THE BURKITT LYMPHOMA SYNDROME IN JOHANNESBURG

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When Burkitt described Central African lymphoma in 1958, it appeared to be a distinct clinical syndrome.[1] Presenting particularly in the jaws, retroperitoneum and abdominal viscera, this tumour was described as having a striking incidence in children living in restricted climatic regions across Central Africa, in these areas accounting for nearly 50% of all childhood malignancies. All races are affected, the maximum incidence being at about 6 years of age.

Since the description of a characteristic histological pattern,[2-4] its wider range of presentation and incidence has been recognized. The most common malignancy in female children of Nigeria is ovarian lymphoma[5,6] with histopathological features identical to that described by Davies.[2] Sporadic cases of a similar syndrome have been reported from the USA[7] and the disease also occurs in New Guinea.[8] An increasing number are being reported from parts of Southern Africa where the climate departs considerably from Burkitt's original criteria.[9-11]

Lymphoma in childhood has until recently been an uncommon disease at Baragwanath Hospital and the occurrence of 5 cases within 10 months is remarkable. Four of the patients have come from the Johannesburg area and 1 from the Eastern Transvaal.

CASE HISTORIES

Case 1

J.L., a 7-year-old Bantu male, lived near Johannesburg from birth. He was first seen in the outpatient department on 14 October 1963, complaining of a swelling (2 in. × 2 in. in size) in the left epitrochlear region noticed for '3 days'. This mass was excised in the outpatient theatre on 5 November 1963. Microscopically it showed an undifferentiated round cell neoplasm, the features of which were considered to be consistent with neuroblastoma or possibly lymphosarcoma.

The patient did not keep his appointment for the week following biopsy and was only seen again on 6 March 1964 when he presented with a painful swelling of the left upper tibia, a recurrent left epitrochlear swelling and large glands in the left groin and axilla. A large mass in the left loin was also found.

A full blood count showed a haemoglobin of 13 G/100 ml., white cell count of 8,400/cu.mm. (normal differential count) and an erythrocyte sedimentation rate of 37 mm. in 1 hour (Wintrobe).

Radiographic studies on admission demonstrated a patchy osteolytic and sclerotic lesion of the proximal metaphysis and diaphysis of the left tibia, cortical destruction and a laminated periosteal reaction with soft-tissue swelling (Fig. 1). The adjoining tibial epiphyseal centre showed trabecular absorption. A large patchy lytic lesion with cortical destruction was present in the lateral supracondylar region of the right femur.

Intravenous pyelography demonstrated enlargement of the left kidney with distortion of its pelvi-calyceal system (Fig. 2). The right kidney was displaced anteriorly but there was no definite evidence of intrinsic renal involvement.

Laparotomy. On 17 April 1964, the patient was submitted to laparotomy. The adrenal gland was normal. The left kidney was seen to be enlarged to about twice normal size and almost entirely replaced by tumorous deposits. A nephrectomy was performed and the cut surface showed diffuse infiltration by whitish tumour tissue with obliteration of the normal markings. The capsule stripped easily, leaving a slightly nodular surface in which there were scattered petechial haemorrhages. Microscopic examination showed a diffuse infiltration of small cells with dark nuclei and showing fairly frequent mitoses. Tubules and glomeruli could still be recognized but were widely separated (Fig. 3). No phagocytic histiocytes were observed in any of the sections.

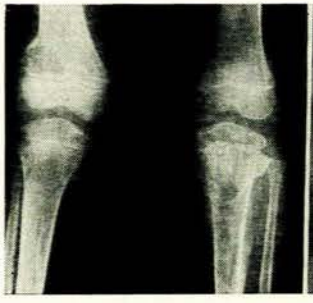

Fig. 1

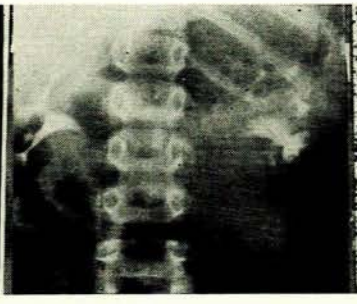

Fig. 2

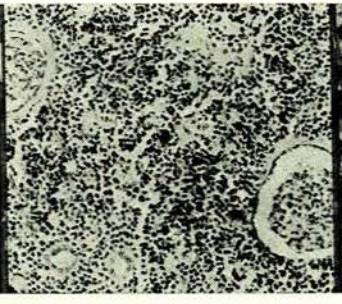

Fig. 3

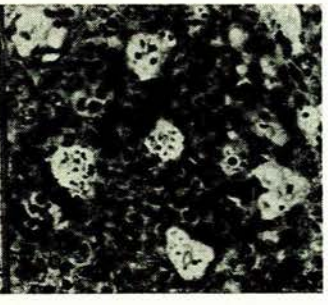

Fig. 4

Fig. 1. Sclerosis and bone destruction of left tibia with periosteal reaction and soft tissue swelling. Lytic lesion of right femur (case 1). *Fig. 2.* IVP showing left renal involvement. Arrows demarcate inferior border (case 1). *Fig. 3.* Section of kidney showing a diffuse infiltration of lymphoid cells (case 1) (H & E x 200). *Fig. 4.* Section of lymph node showing large phagocytic histiocytes scattered among the closely packed lymphoid cells. Several mitoses are evident (case 1) (H & E x 480).

The patient was then presented at a surgical meeting, during which Professor B. J. P. Becker suggested the diagnosis of Burkitt's lymphoma on the basis of rapid clinical progression and multiplicity of lesions and recommended a further biopsy. An inguinal node was excised on 28 April 1964, and this time, findings typical of Burkitt's lymphoma were noted (Fig. 4).

Histological appearance. There were sheets of lymphoid cells with large hyperchromatic round nuclei surrounded by very little cytoplasm. Mitoses were fairly frequent. Scattered throughout the sections between the lymphoid cells there were large pale or clear histiocytic cells. These had large vesicular nuclei and, often, prominent nucleoli. Their cytoplasm was abundant, often granular and vacuolated and frequently contained phagocytosed debris. In addition there were also scattered small foci of karyorrhexis with granular nuclear debris. The tumour produced very little reticulin, the fibres being mainly around small blood-vessels.

Course. During May 1964, the patient was transferred to the Johannesburg Hospital for radiotherapy. Oral Methotrexate was also given. Response to this treatment was excellent; all palpable nodes disappeared and the tibial deposit regressed to a non-painful thickening. Improvement was however temporary, and in mid-June he was readmitted with a palpable right epitrochlear node and pathological fracture of the right lower femur. The left testis was enlarged to one and a half times the size of the right testis. There was no significant change in the blood picture. He was placed on maintenance Methotrexate therapy but his condition gradually deteriorated and he died in October 1964.

Case 2

D.L., a 6-year-old Bantu male, born 20 miles outside Johannesburg, had not changed residence except for a short journey to Piet Retief in 1961. He was admitted on 31 May 1964 complaining of abdominal swelling of 3 weeks' duration and swelling of the lower limbs of 5 days' duration. On examination he was found to be pyrexial (101°F) with gross oedema of the lower extremities, scrotum and lower abdomen. A firm, moderately tender, mass was felt arising out of the pelvis and extending up to the umbilicus. On rectal examination the tumour mass occupied most of the pelvis, infiltrating and compressing the rectum bilaterally. The testes felt normal.

Urinalysis showed the presence of albumin, numerous leucocytes and 4 - 5 erythrocytes per high-power field. The haemoglobin was 10·1 G/100 ml., white cell count 10,200/cu.mm., with a normal differential count, and the erythrocyte sedimentation rate was 44 mm. in 1 hour (Wintrobe).

Intravenous pyelography demonstrated delayed excretion by the left kidney and calyceal clubbing (Fig. 5). The ureters were displaced anteriorly by a soft tissue mass, and pelvic views showed the bladder to be indented on its left side. A full skeletal survey was normal.

Laparotomy. A diagnosis of retroperitoneal sarcoma or neuroblastoma was thought likely and an exploratory laparotomy was performed on 3 June 1964. An irregular firm mass was found arising out of the pelvis, infiltrating rectum, ileo-caecal region and loops of terminal ileum. Its pale, smooth somewhat nodular surface appeared typical of lymphosarcoma. On separating loops of bowel, an abscess cavity within the mass was entered. This abscess was related to a spontaneous ileal perforation. The involved bowel was then resected and an end-to-end anastomosis performed.

Pathological examination of the specimen showed it to consist of two portions of small intestine, 17 and 14 cm. in length, with mesentery in between. The mesentery was about 1½ cm. thick and diffusely infiltrated by homogeneous yellowish-white tumour tissue (Fig. 6).

Microscopic examination showed the typical histological picture of a 'starry-sky' appearance produced by the pale phagocytic histiocytes scattered among the densely-packed small dark malignant lymphoid cells (Fig. 7). The tumour invaded the bowel wall and extended into the mucosa in some areas. Frozen sections stained for fat showed the histiocytes to be packed with small droplets of fat.

The patient was transferred for deep X-ray therapy on 9 June 1964. Considerable improvement in his general condition followed, the mass reduced in size by about a third, and the oedema disappeared. Again, this improvement was temporary with a return of the oedema after 5 weeks and deterioration of his general condition. Methotrexate therapy was instituted with some improvement. The parents, despite his deteriorating condition, insisted upon removing the child from hospital. No follow-up examinations were possible.

Case 3

D.N., a 7-year-old Bantu female, who had resided at Piet Retief in the Eastern Transvaal since birth, was admitted on 17 June 1964, complaining of a tender swelling of the left leg for the previous 5 weeks.

On examination a tender swelling of the left upper tibia was found, considerable lymphadenopathy in both groins and small rubbery nodes in both axillae. In addition, there was a hard fixed irregular mass above the right inguinal ligament which, on rectal palpation, was felt to extend onto the side wall of the pelvis. It was also noted that both upper and lower incisor teeth were loose, though there was no swelling of the jaws.

A full blood count showed haemoglobin 9·2 G/100 ml., white cell count 8,900/cu.mm. (44% neutrophils and 56% lymphocytes) and the erythrocyte sedimentation rate 52 mm. in 1 hour (Wintrobe).

Radiological examination of the left knee showed patchy sclerosis and bone destruction in the metaphysis of the tibia, laminated periosteal reaction, soft tissue swelling and dislocation of the fibula at the proximal tibio-fibular joint (Fig. 8). The proximal epiphysis of the right tibia was sclerotic (Fig. 9). There was also evidence of sclerosis and destruction in both iliac bones (Figs. 10, 12). The head of the right humerus showed an irregular outline with some sclerosis and bone destruction involving the adjacent metaphysis (Fig. 11).

Views of the mandible showed resorption of the lamina dura of a lower molar tooth with an adjacent small cystic lesion. The lower incisors were displaced upwards and small cystic lesions were present to the right of the symphysis menti.

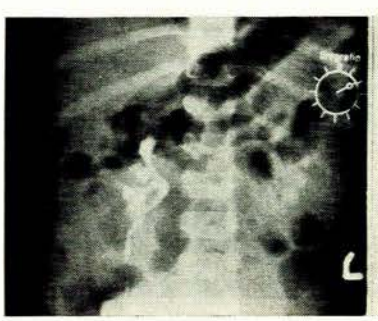

Fig. 5

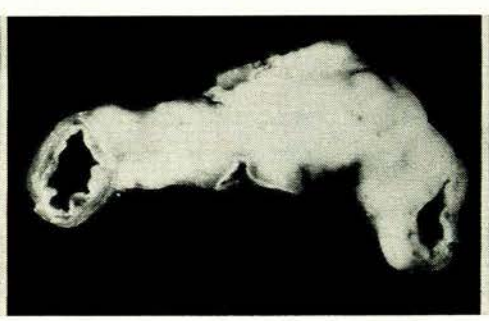

Fig. 6

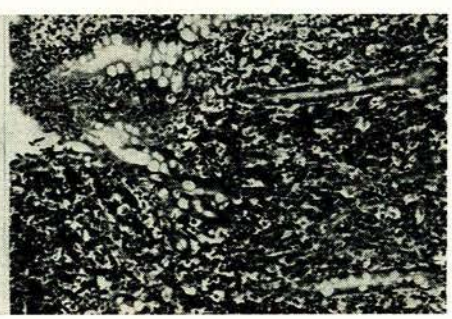

Fig. 7

Fig. 5. IVP demonstrating calyceal clubbing and delayed excretion by the left kidney (case 2). *Fig. 6.* Cross-section of specimen. Note infiltration of the bowel wall and markedly thickened mesentery (case 2). *Fig. 7.* Section of portion of small bowel shown in Fig. 6. There is tumorous infiltration extending into the mucosa—upper left (case 2).

In the chest radiograph a rounded opacity approximately 4 cm. in diameter was observed on the right side posteriorly (Fig. 11). Intravenous pyelography showed no abnormality of the pelvi-calyceal pattern but the bladder showed a large extrinsic pressure defect on its right border (Fig. 12).

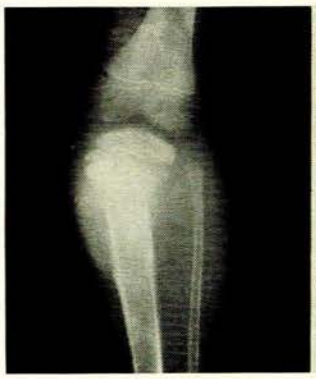

Fig. 8

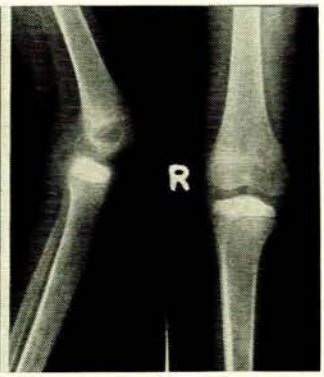

Fig. 9

Fig. 8. Radiograph of left knee showing a patchy sclerotic lesion of the upper tibia, soft tissue swelling and periosteal reaction (case 3). *Fig. 9.* Sclerotic proximal epiphyseal centre of right tibia (case 3).

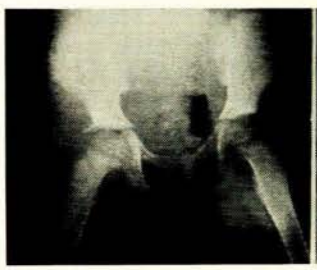

Fig. 10

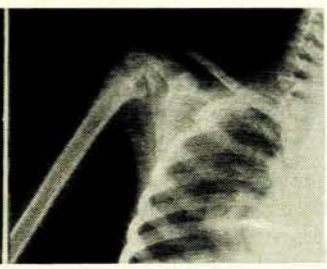

Fig. 11

Fig. 10. Markedly sclerotic lesions of both iliac bones with bone destruction (case 3). *Fig. 11.* Chest radiograph showing rounded opacity. Note also the patchy lesion of the upper end of the right humerus (case 3).

A biopsy of the left tibia and a left inguinal lymph node was performed on 23 June 1964 and again showed similar histopathological features to that of Burkitt's lymphoma, the tumour tissue being densely packed between the bony trabeculae (Fig. 13). Impression smears from the fresh specimen resembled the sections in that there were phagocytic histiocytes scattered among sheets of large lymphoid cells (Fig. 14).

On 28 July she was referred for radiotherapy. After an initial improvement the child deteriorated and died on 21 October 1964.

At autopsy no naked-eye evidence of involvement of the jaw bones was seen. The abdomen was distended. In the left groin there was a tumour 6 cm. in diameter with ulceration of the overlying skin. A smaller swelling was seen in the right groin.

The mass seen in the chest radiographs was found to be a tumour deposit 4 cm. in diameter in the right paravertebral region adjacent to the 5th and 6th dorsal vertebrae. This appeared to extend into the vertebral bodies and formed a smaller mass in the left paravertebral region. It did not extend into the spinal canal. The lungs were normal. On section the tumour tissue was homogeneously white with areas of haemorrhage and necrosis. The entire pelvis was filled with similar tumour tissue and its origin could not be determined. The para-aortic and mesenteric lymph nodes were enlarged to 3 cm. in diameter and adjacent to and infiltrating the sigmoid colon there was a necrotic tumour 5 cm. in size. Both kidneys were enlarged to about 4 times the normal and showed coalescing nodules of tumour with areas of haemorrhage (Fig. 15).

Histological examination of the various tumour deposits, kidneys, mesenteric and para-aortic lymph nodes showed diffuse infiltration by lymphosarcomatous tissue. In some areas scattered histiocytes were present while in others there were small clear spaces. The liver showed periportal and subcapsular infiltration by lymphoid cells and the red pulp of the spleen was similarly infiltrated. The Malpighian corpuscles were small and widely separated. Sections of the head of the right humerus, thoracic vertebral body, right tibia and iliac bones showed invasion by tumour. In some areas there was destruction of bony trabeculae while in others the trabeculae were thick and sclerotic. There were several foci of new bone formation between existing trabeculae. This would account for the sclerotic and patchy lesions seen radiologically.

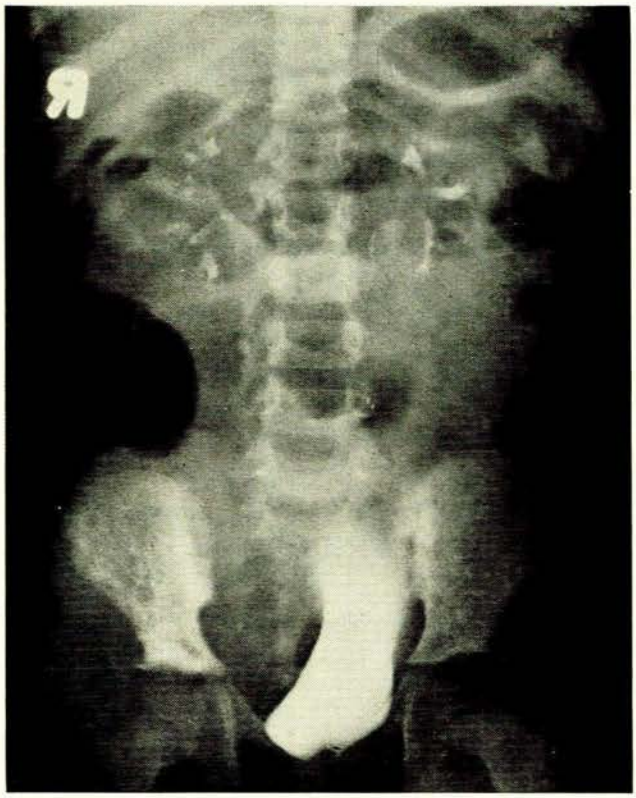

Fig. 12. Intravenous pyelogram. Normal pelvi-calyceal pattern. Extrinsic pressure effect on the bladder (case 3).

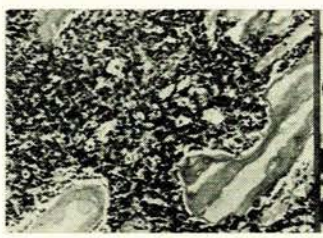

Fig. 13

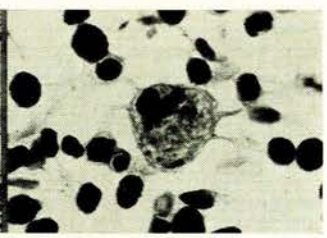

Fig. 14

Fig. 13. Section of biopsy of the left tibia showing complete replacement of the marrow by the tumour tissue (case 3) (H & E x 200). *Fig. 14.* Impression smear of tumour. Large histiocyte in centre containing phagocytosed debris surrounded by pleomorphic lymphoid cells (case 3) (H & E x 1200).

Case 4

T.I., a 6-year-old Coloured male from Kliptown, a few miles outside Johannesburg, was admitted on 18 August 1964 with

a history suggestive of subacute intestinal obstruction of 3 days' duration. A large mass approximately 22 cm. across was palpated in the right lower abdominal quadrant. The swelling was slightly tender with some mobility and was not palpable

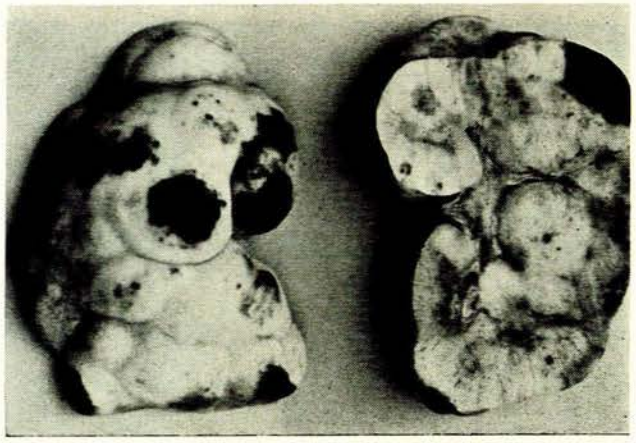

Fig. 15. Necropsy specimen of kidneys, showing diffuse and nodular tumour deposits, some with haemorrhage (case 3).

on rectal examination. There was no enlargement of the liver, spleen or peripheral lymph nodes. A blood count showed a haemoglobin of 8·0 G/100 ml., total white cell count 9,700/cu.mm., neutrophils 56%, monocytes 9% and lymphocytes 35%.

Skeletal survey, intravenous pyelography and barium enema were normal.

At *laparotomy* on 1 September, a firm, lobulated mass encircled a loop of distal ileum and did not appear to arise from retroperitoneal structures. Separation from an adherent loop of transverse colon was easily effected, 30 cm. of involved ileum was excised together with the mass, and an end-to-end anastomosis performed. One enlarged lymph node was noted and removed.

The irregular tumour mass measured $17 \times 12 \times 8$ cm. with flattened loops of small bowel stretched over it and intimately associated with the tumour. On section, almost the entire tumour was occupied by a large necrotic cavity into which the bowel opened. The wall of the cavity consisted of moderately firm homogeneous yellowish-white tumour tissue. Microscopically the typical features as seen in the previous cases were present. Several small lymph nodes dissected from the main tumour mass surprisingly showed only sinus histiocytosis with a normal architectural pattern. In one larger lymph node however there were 2 round tumour deposits resembling metastases, the remainder of the node being normal. This feature has not been noted in the previously reported cases and is not usually seen in malignant lymphomata generally.

The postoperative course was uneventful and the patient was transferred to the Johannesburg Hospital for a course of radiotherapy on 5 October.

Case 5

S.T., a Bantu male aged 3 years and a resident of Rietfontein, a few miles from Johannesburg, was admitted on 27 July 1964 with a history of progressive abdominal swelling for 2 weeks.

An enormous lobulated mass was found to occupy the right middle and upper quadrants of the abdomen. It was firm, fixed and non-tender and extended well across the midline. He also had right axillary and inguinal lymphadenopathy but the liver and spleen were not palpable.

An intravenous pyelogram and X-ray examination of the chest were normal. The haemoglobin was 11·4 G/100 ml., total white cell count 6,700/cu.mm., with 37% neutrophils, 6% monocytes and 57% lymphocytes.

This was clearly an intra-abdominal malignancy and preoperative radiotherapy was indicated in view of the size of the tumour. A half course was administered resulting in reduction of the mass to half its original size within 1 month. At laparotomy on 22 September the mass was found to be retroperitoneal, involving the root of the mesentery and origin of the superior mesenteric artery. Massive lobulated extensions infiltrated the transverse mesocolon, colon, terminal ileum and its mesentery. The tumour involving transverse colon was densely adherent to the under-surface of the liver, but otherwise the liver, spleen and kidneys were not macroscopically involved. Impression smears and sections of tumour biopsy showed the typical features of a Burkitt's lymphoma.

On 5 October the child was transferred to the Johannesburg Hospital for completion of his course of radiotherapy.

DISCUSSION

These cases illustrate the importance of clinical awareness of a condition which appears to be increasing in frequency in South Africa. The question may well be asked whether this is a new entity or whether we have been missing cases in the past. Formerly a rarity in our paediatric surgical ward, malignant lymphoma during 1964 constituted by far the commonest malignant tumour in children. Survey of our material at Baragwanath Hospital during the past 10 years has shown 10 cases of malignant lymphoma in childhood—an average incidence of 1 per year. Only 2 of these could be placed in the category of Burkitt's lymphoma.

The first patient was a 5-year-old Bantu female from Orlando (near Johannesburg) who was admitted on 25 January 1959 with severe abdominal pain. Her blood count was normal and at laparotomy enlarged mesenteric lymph nodes were found. She died on 6 February 1959 and at autopsy there was, in addition to the enlarged mesenteric lymph nodes, an irregular whitish tumour of 10 cm. in the mesentery. There were nodular tumour deposits in both kidneys and bilateral ovarian tumours. The cervical, axillary and mediastinal lymph nodes were normal and the spleen was only slightly enlarged. Histological sections of the various tumour deposits showed features resembling Burkitt's lymphoma.

The second case seen by Mr. Dinner was a 2-year-old Bantu female who had a swelling of the left mandible. She had been taken to a general practitioner who evidently thought it was a dental abscess and removed a tooth. On examination all the teeth on the left side of the mandible were out of alignment and radiologically there were several cystic areas with thinning of the cortex. The biopsy seen by Dr. Isaacson was reported as malignant lymphoma similar to those seen in Central Africa. The child was sent for radiotherapy and has not been seen since.

The occurrence of 5 cases within a single year is thus of great significance. During this period only about 4 cases of lymphoblastic leukaemia have been seen in children.[17] The incidence found in a previous survey at Baragwanath Hospital was 3 cases a year.[18]

Re-examination of the material in the 1953 - 1955 cancer survey in the Transvaal confirmed that these cases had not been seen.[12,13] Pathologists have been on the lookout for Burkitt's tumour since its description, thus Chapman and Jenkins[10] from Natal reported 5 clinically typical cases seen within one year, and review of Cape Town material showed 6 cases (2 White and 4 Cape Coloured) seen in the last 12 years.[11] From the Orange Free State

Lurie and King[14] reported a case with typical jaw tumours in a 30-year-old immigrant Bantu male from Portuguese East Africa, and a further case in a 2½-year-old Bantu male was diagnosed by Brüning[15] in Bloemfontein. This child was born in Johannesburg but had resided in Maseru since the age of 2 months. In the Transvaal Gluckman[9] reported 3 White children suffering from this disease. Prof. B. J. P. Becker reviewed the material of the Witwatersrand Medical School and found no definite case.[16]

In considering Burkitt's lymphoma a variant of lymphoblastic leukaemia, Dalldorf[19] has suggested that Southern Africa may well occupy an intermediate position between Central Africa and North America in respect of the relative incidence of the 2 diseases. In his opinion Burkitt's lymphoma may be a different expression of childhood lymphoma which in North America usually manifests itself as acute lymphoblastic leukaemia. The implication of this is that the aetiological agent or agents concerned in these conditions could be similar and the different clinical presentation is probably due to different host response.[20]

Though 5 cases is rather a small number from which to draw conclusions, it may be reasonable to suggest the possibility that our pattern of clinical presentation may also be intermediate. One of our patients had (radiological) evidence of jaw involvement and 4 had general and/or extensive retroperitoneal and renal deposits. Two of the 5 cases had long bone involvement while 3 cases had peripheral lymphadenopathy.

The uniform histological picture of sheets of immature and atypical lymphoid cells, scattered among which there are large, pale, foamy phagocytic histiocytes, gives the tumour a characteristic appearance rarely seen in other types of malignant lymphoma. Although the majority of tumours show this appearance some may resemble lymphosarcoma initially. Subsequent biopsies in these frequently show the typical features as in case 1 and this had also been seen in Kenya.[19] The lymphoid cells are larger than normal lymphocytes. They have large hyperchromatic nuclei and often show peripheral clumping of the chromatin against a distinct nuclear membrane. Nucleoli may be prominent. Surrounding the nucleus there is an inconspicuous thin rim of cytoplasm. The histiocytes are non-neoplastic components and might be a manifestation of the rapid growth of the tumour with much cell death.[3] The 'starry-sky' appearance which is so often seen in Burkitt's tumour may thus only be a secondary feature.

These tumours fall into the category of poorly-differentiated lymphocytic lymphomas. Histochemical examination of the lymphoid cells has given results similar to those of lymphocytes in reactive nodes.[4] Impression smears often show cytoplasmic vacuolation of the lymphoid cells. This is due to the presence of lipid and is also seen in acute lymphoblastic leukaemia. The lymphoid cells of both these conditions also resemble each other in that in neither disease do they contain glycogen demonstrable by the PAS technique.

Electron-microscopical studies have shown changes similar to those seen in other malignant lymphomas.[21] No virus particles have been observed and the nuclear changes have been considered to be most characteristic, consisting of marginated chromatin masses and spherical granular bodies. Great interest, however, has centered around the reports of isolation of viruses from these tumours. Herpes virus was the first to be isolated from the lymphoma[22] and a similar virus was isolated by Epstein, Achong and Barr.[23] Dalldorf and Bergamini[24] recovered 6 strains of virus from lymphomas in Kenya. Bell *et al.*[25] isolated a reovirus (type 3) from a single case of Burkitt's lymphoma and preliminary serological investigation by them suggests a higher incidence of antibody to this virus in children with lymphoma than in healthy children.

Epstein, Woodall and Thomson[26] inoculated fresh biopsy material from a child with Burkitt's lymphoma into 4 monkeys. A transmissible aetiological agent is highly probable since, in 2 of these, lesions of the long bones indistinguishable from those of Burkitt's lymphoma developed. Virological studies on our 5 patients are in progress at the Virus Cancer Unit of the South African Institute for Medical Research.

Early reports of series of cases have stated that leukaemia was not found in this condition and in this respect it resembles the animal 'leukaemias' in which localized tumours are the commonest manifestation. A recent paper,[27] however, reports leukaemia in 4 cases. In 3 of these aleukaemic leukaemia was seen and one patient developed a frankly leukaemic blood picture. No evidence of leukaemia was found in any of our cases.

The radiographic appearances of Burkitt's lymphoma have been well documented.[28-32] Briefly, the characteristic multifocal jaw lesion commences at the roots of the molars with lamina dura resorption and associated osteolytic foci. The foci spread rapidly, producing a ragged moth-eaten appearance, and the tumour breaks through the bone surfaces forming a soft-tissue mass with loosening, displacement and resultant loss of teeth. Retro-orbital spread causes exophthalmus. Deposits within the cranium increase the intracranial pressure with resultant sutural diastasis. Lytic lesions of the diploe are seen with secondary deposits, spreading rapidly to involve the scalp.

Peripheral bone lesions are not as characteristic nor as common as jaw lesions. Professor Cockshott of Nigeria[33] states that 'as a rule, the shaft is involved in young children in a diffuse fashion and in older children the localization is closer to the metaphysis . . . Sclerotic lesions . . . have not been seen in younger patients with Burkitt's but have been the feature of lymphoma in the late teens and early twenties.' In the Central African cases these lesions are described as mainly lytic with bone destruction and soft tissue involvement—periosteal reaction being relatively uncommon. In contrast our cases show involvement of the metaphysis and epiphysis with mixed sclerotic and lytic lesions and laminated periosteal reactions. Involvement of bones on either side of a joint does occur without actual joint involvement—probably due to multifocal lesions or metastases rather than a direct spread. Purely sclerotic lesions of epiphyseal centres of long bones have not been previously reported (Fig. 9).

Lesions of the vertebra are uncommon, but in patients presenting with paraplegia, deposits in the extradural space are demonstrated on myelography.

SUMMARY

The clinical, radiological and pathological features of 5 cases of Burkitt's lymphoma presenting within a relatively short period are presented. An additional 2 cases (1959 and 1961) found after a review of 10 cases of childhood malignant lymphoma seen during the past 10 years are briefly described.

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