NEPHRITIS IN THE BANTU

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Clinical patterns of nephritis are commonly encountered in Bantu patients (Hennesey, Gelfand). Literature on the subject, however, is scanty, and much of what is written is difficult to interpret in the light of the more modern classifications of nephritis.

At the present time the classification suggested by Ellis (1942), is regarded as being the most practical, with the separation of nephritis cases into types I and II. It is important to realize when applying this classification that although the two types appear clinically and pathologically as different entities they are not entirely separate diseases (Ellis, 1949). Mixed types are encountered, and not infrequently there is a transition of type II into type I (Davson and Platt, Allen, Enticknap and Joiner).

An analysis of the records of 220 cases of nephritis in Bantu patients admitted to the Baragwanath Non-European Hospital during the 5-year period from 1 January 1948 to 31 December 1952 was compared with the records of 168 cases of nephritis in European patients admitted to the Johannesburg Hospital and the Transvaal Memorial Hospital for Children over the same period. (The Baragwanath Hospital caters for non-European patients of all ages; the Johannesburg Hospital for European patients over the age of 14 years and the Transvaal Memorial Hospital for European children under the age of 14 years. The admission rates for European and non-European patients at these hospitals are sufficiently similar to allow of comparison.)

Only the earlier stages of nephritis are considered. Inherent difficulties exist in obtaining early records of cases of the chronic forms of nephritis (the Baragwanath Non-European Hospital was only instituted in 1948). This is obviously an important aspect still to be covered. Pathological confirmation is very necessary in any study of renal disease. Little is available for the present series, for the mortality is low in the early stages of nephritis. This confirmation may only be obtained in years to come.

Classification

Using the criteria of Ellis (1942), the cases were classified into nephritis of types I and II as shown in Table I.

TABLE I. CLASSIFICATION OF CASES OF NEPHRITIS INTO TYPE I AND TYPE II

	Total	*Type-I Nephritis*	*Type-II Nephritis*
European	168	126 (75%)	42 (25%)
Bantu	220	150 (68%)	70 (32%)

The few cases encountered that were considered to be examples of a mixed form of nephritis were classified under what was regarded as the predominant type of nephritis in the particular case.

Age Incidence

In order to compare the age of onset of the two types of nephritis in the European and the Bantu patients, the frequency distribution of the cases was determined in

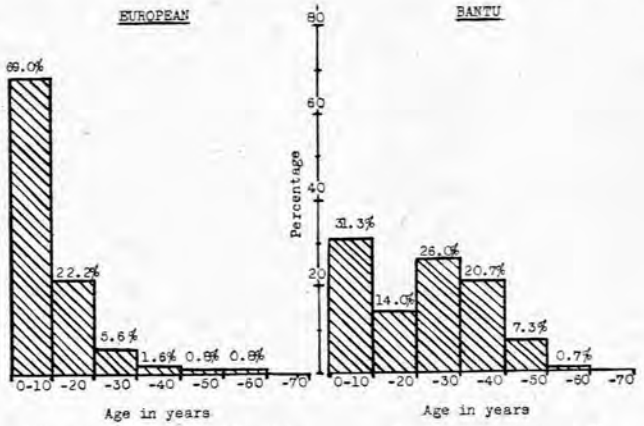
TABLE II. DISTRIBUTION OF THE TYPE-I AND TYPE-II NEPHRITIS CASES IN 10-YEAR AGE-GROUPS (AGE OF ONSET OF DISEASE)

Age (years)	*European* *Type I*	*European* *Type II*	*Bantu* *Type I*	*Bantu* *Type II*
0–10	87 (69·0%)	24 (57·1%)	47 (31·3%)	—
11–20	28 (22·2%)	5 (11·9%)	21 (14·0%)	18 (25·7%)
21–30	7 (5·6%)	4 (9·5%)	39 (26·0%)	40 (57·1%)
31–40	2 (1·6%)	4 (9·5%)	31 (20·7%)	11 (15·7%)
41–50	1 (0·8%)	3 (7·2%)	11 (7·3%)	1 (1·5%)
51–60	1 (0·8%)	1 (2·4%)	1 (0·7%)	—
61–70	—	1 (2·4%)	—	—
Total	126 (100%)	42 (100%)	150 (100%)	70 (100%)
Average age	10 years	15 years	21 years	27 years

10-year age-groups and is set out in Table II and illustrated by means of histograms in Figs. 1 to 4.

The onset of type-I nephritis in the European patients is at its maximum in the first 2 decades (91·2%). This contrasts with the Bantu, in whom the disease shows a relatively more even distribution over the age-groups up to the end of the 4th decade (Figs. 1 and 2).

The type-II patients of the two races also show a difference in the age of onset of the disease. The European patients have a maximum incidence in the 1st decade (57·1%), and a low incidence of cases occurs at all ages up to the end of the 7th decade. In the Bantu patients there were no cases with onset of the disease during the 1st decade. Almost all the cases began their illness between the ages of 11 and 40 years, with a maximum incidence during the 3rd decade (57·1%). Thus it appears that type-II nephritis is predominently



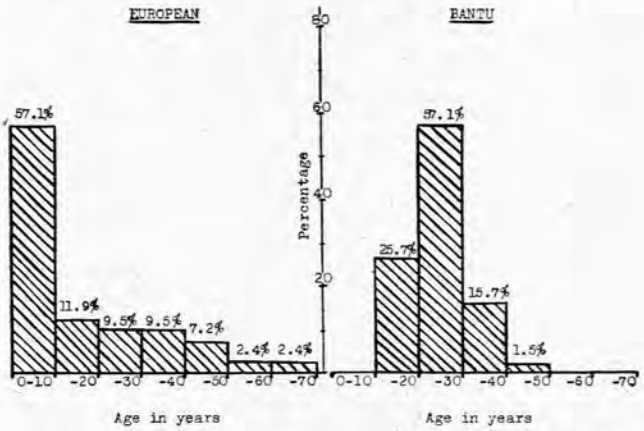
Figs. 1 *and* 2. Type I. Histograms showing percentage distribution in 10-year age-groups of 126 European and 150 Bantu cases of type-I nephritis (age of onset of disease).

a disease of infants in the European, and a disease of young adults in the Bantu. (Figs. 3 and 4).

Sex Incidence

The sex incidence of the cases is set out in Table III.

The admission rate of male patients to the Baragwanath Hospital is much greater than that of females, owing to the larger male Bantu population in the Johan-



Figs. 3 *and* 4. Type II. Histograms showing percentage distribution in 10-year age-groups of 42 European and 70 Bantu cases of type-II nephritis (age of onset of disease).

TABLE III. SEX INCIDENCE OF TYPE-I AND TYPE-II NEPHRITIS

	European *Type I*	*European* *Type II*	*Bantu* *Type I*	*Bantu* *Type II*
Males ..	80 (63%)	16 (38%)	106 (71%)	39 (56%)
Females	46 (37%)	26 (62%)	44 (29%)	31 (44%)
Total ..	126	42	150	70

nesburg area. This might account for the greater percentage of male cases of both types of nephritis in the Bantu as compared to the European.

Mortality Rates

Four of the Bantu type-I cases (2·7%) died in the acute phase, and 7 of the type-II cases (10·0%) died while in hospital with oedema.

Three of the European type-I cases (2·4%) and 10 of the type-II cases (23·8%) died in the acute phase.

The pathology is not reviewed at this stage; further analysis is still necessary.

Most of the deaths in the European type-II cases occurred in children under the age of 4 years. In these children type-II nephritis appears to be a more serious disease than that seen in the young Bantu adult.

CASE REPORTS

The following case histories are all of Bantu patients, and are included to illustrate some of the points mentioned in the discussion.

Case 1

J.M., male aged 33 years. Admitted 3 June 1953.

Complaint. Swelling of whole body for 4 days. Slight breathlessness on exertion for same period. Previously quite well. No sore throats, and no haematuria noted.

Examination. Patient lying comfortably in bed. Temperature 97° F., pulse rate 68 per minute, respiration rate 20 per minute. Generalized oedema involving face, trunk and limbs. Nasal and pharyngeal mucosa congested. Heart not enlarged, sounds normal. Blood pressure, 180/100 mm. Hg. Lungs, abdomen, and central nervous system including fundi, normal.

Urine. Clear, amber-coloured. Specific gravity 1020. Albumin present, 0·5 g. per 100 ml. Microscopic examination of the centrifuged deposit showed moderate numbers of erythrocytes together with a few polymorphonuclear leucocytes and hyaline and epithelial casts.

Blood. Blood urea 47 mg., plasma cholesterol 205 mg., haemoglobin 16 g. (all per 100 ml.). Leucocyte count 7,900 per c. mm. Serum proteins: total 5·6 g. (albumin 2·3 g., globulin 3·3 g.) per 100 ml. Streptococcal antihaemolysin titre 150 units.

Treatment. Rest in bed on low sodium diet. Procaine penicillin 600,000 units intramuscularly daily for 14 days.

Progress. After one week's stay in hospital the oedema had subsided and could no longer be detected clinically. The blood pressure dropped to 120/90 and remained at that level. The blood-urea level dropped to 23 mg. The amount of urinary albumin diminished steadily and after the 17th day in hospital was no longer detectable, and only an occasional erythrocyte could still be detected on microscopic examination of the urine.

Comment. This is an example of an attack of type-I nephritis, with improvement after a short clinical course. The absence of macroscopic haematuria is in no way unusual.

Case 2

G.M., male aged 33 years. Admitted 28 March 1953.

Complaint. Swelling of legs and abdomen of gradual onset for 2 months. He was able to continue with his work as a farm-labourer until 1 week before admission, when the swelling began to increase more rapidly, and he found it difficult to walk about; also he became increasingly breathless on exertion. Two days before admission he noted haematuria for the first time. There is no history of sore throats.

Examination. Patient lying flat in bed, a little dyspnoeic and unable to sit up because of marked abdominal distention. Moderate oedema of legs and over sacrum. Temperature 98° F., pulse rate 90 per minute, respiration rate 30 per minute. Heart not clinically enlarged, sounds normal. Blood pressure 122/80 mm. Hg. Dullness on percussion, diminished air entry, and a few fine crepitations were present at both lung bases posteriorly. X-ray appearance of heart and lungs normal. The abdomen was tensely distended with ascites. The nervous system and fundi were normal.

Urine. Frank haematuria was present on admission.

Paracentesis of the abdomen was performed on the day of admission, and 7 litres of amber-coloured fluid withdrawn. The fluid had a specific gravity of 1010 and contained 1·4 g. of protein per 100 ml. After the paracentesis a smooth soft non-tender liver edge was palpable at the level of the costal margin.

Blood. Blood urea 72 mg., blood cholesterol 186 mg., haemoglobin 11·1 g. (all per 100 ml.). Serum proteins: total 3·9 g. (albumin 0·7 g., globulin 3·2 g.) per 100 ml.

In view of the disproportionately marked ascites, investigations were carried out to determine if liver disease and portal hypertension were present as well as nephritis. Rectal examination revealed no haemorrhoids. Fluoroscopy with barium swallow failed to demonstrate oesophageal varices.

Liver Function Tests. Thymol tubidity 1·0 unit. Thymol flocculation test—negative. Cephalin flocculation test—negative. Takata-Ara reaction—++++. Alkaline phosphatase 7·2 units (King-Armstrong).

Aspiration Liver Biopsy. Showed slight haemosiderosis, and some nuclear irregularity of the parenchymal cells. No evidence of cirrhosis or amyloidosis.

Treatment. Bed rest and salt-free diet.

Progress. The ascitic fluid re-accumulated rapidly and paracentesis was necessary at weekly intervals during the first 5 weeks' stay in hospital. The oedema of the legs and over the sacrum persisted. The macroscopic haematuria subsided rapidly. By the 4th day in hospital red blood-cells were only visible on microscopic examination, together with polymorphonuclear leucocytes and numerous hyaline and granular casts. These microscopic findings and albuminuria (0·5 to 1·0 g. per 100 ml.) persisted throughout the patient's stay in hospital.

The blood-urea level increased steadily until on 4 May, 5 weeks after admission, it had reached 156 mg. and then it began to subside slowly until, on 1 June, it was 38 mg. Paracentesis of the abdomen was found necessary for the last time on 8 May; thereafter the ascitic fluid did not re-accumulate to any appreciable extent, and the oedema of the legs started to diminish.

The patient was discharged from hospital on 15 June, after almost 12 weeks' stay. By this time only minimal oedema of the ankles was present. No ascites could be detected. Blood pressure was 130/80 mm. Hg. Albuminuria (0·5 g. per 100 ml.) was still present and microscopic examination of the urine still showed the presence of erythrocytes and hyaline and granular casts. Blood-urea level was 38 mg.

Comment. This is predominantly a case of type-I nephritis, in which the 'acute' phase has run a rather prolonged course. The oedema and blood urea increased steadily during the first 5 weeks in hospital and then subsided. The persisting of the urinary findings after the oedema and uraema had subsided suggests that the disease remains latent and may progress to chronic (type I) nephritis. The possibility that this was originally type-II nephritis that was converted to type-I nephritis shortly before admission must be considered in view of the history of two months' oedema before admission. As yet we have no explanation for the disproportionately marked ascites in this case.

Case 3

A.M., male aged 36 years. Admitted 3 June 1953.

Complaint. Swelling of body for 3 days. A slight cough 2 weeks before admission, otherwise was quite well until onset of present illness. No sore throats and no haematuria noted. Patient said he was in the habit of consuming about a gallon of kaffir beer a day.

Examination. Lying comfortably in bed. Temperature 98° F., pulse rate 84 per minute, respiration rate 26 per minute. Well-marked oedema of face and limbs and slight oedema detectable in soft tissues over trunk. Parotid glands prominent. Heart was clinically normal. Blood pressure 166/100 mm. Hg. Scattered rhonchi in both lungs. In the abdomen a hard non-tender liver edge was palpable 4 cm. below the costal margin. Free fluid could not be detected clinically. Testes were atrophic.

Urine. Clear, amber-coloured. Specific gravity 1010. Albumin present (0·4 g. per 100 ml.). Microscopic examination of the centrifuged deposit showed moderate numbers of erythrocytes and a few polymorphonuclear leucocytes and hyaline casts.

Blood. Blood urea 72 mg., blood cholesterol 145 mg., haemoglobin 13·7 g. (all per 100 ml.). Serum proteins: total 6·2 g. (albumin 2·4 g., globulin 3·8 g.) per 100 ml.

Liver Function Tests. Thymol turbidity—5 units. Thymol flocculation—++++. Takata-Ara reaction—++. Colloidal-red test—++++++.

Treatment. The patient was restricted to 2 pints of fruit juice daily for the first 4 days. He was then placed on a low-sodium, protein-free diet for a further week; followed by a diet with sodium

restriction only. Intramuscular injection of procaine penicillin 600,000 units daily was given for 10 days.

Progress. The oedema subsided steadily, and was no longer detectable after 1 week in hospital. The urinary findings present on admission persisted; the specific gravity remained constant, in the region of 1010, and did not vary with water loading or water restriction. The blood urea dropped to 46 mg. by the end of the 1st week and to 37 mg. after a further week, but increased to 75 mg. by the 4th week and remained at that level throughout the patient's stay in hospital. The hypertension present on admission persisted with only slight daily variation.

After two months in hospital, the patient's condition remained static, although he was asymptomatic and feeling well, and he was discharged from hospital on 4 July and allowed to return to work.

Comment. This is a case of type-I nephritis that presents the features of chronic renal failure after only a short acute phase, and has most probably progressed rapidly to chronic (type-I) nephritis. This patient contrasts in some respects with the previous one in that although liver disease (probably alcoholic cirrhosis) is present, the oedematous phase is not prolonged and ascites is not a feature.

Case 4

J.N., male aged 28 years. Admitted 24 October 1951.

Complaint. Swelling of legs and abdomen for 2 months. No sore throats or haematuria noted.

Examination. Face puffy, gross anasarca of lower limbs and sacral region. Marked ascites. Heart clinically normal. Blood pressure 115/80 mm. Hg. Dullness and diminished air-entry at both lung-bases posteriorly. X-ray of the chest showed bilateral pleural effusion.

Urine. Heavy albuminuria (1·2 g. per 100 ml.). Microscopic examination of the centrifuged deposit showed a few hyaline and granular casts, with only an occasional erythrocyte.

Blood. Blood urea 30 mg., blood cholesterol 680 mg., haemoglobin 16·3 g. (all per 100 ml.). Serum proteins: total 4·4 g. (albumin 0·2 g., globulin 4·2 g.) per 100 ml.

Treatment. Salt-free high-protein diet.

Progress. The oedema increased steadily despite treatment. Three weeks after admission the patient had a sudden attack of breathlessness associated with a brisk haemoptysis. He suffered repeated small haemoptyses during the following 3 weeks, and died suddenly on 15 December 1951 after another acute attack of dyspnoea followed by circulatory collapse.

Post-mortem examination showed 2 recent infarcts in the left lower lobe of the lungs. The kidneys were large and pale and on microscopic examination showed the typical features of type-II nephritis.

Comment. This is a case of type-II nephritis. Throughout the stay in hospital hypertension was never observed. Blood-urea estimations were carried out repeatedly, and 40 mg. per 100 ml. was the highest recorded. Heavy albuminuria was constantly present.

DISCUSSION

The classification of Bantu cases of nephritis in this series indicates that type-I nephritis is encountered more frequently (68%) than type-II nephritis (32%).

There is a popular misconception that type-I nephritis is rare in the Bantu, and that type II is the more common form of nephritis in these people. (Davidson.)

In the adult Bantu patient with type-I nephritis macroscopic haematuria is uncommon, although microscopic haematuria is almost invariably present at some stage of the disease. The oedematous phase although acute in onset may occasionally last as long as 3-4 months even in cases that eventually make a complete recovery.

The clinical picture of one of these patients when admitted to hospital, often several weeks after the onset of the illness, is superficially similar to that of a patient with type-II nephritis. Temporary hypertension and nitrogen retention are usually noted. These signs are absent as a rule in type-II nephritis except in the terminal stage of the disease.

Low serum-albumin levels are often encountered in patients with either type of nephritis. Liver disease and malnutrition may be contributory factors. It seems unlikely, however, that liver disease and malnutrition are causes of the prolonged oedema in some of the Bantu cases of type-I nephritis (see cases 2 and 3). Very low serum-albumin levels have been noted in patients that have had only a relatively short oedematous phase, and these low levels usually remain unchanged long after the patients have lost their oedema.

On the other hand the course of type-I nephritis in most of the Bantu children of this series appeared to correspond more to the classically accepted pattern; i.e., acute onset of oedema and haematuria following upper respiratory or other infections, often associated with transient hypertension and nitrogen retention, the signs and symptoms usually subsiding fairly rapidly with rest in bed.

It is interesting that the average age of the Bantu patients with type-I nephritis is 21 years, and that the cases are distributed more evenly above and below this age as compared to the European, in whom it is predominantly a disease of young children (Figs. 1 and 2); also that in the Bantu type-II nephritis is more a disease of young adults as compared with the European, in whom it is commonest in children (Figs. 3 and 4).

SUMMARY

An analysis of the records of 220 cases of nephritis in Bantu patients is made and compared with the records of 168 cases in European patients. In the Bantu, type I is the commonest form of nephritis, and occurs with almost equal frequency in children and in adults.

Absence of macroscopic haematuria, and a prolonged oedematous phase, are not uncommon features of type-I nephritis in Bantu patients.

In the Bantu, type-II nephritis is predominantly a disease of young adults.

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