive in places. Non-necrotic portions of the liver lobule showed feathery degeneration and focal fatty change. (3) Sections of the lung showed no significant pathological change.

Chemical Analysis (*Dr. B. W. Marloth*):

Organ and weight	*Chromium calculated as potassium dichromate*
Liver, 1,050 g.	0·90 gr.
Kidneys, 320 g.	0·23 gr.
Stomach and contents, 300 g.	0·14 gr.
Intestines (large and small), 2,086 g. ..	0·47 gr.
Total	1·74 gr.

Case 3. M.M., an African male aged 50 years, had been given some medicine by a witchdoctor 5 days before admission which he used as an enema. Shortly afterwards he started purging; within 1 or 2 days his stools became bloody and he began to vomit. On examination he showed the now-familiar signs of jaundice, urea frost, drowsiness, 'acidotic' breathing, hiccough and abdominal distention; on the 5th day in hospital he developed a maculopapular rash over his arms and legs. In addition he showed certain features not previously encountered: at the height of his illness he bled from the gums and mouth, developed a brisk haematuria and his haemoglobin level fell from 12·1 g % on admission to 7·0 g. % within a week (Table I). His urinary output was never less than 0·5 l. daily and he recovered in 3 weeks.

In this case the exereta were not analysed and the 'medicine' was not available but the patient was certain that it had contained *ndonya*.

Case 4. W.T., an African male aged 30 years, had taken a black pill given him by a friend to cure a stomachache one week before admission. He complained of vague pains and that his 'eyes were yellow'. He resembled the previous cases in being jaundiced and confused but was never uraemic or acidotic. His urinary output remained over 1 l. per day and his urine contained bilirubin but no cells or casts. Though chromium was detected in the urine he could not remember experiencing an episode of vomiting or purging. He was treated like case 1 and although his general state improved he remained jaundiced for 3 months and was almost certainly developing cirrhosis of the liver when he was discharged.

Case 5. H.S., an African male aged 45 years, had drunk a solution containing ¼ teaspoonful of crystals given him by a witchdoctor 24 hours before admission; 5 minutes later he vomited 4 times, and during the night vomiting recurred and he collapsed. He had not passed urine during the 24 hours since taking the medicine. Apart from drowsiness no abnormality was found on physical examanation and there were no burns of the mouth or throat. His bliadder contained about 60 c.c. of urine which showed 20 red blood corpuscles and a few granular casts per high-power field; blood urea was 90 mg. %. The crystals produced by his wife were identified on analysis as potassium dichromate.

He was treated by Bull's method and started passing urine the following day. Within a week he was well and his blood urea had returned to normal limits (Table I).

DISCUSSION

Several post-mortem examinations on cases of chromate poisoning are recorded in the literature[5, 6, 7]. The microscopic findings are not striking. They consist of petechiae and superficial erosions of the stomach, and ochre discolouration of the liver and kidneys due to chromic oxide. Where the poison has been used as an enema, similar superficial or deeper lesions are found in the rectum[8]. Macroscopically, the chief lesions are found in the kidneys, which show all degrees of damage from degeneration to necrosis, and in the liver, which usually shows degeneration. The clinical and pathological findings in the 5 cases reported here confirm that potassium dichromate may produce degrees of renal and hepatic damage ranging from the reversible to actual death of tissue. Moreover the action of the poison does not appear to be corrosive in the mouth, stomach or rectum.

The clinical features of poisoning vary with the amount of chromium absorbed and the following description is

TABLE I. LABORATORY INVESTIGATIONS IN 5 CASES OF POTASSIUM DICHROMATE POISONING

Case Number	*Hospital Day*	*Blood Urea (mg.%)*	*Serum Bilirubin (mg.%)*	*CO_2 Combining Power (c.c.)*	*LFT*			*AP*	*Blood Sugar (mg.%)*	*PI%*	*Serum Na (mg.%)*	*Serum K (mg.%)*	*Hb (g.%)*	*WBC*	*ESR*
					TT	*TF*	*TA*								
1	2	348	16	39	1·5	—	+++		105	85	335	14·8	16·2	9,300	
	6	119		29	1·0	—	+++		105	94	335	14·4			
	28	16	9	38						100	357	22·0			
	36		2·7	49	1·0	—	++				336	18·8			
2	1	428	3·6	29							300	25·0	17·0	11,400	7
3	1	470	24										12·1	10,200	
	7	120	6										7·0	13,700	
4	7	21	16·2					21·5	110	55	305		15·6	11,900	33
	90	39	18·0					22·2		95					4
5	1	134													
	3	66													
	6	20													

LFT = liver function tests. TT = thymol turbidity (units). TF = thymol flocculation. TA = Ucko's modification of Takata-Ara reaction. AP = alkaline phosphatase (units %). PI = prothrombin index. Hb = haemoglobin. WBC = white blood cells (per c.mm.). ESR = erythrocyte sedimentation rate.

based on case records in the literature and the experience of these 5 cases:

A large amount of poison rapidly produces uncontrollable vomiting and diarrhoea which proves fatal within a few minutes or hours; these cases of acute poisoning are usually first seen by pathologists.

The patients more likely to be encountered by clinicians are those who have absorbed less poison and have recovered from the episode of vomiting and purging. The mildest cases develop transient oliguria and azotaemia, due to slight renal damage or a state of extra-renal uraemia (case 5 in this study.) Those who have sustained more serious hepatic or renal damage develop jaundice or hepato-renal failure after the lapse of 5—7 days. In these delayed, or subacute, cases the bout of vomiting and diarrhoea is apparently not always severe, as in case 4 in the present series.

The diagnosis rests on a history of taking the poison, the findings described, and identification of chromium in gastric contents, faeces or urine. In cases 1 and 2 chromium was still detectable in the urine and gastric contents 5—7 days after taking the poison and in spite of considerable vomiting. The history of taking potassium dichromate may not be forthcoming in Africans for 2 reasons: firstly, the patient or his relatives may be covering some person unqualified to administer the drug and, secondly, the chromate may have been so disguised by the addition of herbs that the patient is ignorant of the contents of the witchdoctor's 'medicine'.

The differential diagnosis in cases of subacute poisoning is that of jaundice, or jaundice in association with uraemia (hepato-renal failure). Haemolytic jaundice can be excluded by a blood count (anaemia, reticulocytosis) and the presence of bilirubinuria; obstructive jaundice due to gall-stones or neoplasm is not commonly seen in this hospital. It is therefore reasonable to consider poisoning as a probability in every case of jaundice in Africans, particularly when it is associated with marked azotaemia. In the past 3 years several cases of hepato-renal failure have been seen in this hospital for which no adequate explanation has been found clinically or at post-mortem examination. Since liver disease is common amongst Africans[9] it is possible that they develop hepato-renal failure more commonly than white races; in such cases, however, the possibility of poisoning cannot be definitely excluded since potassium dichromate may produce so few characteristic lesions at autopsy.

Judging by the outcome in these 5 cases, the immediate prognosis seems to depend on the urinary output. In case 2 the patient was persistently anuric and suffered such extensive renal and hepatic damage that it is doubtful whether he would have benefited by no-matter-what therapy. The patients who recovered all had reasonably good urinary outputs. That chromium may produce chronic or progressive parenchymatous changes is borne out by Major's case[6], in which death eventually took place from uraemia, and case 4 in this series, in which jaundice was still present 3 months after the poison was taken. As corrosion is not a prominent post-mortem feature and since chromium is found in the stomach for several days after poisoning, gastric lavage seems to be indicated when the poison has been taken by mouth.

The following information about potassium dichromate has been gathered from witchdoctors, African patients and vendors of African medicines. Known as *ndonya* potassium dichromate is an old-established African remedy. How it gained a place in the armamentarium of witchdoctors, who use mainly herbal remedies, is not known; presumably it was introduced by European chemists at one time. Certainly it is a powerful emetic and purgative and this, together with the attractive orange colour of the crystals, may be the reason for its popularity amongst the Africans.

Witchdoctors consider it an effective remedy for many of the diseases which they diagnose. It is mixed with herbs and crushed into a grey powder which is taken by mouth or as an enema; sometimes it is mixed with copper sulphate and heated over a fire; those who cannot afford to consult a witchdoctor prepare a solution of the crushed crystals in warm water which they drink or use as an enema. Witchdoctors apparently are aware of its poisonous properties and use it cautiously, but the method and dosage employed in preparing their medicines is so haphazard that it seems to be a matter of chance whether a patient will receive a lethal dose or not.

The frequency of chromate poisoning in this country can be roughly assessed from various sources. At the Government Chemical Laboratories, Johannesburg[10], a survey was made of all deaths from poisoning in South Africa from 1948 to 1952. It was found that, after arsenic, chromium was the commonest metallic poison, that it caused 21 deaths in that period, and that chromate poisoning was increasing in frequency. In 1953 the Johannesburg inquest magistrate dealt with 15 inquests on deaths from chromate poisoning[11]. The 5 cases reported in this paper were encountered over a period of 3 years but the last 2 were seen in the space of one month and doubtless many cases of 'jaundice' have escaped true diagnosis.

Potassium dichromate is widely used by Africans in this country; of the 5 patients reported here one came from Natal and another from the Northern Transvaal. So great is the demand for it that one wholesale manufacturer of African remedies buys the drug by the hundredweight. It is available to the public, as are also copper sulphate, cantharides and mercury, at Native chemists' and at shops with a predominantly African clientele.

CONCLUSION

Apart from its action as an emetic and a purgative, potassium dichromate has no therapeutic value. There can be no justification for allowing the sale of so dangerous a drug when many safe purgatives are available on the market. Its use by witchdoctors has been condemned from the bench[11] and it seems that the time has come for official action to protect ignorant Africans from this and similar 'remedies'.

SUMMARY

1. In South Africa potassium dichromate is used as a medicine by African witchdoctors and laymen and commonly produces poisoning.

2. The clinical and laboratory features of subacute poisoning are described in 5 patients, including results of a liver biopsy in one case and post-mortem findings in another.

3. The differential diagnosis is discussed. It is suggested that in all cases of hepato-cellular jaundice in Africans potassium dichromate poisoning should be considered.

4. The high incidence of chromate poisoning in South Africa is indicated and it is suggested that the sale of this and similar 'remedies' be restricted.

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