Medical-surgical treatment of progressive tuberculous (Pott's) paraplegia in Gabon

P-M Loembe

Department of Neurosurgery, Omar Bongo University, Fondation Jeanne Ebori Hospital, BP 212, Libreville, Gabon, Central Africa

The present study deals with the results of the medical-surgical treatment of 22 patients with Pott's tetraplegia or paraplegia. Seventeen had progressive tetraplegia–paraplegia which failed to respond solely to medical treatment. On admission, four patients exhibited an acute onset tetraplegia–paraplegia, and one had a 'spinal tumour syndrome'. In addition to antituberculous therapy, seven patients had anterior spinal surgery, consisting of four corporectomies, two anterior debridments and grafting, and one debridment alone. Moreover, one patient had a posterior interbody fusion, four had laminotomies, and 10 had laminectomies. The causes of the spinal cord or cauda equina compression, as was determined at operation, were extradural abscess in eight patients, bony compressions in 11, arachnoiditis in two, and posterior neural arch tuberculosis in one patient. Neurological recovery began between 10 and 21 days postoperatively. The mean length of follow-up was 42.36 months (range 8–144 months). Fourteen patients were found to be functionally and neurologically normal at follow-up examinations (63%). Eighty-two per cent recovered sufficiently to walk unaided. Two patients were left paralysed and unable to walk. Two patients were able to get about on crutches. The onset of objective improvement soon after surgical decompression suggests a causal effect. It was concluded that early neural decompression and spinal stabilisation provided the maximum potential for neurological recovery.

Keywords: spinal tuberculosis; Pott's paraplegia; tetraplegia; medical-surgical treatment; Africa; Gabon

Introduction

Involvement of the spinal cord or cauda equina is a serious and distressing complication of tuberculous spondylitis (Pott's disease). Because some patients with tuberculous paraplegia (Pott's paraplegia)¹ respond well to chemotherapy alone,² the exact role of surgery in this disease has been a matter of controversy. Recently, an attempt was again made to assess the value of surgery as a factor in the resolution of neurological deficits in those with spinal tuberculosis.^{3–6} The present study is concerned with the results of medical-surgical treatment in 22 patients with Pott's tetraplegia/paraplegia. Our medical centre is the only one qualified surgically to handle this type of pathology in Gabon, and works in collaboration with the Libreville Antituberculous Centre.

Material and methods

Patients

From January 1982 to May 1994, 22 patients with Pott's tetraplegia–paraplegia underwent surgery in the neurosurgical department of the Fondation Jeanne Ebori Hospital (Gabon). There were 13 male (59%)

and nine female (41%) patients, with an average age of 45.6 years (range 5–68 years). In 95% of the cases the mean age was 47.6 years (range 27–68 years). The preoperative clinical features are summarised in Table 1. Seventeen patients had a progressive tetraplegia–paraplegia which failed to respond to an initial 3 week course of antituberculous therapy. On admission, four patients had an acute onset tetraplegia–paraplegia, and a 'spinal tumor syndrome' was found in one. The duration of the neurological deficit varied from 1 day to 3 months (mean: 4 weeks). Four patients required ambulatory assistance, and 18 were not ambulatory. There was severe pain (neck, back, or radicular) in 91% of the patients. All of them had minor constitutional manifestations of low grade fever, loss of weight, and lassitude.

Preoperative investigations

Twenty patients (91%) had a positive tuberculin skin test, and two (9%) had pulmonary tuberculosis. The systematic examination of sputum, gastric lavage and urine did not demonstrate any tubercle bacilli. Preoperative radiological features are shown in Table 2. The thoracic spine was involved in eight patients,

Table 1 Preoperative clinical features

<i>Symptoms and signs</i>	<i>No. of patients (n = 22)</i>
Initial complaints	
Neck pain	5
Back pain	12
Radicular pain	3
Functional disability	22
Kyphosis	3
Neurological status (grade) ^a	
2a	4 (18.2%)
2b	2 (9.1%)
3	4 (18.2%)
4	8 (36.3%)
5	4 ^b (18.2%)

^aCriteria of Pottis²

^bTetraplegia–paraplegia of acute onset

Table 2 Preoperative radiological features

<i>Investigation</i>	<i>No. of patients (n = 22)</i>
Radiographs/tomograms	22
Normal	1
Vertebral body collapse	12
Erosion/partial destruction	9
Associated lesion of posterior neural arch	1
Myelography	20 (91%)
Total block	12
Partial block	8
Level	
C3–7	6 (27.2%)
T1–11	8 (36.3%)
T12–L2	5 (22.7%)
L3–5	3 (13.6%)

cervical in six, thoracolumbar in five, and lumbar in three. Vertebral body collapse was the most prominent finding. A mean of 1.5 vertebral bodies were involved. In one patient, standard radiographs were normal. Myelography was performed in 20 patients.

The medical-surgical treatment

The medical treatment consisted of the early administration in one group (first eight patients) of rifampin, isoniazid and ethambutol for 18 months; and, in the second group (remaining 14 patients), of rifampin, isoniazid, ethambutol and pyrazinamide for 6 months, according to the World Health Organization recommendations.⁷ This medical treatment was systematically started 3 weeks before surgery in 17 patients (77%), once the disease had been diagnosed radiologically. Four patients with an acute onset of paraplegia, and one with a 'spinal tumour syndrome',⁸ underwent surgery on an emergency basis.

Therefore, the indications for surgery were: neurological (ineffectiveness of medical treatment or acute onset paraplegia) in 13 patients, neurological and mechanical in six, and aetiological and neurological in three cases. The surgical approach was anterior in seven patients, and posterior in 15 (Table 3). The indications for operation, and the operative technique in each patient are specified in Table 4. Our indications for anterior stabilisation was spinal instability due to vertebral body destruction, particularly in the cervical and lumbar segments. We have recently described various surgical procedures used in our centre.^{6,7} Anterior decompression and fusion consisted of a corporectomy/debridement, grafting, with or without plating, in six patients: 1, 10, 12, 13, 15, 17 (Table 4). The use of fixation instrumentation in Pott's disease has been previously discussed.⁶ Posterior stabilisation (posterior interbody fusion) was performed conjointly with a laminectomy in one patient (18), to prevent an instability or a kyphosis. In the thoracic region, a significant kyphosis (of more than 40 degrees) was not found, thus surgical correction was not considered to be necessary. Laminotomy was occasionally indicated for posterior decompression for an abscess. Laminectomy was used for thoracolumbar posterior compression by tuberculous arachnoiditis, sclerotic bone, or associated posterior vertebral tuberculosis.

Operative findings

During the operation (Table 5), the causes of spinal cord or cauda equina compression appeared to be an extradural abscess (eight patients), bony compression (11), arachnoiditis (two), and posterior neural arch tuberculosis (one patient).

Postoperative investigations

The culture of caseous material obtained during surgery gave negative results. Microscopic examination of surgical specimens revealed granulomatous features in 18 patients (82%), but no acid fast bacilli could be isolated.

Table 3 Operative techniques in 22 patients

<i>Techniques</i>	<i>No. of patients</i>
Anterior approach (32%)	
Corporectomy, grafting and plating	4
Debridement with grafting only (H) ^a	1
H and Blount's clip	1
Debridement (only) and biopsy	1
Posterior approach (68%)	
Lumbar interbody fusion	1
Laminotomy	4
Laminectomy only	8
Laminectomy with microsurgery	2

^aH = Hodgson's technique or the Hong Kong operation^{6,15,18}

Table 4 Summary of 22 patients

<i>Patient</i>	<i>Sex</i>	<i>Age (y)</i>	<i>Clinical radiographic data</i>	<i>Indications for surgery</i>	<i>Operative techniques and findings</i>
1	M	51	Neck pain Tetraplegia (grade 5), AO C5 destruction, C5 TB	SpCC C5, C6 instab	C5 Corp, G and SP. Abscess, sequestra
2	M	40	Back pain Paraplegia (G3), PD T3 collapse and TB	SpCC	T3 laminectomy Abscess
3	M	34	Back pain Paraplegia (G5), AO T12 collapse and TB	SpCC	T12 laminotomy Abscess
4	W	30	Neck pain, tetraparesis (G2a) PD, C3 erosion, C3 PB	SpCC	C3 debridement, biopsy Abscess
5	W	68	Back pain Paraplegia (G4), PD T11 collapse and TB	SpCC	T10, 11 laminectomy Sclerotic bone ^a
6	W	33	Paraplegia (G5), AO T3 collapse and TB	SpCC	T2, 3 laminectomy Sclerotic bone ^a
7	W	27	Back pain Paraplegia (G3) PD T12–L1 kyphosis (30°) T12–L1 erosion and TB	SpCC	T12, L1 laminectomy Sclerotic bone ^a
8	W	33	Back pain Paraplegia (G4), PD T12 erosion and T12, L1 TB	SpCC	T12–L1 laminectomy and microsurgery Arachnoiditis
9	M	60	Back pain Paraplegia (G2b), PD T8 collapse and PB	SpCC	T8, 9 laminectomy Sclerotic bone ^a
10	W	67	Back pain Paraplegia (G3), PD L4 erosion, L3 collapse	L2, 4 instab Neurological	LRA: L3, 4 debridement G and Blount's clip Caries, little pus
11	M	65	Back pain, paraplegia (G4), PD T8, 9 collapse, T8 TB and erosion of T8 posterior neural arch	SpCC	T8, 9 laminectomy Caries of T8 posterior neural arch
12	W	56	Neck pain, paraplegia (G5), AO C6 erosion, C7 collapse, C7–T11, TB.	SpCC C6, 7 instab	C6, 7 corp and SP Sequestra, sloughed disk, little pus
13	M	61	Neck pain, tetraplegia (G4), PD C3 discreet erosion C4 collapse, C5 destruction C4, C5 kyphosis (38°), C6 TB	SpCC C3, 4, 5 instab	C4, 5 debridement and G Sequestra, sloughed disk, little pus
14	M	49	Back pain Paraplegia (G3), PD T12–L1 erosion and PB	SpCC	T12, L1 laminectomy Sclerotic bone ^a
15	M	51	Radicular pain Tetraplegia (G4), PD C4, 5, 6 erosion, C6 TB	SpCC C5, 6 instab	C5, 6 Corp G and SP Caries, little pus
16	M	57	Back pain Paraplegia (G4), AO T9 erosion, T8, 9 TB	Aetiological SpCC	T9 laminotomy Abscess
17	M	42	Neck pain, tetraparesis (G2a), PD C5 collapse, C6 erosion C5, 6 kyphosis (30°)	SpCC C5, 6 instab	C5, 6 Corp and G Sequestra Sequestrated disk, pus
18	M	42	Radicular pain Paraparesis (G2a), PD L3 erosion, L3, 4 PB	Aetiological Neurological	L3, 4 laminectomy and posterior interbody fusion. Caseous material and sequestrated disk
19	W	42	Back pain, paraplegia (G4) T8, 9 collapse, T10 PB	SpCC	T10 laminotomy Abscess
20	M	5	Paraparesis (G2a), PD Normal radiographs T12–L1 PB	SpCC	T12–L1 laminectomy and microsurgery Arachnoiditis

Table 4 (Cont)

Patient	Sex	Age (y)	Clinical radiographic data	Indications for surgery	Operative techniques and findings
21	M	45	Radicular pain Paraplegia (G2b), PD L3 discrete erosion, L3, 4 PB	Aetiological neurological	L4 laminotomy Abscess
22	W	47	Back pain Paraplegia (G4), PD T9, 10 collapse, T10 PB	SpCC	T10 laminectomy Sclerotic bone ^a Sheets of fibrous tissue

^aPosterolateral compression by sclerotic bone.

AO: acute onset; TB: total block (myelography); PB: partial block; SpCC: spinal cord compression; Instab: instability; Corp: corporectomy; G: grafting; SP: screwed plate; PD: progressive deterioration; LRA: left retroperitoneal approach

Follow-up

Postoperatively, all patients had to follow a re-education programme. Radiographs were taken weekly when the patient was in hospital, and then monthly until the spinal column was stable. All of the patients were then followed at 3 monthly intervals for 1 year, and at 6 monthly intervals thereafter. Nevertheless, two patients were followed up for a year and did not return for the semi-annual examinations. The effective medical recovery of the patients was announced by the anti-tuberculous centre.

Results

There were no major intraoperative accidents in this series, and no intraoperative deaths. There were no neurological, mechanical or miscellaneous infectious complications. Neurological recovery began between 10 and 21 days postoperatively. The mean length of follow-up (Table 6) was 42.36 months or 3.5 years (range 8–144 months). The results of therapy are summarised in Table 7, showing the findings at the last neurological examination. Sixty three per cent were found to be functionally and neurologically normal. Eighty two per cent recovered sufficiently to walk unaided. Two patients were left paralysed and unable to walk. Two patients were able to get about on crutches. Relief of pain and osseous consolidation occurred rapidly. All patients were considered to be medically cured.

Table 5 Summary of potential causes of spinal cord or cauda equina compression, according to the operative findings

Causes	No. of patients	%
Abscess	8	36.3
Bone compression:	11	50
Posterolateral compression by sclerotic bone,	6	
Anterior osseous focus, with little pus	5	
Arachnoiditis	2	9.1
Posterior neural arch tuberculosis	1	4.5

Table 6 Duration of follow-up

Duration (months)	No. of patients (n = 22)	%
8–12	6	27.2
13–24	7	32
25–36	1	4.5
37–48	1	4.5
49–60	3	13.6
61–120	2	9.1
121–144	2	9.1

Table 7 Postoperative neurological status

Grade	No. of patients	%
0	14	63.6
1	4	18.2
2a	2	9.1
2b	1	4.5
3	—	
4	—	
5	1	4.5

Discussion

Despite the World Health Organization efforts to improve public health, tuberculosis remains a serious problem in Africa, and more especially in Gabon. In North American Society⁵ and perhaps in Europe, there appears to be a recrudescence of tuberculosis. The incidence of neurological impairment varies according to the series and the country concerned.^{6,8,9} In our experience,¹⁰ it was 21% as compared to 25% in the report of Auregan *et al* in Djibouti.¹¹ As can be seen from the present data, our patients were mostly in the middle age group; this is a tendency in our medical centre, for approximately 6 years. Our patients developed an entirely different kind of clinical spinal tuberculosis from that prevalent in the Orient and in Africa, 20–40 years ago.^{8,10,12} That is to say it was more localised and less pus was produced.

Eighteen patients (82%) had a progressive tetraplegia–paraplegia, whereas four tetraparetic–paraparetic patients (18%) exhibited acute worsening of their neurological deficit (paraplegia grade 5). Rail and air transport facilities had allowed the transfer of patients in a reasonable time to our centre. Unfortunately, this is not always the case in most developing countries.

The most important cause of paraplegia in patients with spinal tuberculosis is compression; other factors may rarely be contributory or solely responsible.⁸ Tandon has never seen a patient with paraplegia in association with tuberculosis of the spine, in the absence of a demonstrable compression of neural tissue.⁸ Most recent authors favour this view.^{3,4,9,12} According to Martin,¹² abscess formation is more frequently seen in Asian and African people compared to Europeans; moreover, he found pus to be responsible for paraplegia in only 29% of his European patients. The present study supports the view that several factors may contribute to the development of tuberculous paraplegia.^{6,8,12} Indeed, the main causes of spinal cord or cauda equina compression appear to be bone-linked (50%) or abscess-linked (36.3%). Nevertheless, as is shown in Table 4, they are generally intricate. Most patients were seen in the advanced stages of the disease, with bone destruction, associated with collapse of vertebrae in 12 patients (54%). In the report of Acikgöz *et al*,³ 70% of the patients had vertebral body collapse, and in more than half an epidural abscess was found. Sequestra are formed from avascular portions of a vertebral body or intervertebral disc and these add to the effect of the spur in narrowing the spinal canal. It must be emphasised that very considerable deformity of the canal can occur without paralysis which develops in those patients with a bony disorder, because of the formation of bony spurs and sequestra.⁸ Manifestations of tuberculosis of the coverings of the spinal cord include arachnoiditis, and subdural and extradural granulomas. Tuberculous arachnoiditis is seen in patients who have had tuberculous meningitis in which thick, densely organised exudates encase the entire length of the spinal cord.⁹ Such widespread infection may cause arteritis and secondary vascular changes leading to ‘transverse myelitis’ or motor loss. Excision of local arachnoidal granulomas may provide some relief.¹³ Two patients in our series had arachnoiditis (8 and 20, Table 4). They had, respectively, a partial (grade 2a) syndrome and full recovery. One of them (20) had no bone changes associated with tuberculosis. Such patients may be misdiagnosed and therefore mismanaged. Myelography demonstrated a partial block. Surgery allowed confirmation of the diagnosis.

In a series of 175 cases Griffiths *et al*¹⁴ found no instance of interstitial tuberculous disease of the dura, and concluded that in Pott’s paraplegia tuberculous pachymeningitis is not an important factor in the causation of the paralysis. The tuberculous granulation tissue around the dura may contract and cause, by this cicatrising mechanism, constriction of the dural tube

and its contents. This ‘peridural fibrosis’ is an important surgical complication in Pott’s disease; in the early cases the constriction of the cord can be relieved by stripping off the granulation tissue. In very chronic cases Hodgson *et al*¹⁵ and Martin¹² described a thick transverse ridge of fibrous tissue, pressing anteriorly on the dura and causing indentation or even acute angulation of the spinal cord with localized ischaemia. In the present study, the cause of compression in five patients (cervical level: four, lumbar: one) appeared to be an osseous focus resulting in exuberant granulation tissue with little pus (10, 12, 13, 15 and 17). Neural decompression and mechanical stabilisation provided the maximum potential for neurological recovery. Anterior decompression was performed without complication. All five patients achieved improved cord function. Beside compression, we should stress the almost constant co-existence of concomitant vascular troubles, which influence the neurological prognosis.^{6,8} Therefore, we believe, in agreement with Wright,¹⁶ Ferro *et al*¹⁷ and Acikgöz,³ that in patients with paraplegia of acute onset, decompression must be carried out promptly. In our experience surgery was also effective in these patients.⁷

In this study, the spinal cord was compressed from its anterior aspect, but sometimes from its posterior aspect (Table 5). Moreover, progressive narrowing of the spinal canal from degenerative disease, loss of flexibility of the spine and inelasticity of the prevertebral fascia are other reasons for the higher incidence of Pott’s paraplegia or tetraplegia with increasing age.¹⁸

The neurological involvement in our series was generally of gradual onset with bilateral symmetrical involvement of the limbs. This is in contrast to metastatic disease in the elderly where the onset of paraplegia is often acute and the neurological involvement asymmetrical. The clinical evaluation of the therapeutic approaches to tuberculosis of the spine is difficult. Although it is difficult to measure neurological function, measurement is necessary for the evaluation and prediction of outcome. The scale presented by Pattison² for the evaluation of long tract function is objective and assesses a range of functional capacity. This allows better evaluation of function and bridges the gap between functional levels of useless motor function and walking and between walking with difficulty and normal walking by adding a grade between each of these. If all of the articles on tuberculosis of the spine followed a similar uniform scale, then we will be able to evaluate results from institution to institution and will be better able to tell which innovations really help outcome and which are unimportant regarding outcome. Sensory examination is much more difficult to assess quantitatively. Therefore, the only sensory evaluation in this grading system could be the measurement of its presence or absence. Urological impairment also parallels motor impairment and appears to be prognostically significant only in differentiating between grade 4 and grade 5 patients. Nevertheless, apparent grade 5 patients need careful neurological evaluation with attention to details, such

as sacral sparing, scrotal and rectal sensation, and any patchy sensation between the levels of compression.

It is possible that the neurological improvement after operation was coincidental rather than secondary to the surgical treatment. This, however, does not appear to be likely. Considering the causes of the compression in our patients (Table 5), and the ineffectiveness of medical treatment above, we may reasonably presume that the improved function shortly after surgical decompression/stabilisation indicates a causal relationship. We had no instance of postoperative neurological worsening or of progressive kyphosis.

The best treatment for Pott's paraplegia is prevention by early detection and treatment of tuberculous spondylitis. Once tetraplegia or paraplegia begins, antituberculous chemotherapy is the mainstay of treatment. However the care of such patients requires time and there is high financial cost. On the other hand, traditional management strategies are now largely irrelevant because of the development of multiple-drug-resistant strains of tubercle bacilli,^{5,10,19,20} infection in large numbers of immunocompromised hosts, and the development of newer and more effective spinal reconstruction techniques. That is why, for eight years, we used the 'short regimen' consisting of the early administration of rifampin, isoniazid, ethambutol and pyrazinamide for 6 months, according to the World Health Organization recommendations. This regimen appears to be effective in our setting, even after spinal surgery for Pott's paraplegia, as is suggested by our present results. In our experience medical treatment alone appears to be indicated when the main cause of cord compression is abscess-linked, without significant bony lesions. In other clinical circumstances (bone-linked, arachnoiditis, or mixed factors) a medicosurgical approach is preferred. Elsewhere, the value of magnetic resonance imaging studies to plan therapeutic strategy and to assess the differential diagnosis is now admitted.²¹ Our surgical approach for tuberculosis of the spine has been recently described.^{6,7} Anterior or posterolateral decompressions are best performed conjointly with an orthopaedic surgeon so that he can judge the degree of instability of the spine and when necessary proceed with a fusion once the sequestered and overgrown bone and granulation tissue have been resected to restore patency of the spinal canal. If we were confronted by a significant kyphosis (more than 40 degrees), costotransversectomy would appear to be a reasonable procedure in the lower thoracic region. By contrast, for upper and midthoracic lesions, we would prefer solely medical treatment, the thoracotomy being invasive.^{3,7}

Conclusions

In this study, the main causes of spinal cord or cauda equina compression appear to be bone-linked (50%), or abscess-linked (36.3%). However, they were generally complex. The best treatment for Pott's paraplegia is prevention by early detection and the treatment of tuberculous spondylitis. Once tetraplegia or paraplegia

begins, antituberculous chemotherapy is the mainstay of treatment. However, where operative facilities are available, early surgical decompression/stabilisation provides the maximum potential for neurological recovery. Although the possibility exists that postoperative improvement was fortuitous, the onset of objective improvement shortly after surgical restoration of more normal relationships between neural and bony elements suggests a causal effect.

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