in Zürich, Switzerland, for giving his permission for the publication of this article; to Dr. R. Hess, Head of the EEG Department in Zürich for providing the EEG reports; and to Prof. Dr. D. G. Steyn, Head of the Department of Pharmacology at the Pretoria University, for his interest in the matter.

REFERENCES

ACUTE PORPHYRIA IN A BANTU MALE
A CLINICAL AND CHEMICAL REPORT

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Acute porphyria was first described by Ranking and Pardington in 1890, and since that date numerous cases of this uncommon metabolic disturbance have been described in many parts of the world.

Waldenström by 1937 had found 89 cases in the literature, and to these he added his own 103 Swedish cases. With the exception of five from the United States of America all these had occurred in Europe. Since then many accounts of single and small groups of cases have been recorded from many sources. South African cases have been reported by Kooy, Linder and Barnes.

Porphyria in man occurs in three syndromes:
1. The first is the rare congenital porphyria characterized by photosensitivity of the skin, pigmentation of the bones and teeth and passage in the urine of uroporphyrin type I. The symptoms are recognized in infancy.
2. The second is the more common acute porphyria which is manifested by susceptible persons in the form of recurrent attacks often apparently precipitated by periods of stress. Clinical symptoms, which vary considerably from case to case, include acute abdominal or neuritic pain; constipation; vomiting; muscular wasting, weakness or paralysis and variable psychotic manifestations. Not all of these need be present. The urine during attacks contains porphobilinogen and porphyrin, usually in considerable amount though the latter may only become evident on standing. The porphyrin was regarded as being uroporphyrin III, but more recent investigations indicate that several of these pigments are present.
3. The third type, porphyria cutanea tarda, is characterized by a dermatitis, frequently bullous in type, which becomes manifest in adult life. The pink or brownish urine contains porphyrin but no porphobilinogen. Neurological and psychotic symptoms are not present.

Some cases have been reported which appear to be a combination of the last two forms, e.g. Gray et al.

The case to be described falls into the second category and, as the clinical picture varies, some additional facts will be described. The classical symptoms have already been mentioned, but it must be stressed that they do not necessarily occur simultaneously, and nervous features in particular may never be present. The urine, which is usually port wine in colour, may be normal when passed and darken to almost blackness on exposure to sunlight. The abdominal pain, often accompanied by visible peristalsis, may, in the absence of neurological symptoms and discoloration of the urine, be attributed to an abdominal catastrophe. Waldenström mentions 29 cases of acute porphyria in which a laparotomy was performed. Constipation is more usual, but diarrhoea may be present in some cases.

The nervous symptoms usually do not fit into any definite syndrome. The two common manifestations are either a condition resembling Landry's paralysis, or a bizarre type of polyneuritis. These may be accompanied by varying psychological disorders, particularly confusion, hallucinations or occasionally coma. At times the psychological manifestations overshadow the physical accompaniments. Hypertension is a common finding.

The mortality rate is about 50%; but where the nervous system is involved it is thought to be even higher.

CASE REPORT

On June 17 1950 a Native male, aged 24 years, was admitted to the King Edward VIII Hospital, Durban, complaining of abdominal pain and weakness of all his limbs, especially his arms.

History. The man stated that three weeks before admission he had developed severe abdominal pain. The pain was intermittent and moved all over the abdomen, being associated with vomiting and hicouche. Neither constipation nor diarrhoea had been present. Two weeks after the abdominal pain had started he developed pain in the legs, which was followed shortly by weakness and difficulty in walking. At this stage his hands became tremulous and he developed a wrist drop.

Chronologically, the history of sensory changes is rather vague, but he suffered from pains in the arms and back...
of the neck, and also numbness in his legs. At the height of his illness he had visual disturbances, was unable to talk, and was incontinent of urine and faeces.

While he did not volunteer the information, later questioning elicited the fact that his urine had been red from about one week after the onset of his abdominal pain. There was no history of the taking of any medicine before admission.

Past History. In August 1948 he had been admitted to this Hospital suffering from abdominal pain and had passed red urine. No diagnosis was made. This was the only previous occasion on which he had had these symptoms and he only noticed that his urine was red on this and on the previous admission.

Physical Examination. He was a thin, intelligent Swazi male of average height. With the exception of the nervous system and skin, all the other systems were normal, and the blood pressure was 130/90.

Central Nervous System. The cranial nerves were all intact.

Upper Limbs. There was a bilateral wrist drop, the following muscles being completely inactive: Extensor carpi radialis longus and brevis, brachioradialis, extensor digiti communis, extensor pollicis longus and brevis, and abductor pollicis longus. There was weakness of the intrinsic muscles of the hands. All other muscles of the upper limbs were normal.

Sensory Changes. While there was symmetrical analgesia to pin-prick up to both elbows, light touch, joint and vibration senses were normal.

Reflexes. The supinator was absent but the biceps and triceps jerks were brisk and equal.

Lower Limbs. Both legs were thin and very weak, but there was no loss of movement, though tone was reduced.

Sensory Changes. There was analgesia to pin-prick up to the mid-calf on both sides, and a zone of marked hyperaesthesia, starting one inch above Poupart’s ligament and extending four inches down the thighs, completely encircling the body. Other forms of sensation were normal.

Reflexes. The knee jerks, ankle jerks, and abdominal reflexes were brisk and equal. The plantar response was flexor.

Skin Changes. There were three small areas of depigmentation, 1-2 mm. in diameter on the wrists, these had apparently been caused by small pimples which had discharged white matter. On the ankles there were five areas of thin skin about 1 cm. in diameter covering old ulcers which had formed spontaneously in 1939.

Psychological Manifestations. No unusual behaviour was noted at any time and consciousness was unclouded up to the end of his life.

Other Investigations. The urine when first seen showed a striking salmon-pink colour and the presence of porphyrin was suspected. Spectroscopic examination showed the presence of metal porphyrin complex and the porphobiligenin reaction was strongly positive. Further examination of the urinary porphyrin is described later. The lead content of the urine was not significantly increased. Blood sugar, cytology and chemistry of the cerebrospinal fluid, were normal and the Wassermann reaction was negative. No abnormal findings were reported on radiological examination of the chest and of the alimentary tract after a barium meal. A blood count showed 13.3 gm. per cent of haemoglobin, 4.1 million erythrocytes per c.mm., mean corpuscular volume 85.3, 6,600 leucocytes per c.mm. Punctate basophilia was not observed.

PROGRESS

Within a few days of admission his urine had lost its characteristic colour, but still showed a faint brownish tinge, and darkened considerably on standing. Improvement was slow but steady, and 10 days after admission the power in his legs had increased considerably. The condition of his arms was unchanged, but the abdominal pain was much reduced. Two weeks later all sensory changes had disappeared. The abdominal pain had practically gone, and he complained only of occasional twinges. He was walking well.

On 24 July, 38 days after admission, he was discharged at his own request. While the wrist drop persisted, there was evidence that the intrinsic muscles of both hands were stronger.

On 8 August he was seen again, and on this occasion there was definite improvement in the condition of his wrists. The brachioradialis, extensor digiti communis, extensor carpi radialis longus and brevis were still paralysed, but the extensor and abductor pollicis longus were acting weakly, and the intrinsic muscles of both hands showed good function.

On 18 October he was re-admitted complaining of abdominal pain, vomiting and pain in the legs. For the previous three days he had been constipated. There was considerable improvement in the power of his arms and legs. The wrist drop had disappeared and detailed examination showed that with the exception of the extensor carpi radialis longus and brevis, all the other muscles were functioning well.

Sensory changes had reappeared in the same form and distribution as before, but were very mild. The urine was red in colour and gave positive chemical and spectroscopic tests for porphyrin.

Over the next 12 days his condition improved considerably but on 30 October he suddenly relapsed. The wrist drop returned overnight with weakness of all his limbs and for the first time phonation became impaired. The original area of hyperaesthesia became intensely painful.

Apart from one brief period of improvement his condition steadily deteriorated. He was always able to move his legs but his arms became paralysed and his voice a faint whisper. The abdominal pain persisted and was only relieved by morphine. Constipation was extreme and repeated enemas were the only means of evacuating the bowel.

Later the diaphragm became paralysed and on 18 November he developed a terminal bronchopneumonia and died on 21 November, five weeks after re-admission.

Post-Mortem Examination. Apart from confirming the presence of the terminal bronchopneumonia there was little of note except recent superficial ulceration of the duodenum. Sections were made of liver, bone marrow, brain and spinal cord but these also showed nothing remarkable.
TREATMENT
There is no known specific treatment for this disease. Abrahams has suggested that Kaolin may be of value as it absorbs porphyrin. This was used during both attacks but as it had no apparent effect during the second its contribution to his recovery on the first is open to question.

Treatment was directed into two channels. Firstly, to relieve abdominal pain, Kaolin, alkalis and belladonna were tried. These were ineffective and Pethidine and morphine had to be substituted. Secondly, in an effort to check the metabolic disorder the following drugs were tried: all the vitamins of the B group including B12, vitamins E and C, crude liver extract, glycine and landrax. As is apparent, no success was obtained with any of the drugs employed.

CHEMICAL EXAMINATION OF THE PORPHYRIN

Direct spectroscopic examination of a 24-hour specimen of urine sent by post from Durban to Johannesburg showed the two absorption bands at 580 and 540 m, characteristic of a metal-porphyrin complex; on addition of hydrochloric acid the spectrum reverted immediately to that characteristic of acid porphyrin. A fairly strong band was present at 500 m, in both the untreated and acidified specimens of urine. Schlesinger's reaction for urobilin was only very faintly positive whence it is inferred that the 500 m band was mainly due to porphobilinogen. The positive porphobilinogen reaction was negative but this was inconclusive in view of the long delay in transit. As stated earlier this reaction was strongly positive on fresh specimens of urine examined in Durban.

Quantitative determinations were made of the coproporphyrin extracted by ether and of the porphyrin adsorbed on a calcium-phosphate precipitate. The 24-hour excretions were: coproporphyrin - 130 g., phosphate-adsorbed porphyrin 7.7 mg.

Continuous collection was then made for a period of nine days, at first the urine was paler in colour than when the diagnosis was originally made but darkened again towards the end of this period.

On adjusting the pH of the urine to 3.1 no flocculent material separated, but on the addition of alkali a discoloured sediment of calcium salts settled which was separated and sent to Johannesburg for further treatment.

This sediment was centrifuged and the supernatant urine showed no porphyrin on spectroscopic examination after acidification with hydrochloric acid. The precipitate was dissolved in sufficient 2 N hydrochloric acid to convert the porphyrin to its acid form, filtered and the porphyrin flocculated out by adjusting the pH to 3.1. At this and subsequent stages, chromatographic analysis on filter paper strips was performed, the results of which are discussed later.

The flocculent material was collected on a filter paper, dried and esterified in methyl alcohol containing 2% sulphuric acid. The ester was recovered in chloroform solution in the usual manner, washed thoroughly with dilute ammonia and several successive quantities of distilled water. On concentrating to small bulk and adding warm methyl alcohol, a moderate crop of crystals separated slowly from a strongly coloured mother liquor. After recrystallization twice from chloroform-ethyl alcohol typical fine acicular crystals were obtained which melted at 253-255°C, which corresponds closely to the figure 257-258°C for Waldenström's uroporphyrin III methyl ester.

The mother liquors from all three crystallizations were pooled, dried, moistened with methyl alcohol and an excess of approximately 6 N hydrochloric acid added. After standing at room temperature overnight sodium acetate and sodium hydroxide solutions were added until the mixture was no longer acid to Congo red. One-fifth volume of glacial acetic acid was added and the mixture shaken with two portions of ether which removed part of the porphyrin. Both these fractions were recovered in flocculent form from aqueous solution at pH 3.1 and samples chromatographed. The remainders were esterified but while the ether insoluble fraction yielded crystals similar to the main crop of uroporphyrin ester the ether soluble fraction did not crystallize.

Strip chromatograms were made of the recovered porphyrin before and at different stages during the partition into fractions. Chromatograms of the total porphyrin showed a marked preponderance of uroporphyrin and a smaller quantity of coproporphyrin thus confirming the qualitative determination recorded above. The mother liquors after removal of the uroporphyrin-ester crystals still contained both these porphyrins but the latter was present in relatively much higher concentration.

The two fractions into which the mother liquor pigments were divided by partition with ether showed mainly coproporphyrin in the ether soluble and uroporphyrin in the ether insoluble fraction, but each was contaminated with a trace of the other. All the strips showed faint spots suggesting the presence of very small quantities of porphyrins with three, five, six and seven free-carboxyl groups. Further examination of these could not be carried out as the amount of material available was inadequate.

DISCUSSION

As indicated earlier acute porphyria is not so rare a metabolic anomaly as to be dismissed from clinical consideration. At the time this case was encountered its occurrence in a Negro had not been reported although the diagnosis had been made in a number of cases in white-skinned South Africans. While the investigation of this case was in progress a fatal case in an African Negro student in London was reported and a third presented at the Far East Rand Hospital, Springs, details of which will be reported elsewhere.

Unless the possibility of acute porphyria is in mind, the findings presented by this type of case are very bewildering. The salmon-pink colour of the urine was suggestive and the positive porphobilinogen reaction and spectroscopic evidence of the porphyrin metal complex clinched the diagnosis.

The combination of abdominal colic, wrist drop and porphyrinuria was compatible with lead poisoning. The patient was employed by a jeweller who was interviewed and stated that contact with lead was slight. Further evidence against lead poisoning were the paralysis of the brachioradialis and the sensory changes. The blood film showed no punctate basophilia and subsequent tests of the urine showed the presence of a large amount of uroporphyrin and only a slight increase in coproporphyrin; in lead poisoning the latter is increased and uroporphyrin has not been reported. The normal blood sugar and absence of sugar from the urine precluded diabetic polyneuritis.

The slight skin changes observed were not unlike the healed lesions seen in porphyria cutanea tarda which is not infrequently encountered in South African Negroes. However, a random survey of other patients without porphyrinuria showed that the majority had similar scars on hands and feet which were probably due to past trauma. While the absence of active skin lesions at a time when porphyrin was present in the urine excluded the condition at this stage, this patient may have been of the type that commences with skin lesions and later develops the acute type of porphyria.

All the features present and the history of this case are compatible with acute porphyria and despite its very rare occurrence in Negroes it is presented as such. It is perhaps noteworthy at this stage that whereas acute porphyria is
more common in females than males this and the two
other Negro patients referred to were all males.

SUMMARY
The clinical and chemical findings in a fatal case of acute
porphyria occurring in a Bantu male have been presented.
No success attended the use of any of the drugs
employed.
We wish to express our thanks to Dr. G. A. Drummond and
Dr. J. Wainwright for their valuable assistance with this case.

REFERENCES
   J. Med., 17, 66.

A FOOD BALANCE SHEET FOR THE UNION OF SOUTH AFRICA

An attempt is made to balance the needs of South Africa’s
heterogeneous population against estimated production
figures.

The Protein Position. From a protein point of view the
major imbalance is caused by two factors, namely
1. Purchasing power.

TABLE I: DIET SCALES ON WHICH CALCULATIONS ARE BASED

<table>
<thead>
<tr>
<th>Food Stuffs</th>
<th>Europeans, Coloureds and Asians</th>
<th>Urban Bantu</th>
<th>Rural Bantu</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unsifted Wheat Meal</td>
<td>6 oz.</td>
<td>4 oz.</td>
<td></td>
</tr>
<tr>
<td>Maize Meal</td>
<td>4 oz.</td>
<td>12 oz.</td>
<td>16 oz.</td>
</tr>
<tr>
<td>Meat, Fish, Eggs</td>
<td>6 oz. (as purchased)</td>
<td>4 oz.</td>
<td>4 oz. (as purchased)</td>
</tr>
<tr>
<td>Milk</td>
<td>½ pint</td>
<td>½ pint</td>
<td>½ pint</td>
</tr>
<tr>
<td>Fruit and Vegetables</td>
<td>16 oz. (as purchased)</td>
<td>8 oz. (as purchased)</td>
<td>10 oz.</td>
</tr>
<tr>
<td>Sugar, Jam</td>
<td>3 oz.</td>
<td>3 oz.</td>
<td>1 oz.</td>
</tr>
<tr>
<td>Butter, Margarine, Fat</td>
<td>1½ oz.</td>
<td>1 oz.</td>
<td>1 oz.</td>
</tr>
<tr>
<td>Pulses</td>
<td>1 oz.</td>
<td>2 oz.</td>
<td>4 oz.</td>
</tr>
</tbody>
</table>

These diet scales are merely illustrative examples of daily
requirements in terms of specific foodstuffs, but there are other
combinations of foods which would be equally suitable.

Approximately three and a half million natives in the
Reserves appear to be getting a very small amount of
animal protein, which is made up mainly of animals dying
of disease and drought and a small amount of milk which
is taken from the cows of the herds of native cattle.
Possibly a certain amount of abattoir offal is also con­
sumed by this group.
All these sources of protein do not make up an
adequate supply of protein of animal origin. This group,
namely the Bantu residing in the Reserves, must receive
special attention in connexion with the protein aspect of
his diet.
With regard to the Europeans and the better favoured

TABLE II: PER CAPITA DAILY REQUIREMENTS

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Europeans, Coloureds, Asians</th>
<th>Bantu</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories</td>
<td>2,500</td>
<td>2,900</td>
</tr>
<tr>
<td>Protein (Grams)</td>
<td>66</td>
<td>66</td>
</tr>
<tr>
<td>Fat (Grams)</td>
<td>70</td>
<td>65</td>
</tr>
<tr>
<td>Calcium (Grams)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Iron (Milligrams)</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Thiamin (Milligrams)</td>
<td>1·28</td>
<td>1·28</td>
</tr>
<tr>
<td>Vitamin C (Milligrams)</td>
<td>36</td>
<td>36</td>
</tr>
<tr>
<td>Vitamin A (International Units)</td>
<td>3,250</td>
<td>3,250</td>
</tr>
</tbody>
</table>