Peripheral neuropathy and solitary plasmacytoma

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SUMMARY Three patients with peripheral neuropathy and a solitary plasmacytoma are presented, and the literature is reviewed. It is suggested that middle-aged men with an obscure progressive sensorimotor neuropathy, a raised CSF protein, and otherwise negative investigations should have a full skeletal survey since irradiation of a plasmacytoma may lead to a considerable improvement in the associated neurological disability.

The occurrence of peripheral neuropathy as a remote effect of multiple myeloma is well established. The association between a solitary plasmacytoma and peripheral neuropathy is, however, rare, and the literature consists mainly of reports of single cases. We report a further three cases, review the literature, and suggest that this association may not be as rare as previously thought and that the neuropathy, unlike most neuropathies secondary to malignant disease, may respond to treatment.

Case reports

CASE 1 (VS, UOH NO 508715)
A 39 year old Indian presented to hospital in Kenya with a three week history of pain in the back and calves. His calf pain became severe after walking 30 m which he could do only with difficulty. The left iliac crest was tender, he had a wide-based gait, diminution of vibration sense at the ankles, and absent ankle reflexes. Haematological and biochemical tests, including a glucose tolerance test and screening for porphyrins, were normal. Radiographs of skull, spine, and chest were normal but the otherwise normal intravenous pyelogram incidentally revealed a soap bubble appearance of the left ilium. The CSF contained 0.8 g/l of protein. Bone marrow examination was normal.

Two months later the patient was admitted to the Churchill Hospital complaining of difficulty in standing and climbing stairs, and slight weakness of both hands. He could just walk with the aid of two sticks. General examination was normal. Abnormal neurological signs were confined to the limbs where there was total areflexia without sensory loss. Power and co-ordination were normal in the arms. There was no voluntary movement in the toes or ankles; knee flexion and extension were weak on both sides with slight bilateral weakness of hip flexion.

Electrophysiological studies confirmed the diagnosis of a motor and sensory polyneuropathy and suggested axonal degeneration as the dominant pathological process. The following investigations were normal: full blood count; ESR; urea and electrolytes; liver function tests; serum calcium; fasting plasma glucose; urine and serum lead levels; screening for porphyrins; serum B12; LE cells; antinuclear factor (ANF); Wasserman reaction (WR) in blood and CSF; iophendylate myelography. The CSF protein was again raised at 1.25 g/l with two lymphocytes per mm³. Sural nerve biopsy showed severe axonal degeneration but no cellular or amyloid infiltrate. Radiographs of the left ilium showed a cystic lesion close to the sacro-iliac joint with some sclerotic areas suggestive of a plasmacytoma (Fig. 1). A biopsy sample of the lesion contained sheets of plasma cells. Investigations for myeloma were then carried out: skeletal survey and bone scan, chest radiograph, sternal bone marrow and serum protein electrophoresis were all normal. There was no Bence-Jones proteinuria; serum immunoglobulin levels (in g/l) were: IgG 10 (normal=8–16); IgA 0.86 (1.4–4.2); IgM 0.48 (0.5–1.9).

Over the next six months the patient received four-day courses of melphelan and prednisolone at intervals of six weeks but his condition worsened. Seven months after the onset he had developed weakness and wasting of both hands with paraesthesiae in the fingers; he was unable to walk more than 50 m with sticks and could stand for no more than eight minutes. Radio-
sent to the iliac crests and joint position sense impaired at and below the knees.

Electrophysiological studies confirmed the diagnosis of a mixed motor and sensory polyneuropathy, principally affecting the lower limbs with evidence of both axonal degeneration and segmental demyelination. Chest radiographs showed a slightly expanded right fifth rib with some lytic areas, and a lumbar spine film revealed slight uniform enlargement of a dense L2 vertebra (Figs 2 and 3). On the basis of tomography and of the negative investigations detailed below, the vertebral lesion was diagnosed as Paget's disease, and the rib abnormality was also regarded as an uncommon manifestation of the same condition (Dr

graphs had shown increasing size of the left iliac lesion, and local radiotherapy was given to a total tumour dose of 3600 rads.

Six months later his walking had improved so that he was able to walk for 30 m without the aid of sticks. Three and a half years later he was back at work, and after five and a half years the only residual signs were slight weakness of dorsiflexion at both ankles, absent ankle and supinator reflexes, and loss of vibration sense in both great toes. At intervals in the fifth and a half years after diagnosis he had repeat bone marrow examinations and skeletal surveys which have shown no progression of the original lesion or involvement of other sites. Measurements of the immunoglobulin levels every three months have shown no paraproteinemia and consistently normal values for IgG and IgM with persistent slight decrease in IgA.

CASE 2 (RM, UOH NO 506008)
A 49 year old man was admitted with a four month history of increasing difficulty in walking, and lower limb paraesthesiae. General examination was normal. In the arms there was minimal blunting to pinprick on the finger tips and absent supinator and triceps reflexes with just obtainable biceps reflexes. In the legs there was bilateral flaccid foot drop with slight weakness of the more proximal muscles, absent tendon reflexes and flexor plantar responses. Vibration sense was ab-

Fig. 1 VS: lytic/sclerotic appearance of the left iliac lesion.

Fig. 2 RM: radiological appearance of right fifth rib in 1971.

Fig. 3 RM: lumbar spine showing Paget's disease of the second lumbar vertebra.
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F. H. Kemp). Skeletal survey was normal as were a myelogram, an abdominal lymphangiogram with intravenous pyelogram, and a barium meal and follow-through. The following investigations were also normal or negative: full blood count; ESR; serum urea and electrolytes; liver function tests; serum calcium; glucose tolerance test; serum acid phosphatase; faecal fat excretion; faecal occult blood, serum folate; serum protein electrophoresis and immunoelectrophoresis; ANF; LE cells; Bence-Jones protein; urinary porphyrins. One serum B12 level was slightly low at 150 pg/ml (normal >170) but a double Schilling test and bone marrow examination were both normal, and no antibody against intrinsic factor was detected. The CSF contained a protein level of 1.3 g/l and the WR was negative in both blood and CSF.

The patient was discharged on monthly injections of vitamin B12 but deteriorated slowly over the next two years. He was then readmitted with gross wasting and weakness of the hands and forearms, and early clawing of the fingers. Tendon reflexes were absent and vibration sense lost to the shoulders though other modalities were intact. The lower limb signs remained unchanged.

Electrophysiological studies confirmed severe denervation of the hands, and repeat radiography of the chest and lumbar spine showed no change in the previous abnormalities. Routine biochemical and haematological tests were repeated with no abnormality, and, in particular, the protein electrophoretic strip remained normal. Repeat CSF examination showed a fall in the protein content to 0.8 g/l.

Slow deterioration continued, and 34 months after the onset prednisolone 40 mg on alternate days was started with initial improvement. It was reduced to 5 mg on alternate days over the next 21 months. The patients' condition worsened, and he was readmitted four and a half years after the onset. He could walk only 10 m with the aid of two sticks which he held with difficulty because of his hand deformity. All limbs were wasted and weak with fixed flexion deformities of both hands and areflexia. All sensory modalities were diminished in a glove and stocking distribution, but with preservation of joint position sense in the hands.

Routine haematological and biochemical tests were again all normal, the serum B12 level on this occasion being >750 pg/ml. No organ specific antibodies or ANF were detected. Protein electrophoresis showed a faint IgG λ monoclonal band too small to quantify. Serum immunoglobulin levels were within the normal range [IgG 9.24 (7.2–19); IgA 2.77 (0.8–4.8); IgM 1.2 (0.5–2.00) all g/l], and electrophoresis of concentrated urine showed neither abnormal proteins nor Bence-Jones proteose. The CSF protein concentration was 0.55 g/l. Sural nerve biopsy showed considerable axonal degeneration but no infiltration with amyloid. Lumbar spine radiography showed no change in the L2 vertebra (which was still regarded as Paget's disease (Dr F. W. Wright) but a chest radiograph revealed considerable expansion of the right fifth rib (Fig. 4). On biopsy this was found to consist of oedematous fibrous tissue infiltrated with masses of plasma cells. Bone marrow from two other sites (sternum and iliac crest) was normal.

The rib was irradiated with a total tumour dose of 3500 rads, and in the month after this the patient's walking ability improved considerably (one mile with sticks) but his upper limbs did not. Eighteen months after irradiation of the plasmacytoma his walking had improved further, he was back at full time clerical work, and was able to dig his garden. Power at his shoulders, elbows, hips, and knees was normal, but his ankle dorsiflexors were weak and his forearm muscles usefully strong though not normal. The small muscles of the hands remained paralysed but there was no sensory loss in either arms or legs.

CASE 3 (EW, UOH NO 652966)
A 58 year old man presented with a three month history of progressive flaccid foot drop with increasing difficulty in walking such that he had to use two sticks. In addition he gave a six week history of paraesthesiae in both feet, calves, and hands. There were no other neurological symptoms, and his general health was good. Two months before admission he had noticed a swelling over the left clavicle associated with a patch of red
scaly skin. This was not painful and had not altered in size since first noticed.

On examination there was a patch (90 × 150 mm) of warm, erythematous, hyperaemic, and slightly scaly skin overlying a hard but non-tender expansion of the outer end of the left clavicle. General examination showed an enlarged left axillary lymph node but was otherwise normal. There was slight weakness of both wrists and more pronounced weakness of the small muscles of the hands. Power at hips and knees was normal but there was marked weakness at the ankles and no voluntary movement of the toes. He was areflexic with a glove and stocking distribution of sensory loss to all modalities. A dermatological opinion on the skin lesion suggested a diagnosis of malignant angioendothelioma.

Electrophysiological studies confirmed a polyneuropathy worse in the lower limbs, with evidence of much axonal degeneration in the legs and segmental demyelination in the arms. Radiographs of the clavicle revealed an expanded cystic lesion suggestive of a plasmacytoma (Fig. 5).

A skeletal survey did not show any other lesions, and the chest film was normal. The enlarged left axillary gland, the clavicle, and the overlying skin were all biopsied. Neither the node nor the skin showed any evidence of malignant or amyloid infiltration, but the left clavicular biopsy specimen contained sheets of mature plasma cells in which immunoperoxidase stains failed to demonstrate a monoclonal population. Iliaic crest bone marrow was normal, and the CSF protein level was 0.6 g/l. The following investigations were either normal or negative: full blood count; ESR; fasting plasma glucose; serum urea and electrolytes; liver function tests; serum calcium; serum B12; WR in blood and CSF; serum protein electrophoresis. There was no Bence-Jones proteinuria and serum immunoglobulin levels were within the normal range.

During the next month weakness progressed to involve movements at both knees and elbows. The patient was then given a course of radiotherapy to the clavicular lesion (3600 rads) after which he reported subjective improvement in both the paraesthesiae and sensory loss but only eight months after treatment did his signs show improvement with increased strength at the left wrist and at both ankles.

Discussion

The neuropathy of multiple myeloma is well documented and occurs clinically in about 3% of cases (Silverstein and Doniger, 1963; Currie et al., 1970). Walsh (1971) demonstrated electrophysiological abnormalities in a much larger proportion (39%) and, histologically, demyelination has been observed in 65% (Hesselvik, 1969). By comparison, the association between a solitary plasmacytoma and polyneuropathy is much rarer. We have found a total of 16 acceptable cases of peripheral neuropathy associated with a solitary plasmacytoma including the three described in this paper (Table 1), and another case (Currie et al., 1970) mentioned in passing but not included in our review as no detailed information was given. One case claimed as a solitary lesion by the authors (Morley and Schweiger—case 2) was not included since the bone marrow contained a 7% increase in abnormal plasma cells, indicating a generalised process. A further case (Victor et al.—case 2) was included, though not accepted by the authors as solitary on the grounds that at postmortem examination the bone marrow was not examined. Discussing the case, however, they state that “repeated radiological and sternal marrow examination did not reveal evidence of disseminated myeloma.”

The age of the patients at presentation ranged from 34 to 67 years with a mean of 49 years. Previous authors have commented on the relatively younger age of patients with multiple myeloma and peripheral neuropathy compared with the average age (59 years) of presentation of patients with uncomplicated multiple myeloma (Victor et al., 1958; Morley and Schweiger, 1967; Davis and Drachman, 1972). This has been attributed to the fortuitous early discovery of the myeloma during investigation of a peripheral polyneuropathy, and this explanation is supported by the present review.

The 16 patients in this series show a striking...
## Table 1 Details of 16 patients with a polyneuropathy associated with a solitary plasmacytoma

<table>
<thead>
<tr>
<th>Case</th>
<th>Source</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Duration of symptoms to diagnosis</th>
<th>Site and type of tumour</th>
<th>CSF protein (g/l)</th>
<th>Serum proteins</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Length of follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Scheinker (1938)</td>
<td>39</td>
<td>M</td>
<td>15 m*</td>
<td>Sternal</td>
<td>2.0</td>
<td>--</td>
<td>Typhus vaccine &quot;Spinal x-rays&quot;</td>
<td>Death in 15 m</td>
<td>---</td>
</tr>
<tr>
<td>2</td>
<td>Crow (1956), case 2</td>
<td>67</td>
<td>F</td>
<td>7 m</td>
<td>Sternal Lytic and sclerotic</td>
<td>0.8</td>
<td>Initially normal†</td>
<td>DXT 2500 rads</td>
<td>Symptomatic improvement</td>
<td>18 m</td>
</tr>
<tr>
<td>3</td>
<td>Victor et al. (1958), case 2</td>
<td>48</td>
<td>M</td>
<td>9 m*</td>
<td>L1 vertebra Lytic</td>
<td>0.9</td>
<td>Normal</td>
<td>None</td>
<td>Death in 9 m</td>
<td>---</td>
</tr>
<tr>
<td>4</td>
<td>Small et al. (1961)</td>
<td>41</td>
<td>M</td>
<td>3 m</td>
<td>L2 vertebra Lytic and sclerotic</td>
<td>2.24</td>
<td>Normal</td>
<td>None</td>
<td>Death in 7 m</td>
<td>---</td>
</tr>
<tr>
<td>5</td>
<td>Rohmer et al. (1962)</td>
<td>34</td>
<td>M</td>
<td>9 m</td>
<td>Left fifth rib Lytic</td>
<td>1.20 to 3.20</td>
<td>Normal</td>
<td>DXT 5250 rads</td>
<td>Improvement</td>
<td>23 m</td>
</tr>
<tr>
<td>6</td>
<td>Rushton (1965)</td>
<td>45</td>
<td>M</td>
<td>20 m*</td>
<td>D11 vertebra Lytic and sclerotic</td>
<td>2.40</td>
<td>Normal</td>
<td>Steroids DXT</td>
<td>Improvement</td>
<td>2 yr</td>
</tr>
<tr>
<td>7</td>
<td>Gupta and Prabhakar (1965)</td>
<td>35</td>
<td>M</td>
<td>75 m</td>
<td>Right scapula Lytic</td>
<td>0.60 to 1.20</td>
<td>Normal</td>
<td>CT</td>
<td>Death in 12 m</td>
<td>---</td>
</tr>
<tr>
<td>8</td>
<td>Morley and Schweiger (1967), case 1</td>
<td>51</td>
<td>M</td>
<td>12 m*</td>
<td>Left third rib Lytic</td>
<td>3.35 to 10</td>
<td>Assumed normal</td>
<td>DXT</td>
<td>Death in 14 m</td>
<td>---</td>
</tr>
<tr>
<td>9</td>
<td>Morley and Schweiger (1967), case 3</td>
<td>63</td>
<td>M</td>
<td>3 m</td>
<td>Right acetabulum</td>
<td>0.7</td>
<td>Raised beta-2 globulin</td>
<td>None</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>10</td>
<td>Davidson (1972)</td>
<td>62</td>
<td>M</td>
<td>6 m</td>
<td>Sacrum and coccyx Lytic</td>
<td>0.56</td>
<td>Normal</td>
<td>Excision DXT 4200 rads</td>
<td>Improvement</td>
<td>6½ yr</td>
</tr>
<tr>
<td>11</td>
<td>Davis and Drachman (1972), case 1</td>
<td>46</td>
<td>M</td>
<td>27 m</td>
<td>Right acromion Lytic</td>
<td>--</td>
<td>IgG λ paraprotein†</td>
<td>DXT 4000 rads</td>
<td>Improvement</td>
<td>10 yr</td>
</tr>
<tr>
<td>12</td>
<td>Davis and Drachman (1972), case 2</td>
<td>49</td>
<td>M</td>
<td>2 yr</td>
<td>Left humerus Lytic and sclerotic</td>
<td>2.25</td>
<td>IgG paraprotein†</td>
<td>DXT 3750 rads (Steroids)</td>
<td>Improvement</td>
<td>2 yr</td>
</tr>
<tr>
<td>13</td>
<td>Getaz et al. (1974)</td>
<td>64</td>
<td>M</td>
<td>12 m</td>
<td>D5–7 vertebrae Lytic and sclerotic</td>
<td>5.0</td>
<td>No paraprotein detected</td>
<td>CT DXT 4500 rads</td>
<td>Lost to follow-up at six weeks</td>
<td>---</td>
</tr>
<tr>
<td>14</td>
<td>This paper, case 1</td>
<td>39</td>
<td>M</td>
<td>1 m</td>
<td>Left ilium Lytic</td>
<td>0.80 to 1.25</td>
<td>Normal</td>
<td>CT DXT 3600 rads</td>
<td>Improvement</td>
<td>5½ yr</td>
</tr>
<tr>
<td>15</td>
<td>This paper, case 2</td>
<td>49</td>
<td>M</td>
<td>4½ yr</td>
<td>Right fifth rib Lytic</td>
<td>1.3</td>
<td>Initially normal†</td>
<td>Steroids DXT</td>
<td>Improvement</td>
<td>18 m</td>
</tr>
<tr>
<td>16</td>
<td>This paper, case 3</td>
<td>58</td>
<td>M</td>
<td>3 m</td>
<td>Left clavicle Lytic and sclerotic</td>
<td>0.6</td>
<td>Normal</td>
<td>DXT 3600 rads</td>
<td>Improvement</td>
<td>8 m</td>
</tr>
</tbody>
</table>

* Diagnosed post mortem.
‡ See text.
mm — months, DXT = radiotherapy, CT = chemotherapy.
male predominance of 94%, and in many ways the only female patient was atypical. The neuropathy in her case was mild, confined almost entirely to the sensory side, and her neoplastic disease, despite radiotherapy, progressed to involve abnormal serum proteins, increasing albuminuria, and lymphadenopathy, which did not occur in any of the surviving males. Male preponderance has also been noted previously in mixed multiple and solitary lesions—74% in Morley and Schweiger’s series and 76% in that of Davis and Drachman.

Symptoms of polyneuropathy were the presenting complaint in all 16 of the patients, and the average interval between onset of symptoms and diagnosis was 13 months, with a range of one to 54 months. The clinical features of the neuropathy were similar in 15 of the 16 cases and have been described as typically a “progressive, symmetrical, atrophic, areflexic, sensorimotor affection of the legs and arms” (Victor et al., 1958). With only one exception (case 2) the lower limbs were affected first with a symmetrical progressive sensorimotor disturbance going on to affect the upper limbs in all but one (case 7), and involving the trunk and respiratory muscles in the severe cases. The severity and rate of progression of the neuropathy varied considerably in the untreated state from death in seven months due to respiratory failure (case 4) to gradually increasing limb disability in four and a half years before definitive diagnosis and treatment (case 15). The cranial nerves were not affected in 14 of the patients but two, both with very severe neuropathies, developed facial weakness. Only seven of the 16 patients had limb pains, a feature said to be characteristic of myelomatous neuropathy (Davis and Drachman, 1972).

The site of the plasmacytoma was unpredictable but there was an unusually high proportion of sclerotic as opposed to the more usual lytic lesions. Ten of the 16 (62.5%) had either entirely or partially sclerotic lesions. The presence of sclerosis in the lesions of multiple myeloma is said to be rare (Lowbeer, 1969), and the correlation between this and the development of polyneuropathy, though at one time thought to be coincidental (Aguayo et al., 1964), is now well established (Morley and Schweiger, 1967; Davis and Drachman, 1972; Getaz et al., 1974; Mathews and Olivier, 1974). Three of the patients had discrete skin lesions over the site of the tumour (cases 1, 5, 16). Two of these were biopsied (cases 1 and 16) and showed only nonspecific increase in collagen fibres and mild lymphocytic infiltration.

Routine investigations were generally unrewarding (Table 2). Bone marrow examination from a site remote from the plasmacytoma was normal in 14 cases and was not performed in the other two (cases 2 and 4). The ESR was normal in the 12 patients in whom it was performed. In none of the 16 was any abnormal urinary protein or Bence-Jones proteose detected. Lumbar puncture was carried out on 15 patients, and in all the CSF showed a rise in protein varying from 0.56–10 g/l. Protein electrophoresis was carried out on the serum of all except case 1. At presentation, 13 patients showed no evidence of a monoclonal paraprotein. Two patients however had IgG para-proteinemias: case 11, who made a good clinical recovery, possessed an IgG k paraprotein which persisted throughout his 10 year follow-up period without any evidence for recurrence of his irradiated plasmacytoma or development of multiple myeloma; and case 12 had an IgG paraprotein and quantitatively elevated IgG immunoglobulin which returned to normal with disappearance of the paraprotein nine months after treatment. Two of the 13 patients with initially normal electrophoretic patterns (cases 2 and 15) later developed “abnormal serum proteins” and a faint IgG λ band respectively. Histological examination of peripheral nerves obtained either postmortem or from open biopsy was performed in five patients (cases 1, 4, 9, 14, 15). In none was infiltration with either amyloid or neoplastic cells found, the picture being one of mixed demyelination and axonal loss.

### Table 2 Characteristics of patients with solitary plasmacytoma and peripheral polyneuropathy—total of 16 cases

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Neurorpathy</th>
<th>Presenting complaint</th>
<th>Tumour site</th>
<th>Tumour type</th>
<th>CSF protein</th>
<th>Urine</th>
<th>ESR</th>
<th>Serum proteins at presentation</th>
<th>Bone marrow</th>
<th>Neuropathy</th>
<th>Skin lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male; one female</td>
<td>Mean 49; range 34–67 years</td>
<td>Symmetrical, progressive, sensorimotor, legs affected first and to a greater degree than arms</td>
<td>Neurorpathy in all 16 cases</td>
<td>Vertebral column in five cases</td>
<td>Sclerotic in 10 cases</td>
<td>Raised in 15 cases, not recorded in one</td>
<td>No Bence-Jones proteinuria in 16 cases</td>
<td>Normal in 12 cases, not recorded in four</td>
<td>Normal in 13 cases, IgG paraprotein in two, not recorded in one</td>
<td>Normal in 14 cases, not recorded in two</td>
<td>No amyloid. Demyelination, and axonal loss in five cases</td>
<td>Over site of tumour in three cases</td>
</tr>
</tbody>
</table>
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In their review of myelomatous peripheral neuropathy Morley and Schweiger (1967) formed the conclusion that “the neuropathy usually progressed relentlessly to death within two years”, making a modified exception in the case of the solitary lesions. Localised radiotherapy to such lesions indeed proves to be effective not only in arresting the progress of the neuropathy but, in most cases, in allowing a degree of recovery to take place. Prognosis and treatment were closely linked (Table 3). Six of the 16 patients received chemotherapy alone.

Table 3 Treatment and prognosis in 16 patients with plasmacytoma and polyneuropathy

<table>
<thead>
<tr>
<th>Treatment groups</th>
<th>Number of patients</th>
<th>Survival Improvement</th>
<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>4</td>
<td>Yes - No 4</td>
<td></td>
</tr>
<tr>
<td>Chemotherapy alone</td>
<td>1</td>
<td>- 1</td>
<td></td>
</tr>
<tr>
<td>Steroids</td>
<td>3*</td>
<td>- 3</td>
<td></td>
</tr>
<tr>
<td>DXT</td>
<td>5</td>
<td>5 -</td>
<td></td>
</tr>
</tbody>
</table>

*Same group of patients.
†One lost to follow-up at six weeks.

either no treatment or some form of chemotherapy or steroids, and all died in seven to 20 months (mean 13 months) from the onset of symptoms. Five died from respiratory failure often complicated by pneumonia, and not as a direct consequence of the plasmacytoma. The mode of death in the remaining patient (case 9) was not recorded. In four of these (cases 1, 3, 6, 8) the definitive diagnosis was pot made until post-mortem examination, though the bony abnormality had been discovered previously in three (cases 1, 6, 8). The other 10 patients all received a course of localised radiotherapy. Up to the time of reporting all these patients had survived with a documented follow-up period from six months to 10 years. The peripheral neuropathy had improved in all and had progressed in none. Two of the patients had been lost to follow-up, one at six weeks (case 13) and the other after 18 months (case 2). Of the remaining eight, five were considered to have made a good recovery and three had achieved some improvement.

The apparent rarity of the association between polyneuropathy and solitary plasmacytoma is surprising considering the appearance of three cases in our department in the last six years, and this may be connected with the neurological mode of presentation of the condition. The neuropathy of multiple myeloma almost always appears as a complication of the established disease (Silverstein and Doniger, 1963) whereas that associated with a solitary lesion presents with the neuropathy. A full skeletal survey is not routinely carried out during the investigation of an obscure peripheral neuropathy, and the chances that a small plasmacytoma will be overlooked must be high, if it is not in the area covered by a routine chest radiograph. The diagnosis of this potentially treatable cause of an otherwise progressive neuropathy must, therefore, be considered in all young to middle-aged men with an obscure progressive sensori-motor neuropathy affecting predominantly the lower limbs and in whom all routine investigations are normal except for a rise in CSF protein. We suggest that all such patients should have a full skeletal survey, isotope bone scans being a less effective means of detecting myelomatous bony lesions. We also suggest that in cases of proven solitary plasmacytoma localised radiotherapy is the treatment of choice.

We are indebted to Professor W. B. Matthews and Dr J. M. K. Spalding for permission to report cases admitted under their care (cases 1 and 2—Dr Spalding, case 3—Professor Matthews) and for much valuable discussion of this paper. We should also like to thank Dr Geoffrey Rushworth who carried out the electrophysiological studies on all these patients, and Dr A. H. Laing and Dr J. M. Holt for advice on the treatment of the plasmacytoma. Our thanks are also due to Vanessa Wilkins for her expert typing of the manuscript.

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