

# Polyarteritis Nodosa in the African

## REPORT OF A CASE

BY

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Makwili Lizzie (48092) was an obese African female about 44 years of age. She was admitted on 16th April, 1969, to Harare hospital, where she died seven days later. Her history on the day of admission was one of nausea and vomiting her food. She was found to be uraemic and a diagnosis of chronic pyelonephritis was made.

She stated that she was well until five days before admission, when she began to vomit twice a day, mainly after food. The vomitus consisted mainly of food, but no bile was noticed in it. She also experienced generalised joint pains which were not migratory. She commented too that she had become feverish with her present illness. She had not been out of Salisbury for many months.

On closer questioning she admitted that her appetite was very poor and that she had been feeling nauseous. Although she was inclined to be constipated, she began to pass loose watery motions which contained blood and mucus. For three days, too, before coming to the hospital she was troubled with a headache, felt mostly over the temple regions of the head. Besides these, she also mentioned a cough, chest pain and palpitations. Also perhaps of significance was that for five days before her admission to hospital she found that it was painful to pass her urine, which was dark and even contained blood. Her last menstrual cycle occurred on 9th March, 1969, but it was scanty. Three years before she had been treated for malaria, but otherwise there was nothing else of note in her previous history. She had eight children who were well.

On examination she looked ill and was febrile (temperature 101.8° F. on admission). She was very obese. There was no clinical evidence of anaemia, jaundice, oedema of the face or extremities or clubbing of fingers. The pulse was 100 per minute and regular. The blood pressure was 160/120 and the fundi normal. The neck veins were not distended, the lungs were clear

and heart apparently normal on physical examination. She evinced some tenderness at the root of her neck, which however was not rigid. Because of her obesity it was not easy to examine her abdomen, but we could not detect any enlargement of the liver or spleen. The cranial nerves were normal and there was nothing to suggest any involvement of the peripheral nerves. The joints were not involved. Some tenderness was noted in the left lateral fornix on vaginal examination. Rectal examination was normal. We made a provisional diagnosis of pyrexia of unknown origin and of hypertension.

The urine contained 1+ albumen and, on microscopy, both hyaline and granular casts and red cells ++ were found. Urine culture: profuse growth of *Esch. coli*. Creatinine clearance 4.8 ml./min. (total volume 300 ml.). Serum: sodium 114 mg./Eq./l., potassium 4.5 m./Eq./l., chlorides 87 m./Eq./l., bicarbonate 19.1 m./Eq./litre. Blood VDRL negative. Stool negative for ova.

Liver function: total bilirubin 0.6 mg./100, alk. phosphate 6.8 KA units/100 ml. Total serum

proteins 6.9 g. per cent., albumen 2.1 g. per cent., globulin 4.8 g. per cent., A/G ratio 0.4:1. Blood pH 7.12. Serum cholesterol 143 mg./100. Cerebrospinal fluid: leucocytes under 1 cell per cubic mm., protein 35 mg. per cent., glucose 78 mg. per cent.

#### COURSE OF ILLNESS

The first blood urea estimation was 156 mg. per cent. Three days later it had risen to 200 mg. per cent. and a week later to 275 mg. per cent.

On 13th April she was vomiting frequently and two days later was looking acidotic. She was deteriorating fairly steadily. Her blood urea crept up steadily and on 19th April it was 275 mg.

On 16th April she passed 300 ml. of urine, none on the next day, 600 ml. on the 18th, 1,015 ml. and 120 ml. on the 19th. It was decided to dialyse the patient in view of her oliguric state and rising blood urea. After the dialysis she passed 450 ml. of urine overnight and indeed reported that she was feeling fine. The blood urea on this day was 190 mg. per cent., potassium 3.4 m./Eq./l., and  $\text{HCO}_3$  22.5 m./Eq./l. On 23rd

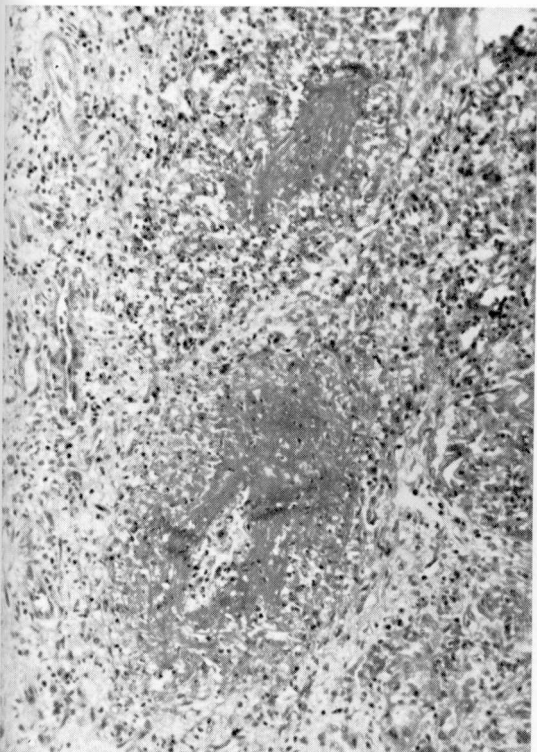


Fig. 1—Kidney x 125. This shows fibrinoid necrosis of the walls of two small arteries. The inflammatory cells round the arteries are histiocytes, plasma cells, lymphocytes and neutrophils.

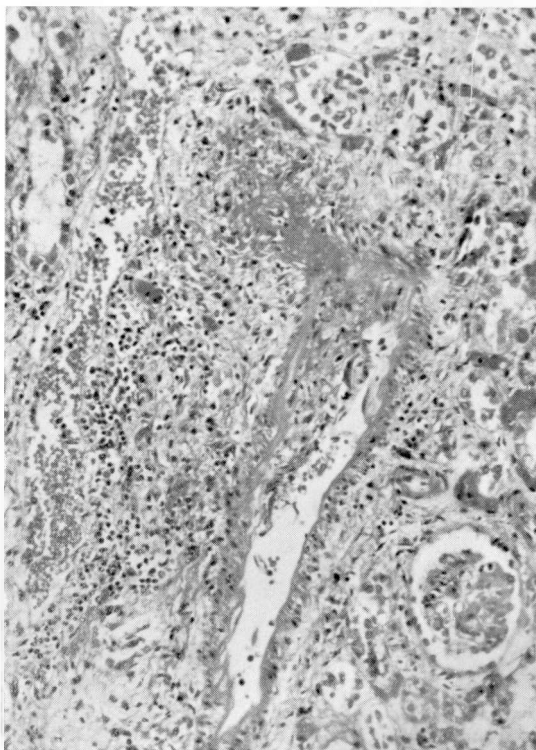


Fig. 2—Kidney x 125. This shows a small artery with an arteriole branching from it. The whole of the arteriole and a segment of the wall of the larger vessel at the junction exhibit fibrinoid necrosis.

April a second haemodialysis was carried out and she voided 600 ml. of urine the following night. However, on this occasion her chest became full of secretions and she became cyanosed, suddenly developed fits and breathed her last.

#### AUTOPSY

##### *External Examination*

The body was that of a very obese female African adult who looked rather older than the stated age of 44 years. No external abnormalities noted.

##### *Internal Examination*

*Serous Cavities.*—Both pleural cavities contained approximately 500 ml. of a clear, pale yellow fluid. Adhesions bound the lower surface of the left lung to the diaphragm. The pericardial cavity contained a few mls. of clear, pale yellow fluid. The peritoneal cavity was healthy.

*Respiratory System.*—There was a moderate congestion of the mucosa of the lower trachea and larger bronchi. Both lungs were uniformly congested throughout. The lungs were not oedematous and there was no evidence of consolidation.

*Cardiovascular System.*—The heart weighed 279 g. The myocardium was pale and flabby. No significant abnormalities were found in the coronary arteries. The aorta showed a minimal degree of atherosclerosis. The great vessels of the neck and cerebral vessels were healthy.

*Digestive System.*—The mucosa of the small bowel was slightly congested. No other significant abnormalities were found in the gastro-intestinal tract. The liver weighed 1,520 g. It was pale and soft. No significant abnormalities were found in the gall bladder, extra-hepatic ducts or pancreas.

*Reticulo-Endothelial System.*—The spleen weighed 310 g. and its pulp was very soft. Lymph nodes throughout the body showed no significant abnormality.

*Endocrine System.*—No significant abnormalities were found in the pituitary, thyroid or adrenal glands.

*Genito-Urinary System.*—The right kidney weighed 198 g. and the left 175 g. Their capsules were slightly adherent and a few shallow depressions were seen on the surfaces of both kidneys. In both, the cortico-medullary patterns were normal, but the cortices had a mottled, pale yellow

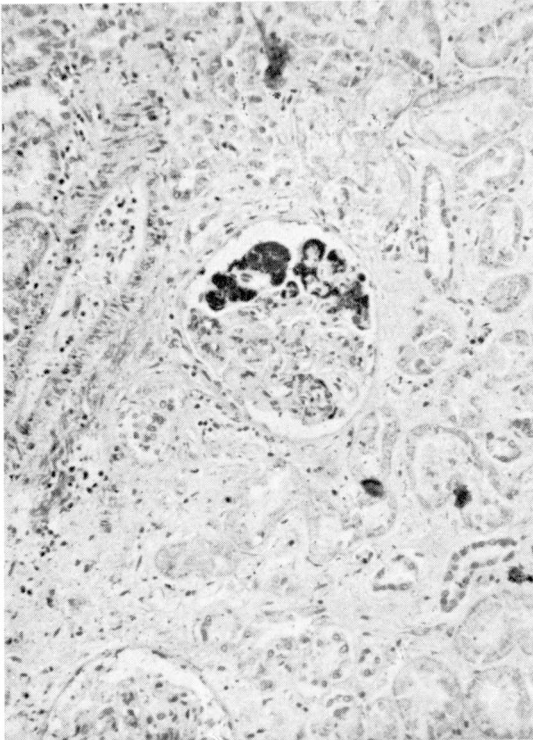


Fig. 3—Kidney x 125. Areas of fibrinoid necrosis are seen in a glomerulus.

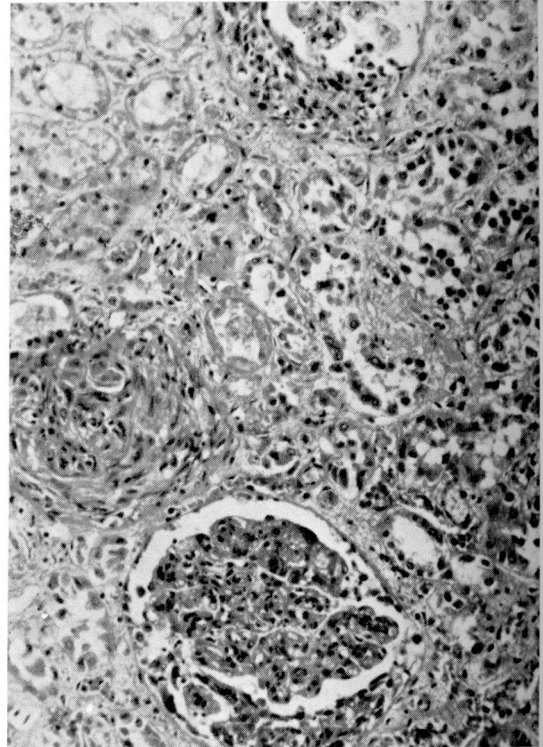


Fig. 4—Kidney x 125. The glomeruli showing conspicuous fibroepithelial crescents.

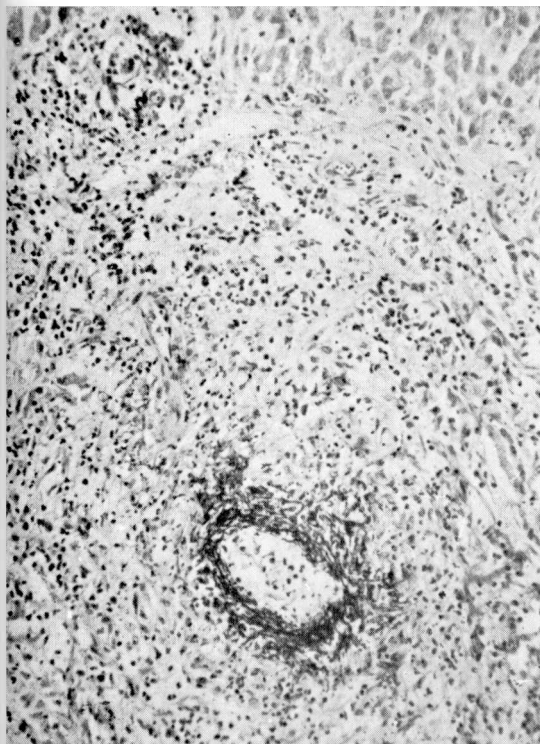


Fig. 5—Liver x 125. This shows fibrinoid necrosis in a small artery in a portal area near the porta hepatis.

and red appearance. the pyramids were intensely congested. The right ureter was healthy. The left was very slightly dilated. The mucosa of the bladder neck was somewhat congested. The uterus was small and there were a number of small retention cysts in the cervix. Both ovaries were atrophic.

*Central Nervous System.*—The brain weighed 1,265 g. Both brain and meninges were moderately congested, but appeared otherwise normal.

#### *Histology*

*Kidneys.*—Many of the small arteries and arterioles show a necrotising vasculitis. In most cases the full thickness and entire circumference of the vessel wall is replaced by fibrinoid material. The surrounding tissues are infiltrated by histiocytes, plasma cells, lymphocytes and neutrophils (Figs. 1 and 2).

Proliferative and fibrinoid degenerative changes are conspicuous in many glomeruli (Fig. 3), and in some fibroepithelial crescents are prominent (Fig. 4). There are small foci of tubular necrosis and a few collecting tubules contain eosinophilic casts.

*Liver.*—In the portal areas near the porta hepatis many of the small arteries show a necrotising vasculitis (Fig. 5). A few of the arterioles in the smaller portal radicles are also involved. The hepatic clymen shows no significant change.

*Heart.*—A very few of the arterioles on the surface of the heart show fibrinoid degeneration of their walls.

*Pancreas and Spleen.*—A number of the small arteries in both organs exhibit a necrotising vasculitis.

*Uterus.*—No abnormalities were seen in the arteries of the sections examined.

Pathologist's diagnosis: (1) Renal failure; (2) polyarteritis nodosa.

#### COMMENT

The term immune disorders of connective tissue includes such conditions as rheumatoid arthritis, rheumatic fever, systemic lupus erythematosus, systemic sclerosis, dermatomyositis, thrombotic thrombocytopenic microangiopathy and various types of necrotising vasculitis, including polyarteritis nodosa (Gardner, 1965). Of these conditions the only one commonly found in Africans is rheumatic fever. Some others, though not common, have been reported on several occasions, e.g., polymyositis (Horsfall, 1965; Gelfand and Taube, 1966; Baker, 1969). Rheumatoid arthritis is uncommon in Africans (Edington and Gilles, 1959; Gelfand, 1969). Trowell (1960), reviewing the literature up till the end of 1959, could find no report of systemic lupus erythematosus in an African. However, Shaper (1961) reported five cases of S.L.E. in Ugandan Africans and suggested that the condition is not so rare in Africans as was believed. Shaper's concept is strengthened by the fact that in New York S.L.E. is more common in Negroes than in whites (Siegel *et al.*, 1962). Systemic sclerosis also appears to be very uncommon in Africans, though one case was reported by Gelfand in 1953. There have been no reports of thrombotic thrombocytopenic microangiopathy, also very few proved cases of polyarteritis nodosa in Africans, have been reported (Trowell, 1960). It has been suggested by Lloyd and his colleagues (1967) that symmetrical gangrene was caused by polyarteritis nodosa. Symmetrical gangrene has been described on several occasions in Africans (Gelfand, 1947; Turpie *et al.*, 1967; Lowenthal, 1967). It is possible that polyarteritis is not so rare in Africans, but that owing to the protean presenting symptoms the diagnosis has been overlooked. It is also of interest that in New York Siegel *et al.* (1962) could find no difference in the incidence of polyarteritis between whites and Negroes.

### SUMMARY

This paper reports a case of polyarteritis nodosa in an African woman of 44 years. The absence of previous reports of this condition in Africans might suggest that it is very rare in these people. It is felt, however, that with better diagnostic aids available nowadays that more cases will come to light.

### REFERENCES

- BAKER, N. M. (1969). *C. Afr. J. Med.*, **15**, 68.
- EDINGTON, G. M. & GILLES, H. M. (1969). *Pathology in the Tropics*. Arnold, London.
- GARDNER, D. L. (1965). *Pathology of the Connective Tissue Diseases*. Arnold, London.
- GELFAND, M. (1947). *Brit. med. J.*, **1**, 847.
- GELFAND, M. (1953). *J. trop. Med. Hyg.*, **56**, 199.
- GELFAND, M. (1969). *C. Afr. J. Med.*, **15**.
- GELFAND, M. & TAUBE, E. (1966). *J. trop. Med. Hyg.*, **69**, 232.
- HORSFALL, P. A. C. (1965). *S. Afr. med. J.*, **39**, 695.
- LLOYD, A., KURBALL, M. & FRASER, G. (1967). *Brit. med. J.*, **1**, 468.
- LOWENTHAL, M. N. (1967). *Brit. med. J.*, **2**, 700.
- SHAPER, A. G. (1961). *East. Afr. med. J.*, **38**, 135.
- SIEGEL, M., LEE, S. L., WIDELock, D., REILLY, EDITH R., WISE, G. J., ZINGALE, S. B. & FUERST, H. T. (1962). *J. chron. Dis.*, **15**, 131.
- TROWELL, H. C. (1960). *Non-Infective Diseases in Africa*. Arnold, London.
- TURPIE, A. G. G., FORBES, C. D. & McNICOL, G. P. (1967). *Brit. med. J.*, **3**, 646.
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