POTT'S PARAPLEgia OF LATE ONSET

THE CAUSE OF COMPRESSION AND RESULTS AFTER ANTERIOR DECOMPRESSION

LOUIS C. S. HSU, C. L. CHENG, JOHN C. Y. LEONG

From the University of Hong Kong

Twenty-two patients with late onset Pott's paraplegia presenting at a mean of 18 years after initial symptoms were reviewed an average of seven years after treatment by anterior decompression and fusion. Fourteen patients had active disease, and in 12 of these, activity at the internal kyphus was the direct cause of the paraplegia. In the other two, a soft healing bony ridge was the cause. The eight patients with healed disease had hard bony ridges compressing the cord.

The response to anterior decompression was faster, better and safer in patients with active disease: nine recovered completely and three significantly. In patients with healed disease, the anterior decompression was technically more difficult and the recovery less satisfactory. Significant complications included two cases with neurological deterioration, two with cerebrospinal fluid fistulae and four with neurapraxia of the cord.

Paraplegia is a well known complication of tuberculosis of the spine. It usually occurs in the early active phase of the disease and, in areas where the disease is endemic, its overall incidence can be 20 to 30%. Many authors have classified paraplegia in Pott's disease. Sorrel and Sorrel-Dejerine (1924), using the time of onset as a guide, classified this type of paraplegia into early onset, the most common type, and late onset, presenting some years after the apparent quiescence of active disease. Sorrel's late onset paraplegia was described by Seddon (1935) as his Type III paraplegia in which the pathogenesis was uncertain and the prognosis doubtful.

We attempt to define the cause of compression in late onset Pott's paraplegia and to record the response to anterior decompression.

PATIENTS AND METHODS

From 1969 to 1985, 22 patients with late onset Pott's paraplegia were seen at the Department of Orthopaedic Surgery, University of Hong Kong. All were reviewed. These patients had first developed tuberculosis at about eight years of age (range two to 24 years) and had either remained symptom-free or had ignored their symptoms for an average of 18 years (range five to 33 years), before presenting with evidence of pressure on the spinal cord. Of the 22 patients, only three had previously received a complete course of anti-tuberculous drugs.

Nineteen of the 22 had disease which involved only the thoracic spine, two had thoracic and lumbar disease, while one had involvement of the cervical spine. With the exception of the patient with cervical disease, all patients had severe disease (see Figs 1 and 4). The number of vertebrae involved averaged 6.3 (range four to 10). The kyphosis averaged 121°, ranging from 80° to 160°. The patient with cervical disease had two vertebral bodies involved and a kyphosis of 26°.

The age at presentation ranged from 12 to 38 years with an average of 26. There were 15 males and seven females. None of the patients had any previous history of a discharging sinus.

On presentation, three patients were totally paralysed (complete paraplegia), four had some muscle power but were unable to walk unaided (severe), and nine had objective weakness but could still walk (moderate). Four had subjective weakness with only upper motor neurone signs in the legs (minimal paraparesis) (Table 1). Sensory changes were of less importance and therefore have not been included in the classification.

Investigations to identify the level and nature of compression included tomography and myelography, or a combination of the two; and in two recent patients, CT scans.

All patients were treated by surgical decompression via an anterior approach. Nine also had an anterior spinal fusion at the time of decompression; the other 13, as well as the decompression, also had correction of the kyphosis by halo-pelvic distraction with staged anterior

L. C. S. Hsu, FRCS Ed, FACS, Reader
C. L. Cheng, FRCS(Glasg), FRACS, Senior Medical Officer
J. C. Y. Leong, FRCS, FRCS Ed, Professor and Head of Department
Department of Orthopaedic Surgery, University of Hong Kong, Queen Mary Hospital, Pokfulam Road, Hong Kong.

Correspondence should be sent to Dr L. C. S. Hsu

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An 18-year-old girl with moderate late onset Pott's paraplegia. Figure 1 – Tomogram showing severe kyphosis, active disease and large sequestra. Figure 2 – After anterior decompression, correction of the kyphosis by halo-pelvic traction, and anterior fusion with rib grafts. Anti-tuberculous drugs were given for 18 months. Her paraplegia recovered fully; the rib grafts became incorporated. Figure 3 – Follow-up at 10 years showed consolidation of the graft but some loss of correction.

A 23-year-old woman who had been confined to bed by severe late onset Pott's paraplegia. Figure 4 – The spinal involvement was from T10 to L5 and kyphosis was extreme. Figure 5 – At anterior decompression and fusion a large sequestrum was found to be responsible for cord compression. After operation, she recovered well and could walk normally at three months. Anti-tuberculous drugs were continued for 12 months. Figure 6 – Follow-up at nine years, showing healing of the disease with solid anterior fusion. The patient had remained asymptomatic.

and posterior spinal osteotomies and fusion (Figs 1 to 6). None of these later procedures, with one possible exception, changed the outcome of the anterior decompression. The possible exception was in a patient who developed severe neurapraxia of the cord following posterior spinal osteotomy. An earlier anterior decompression had significantly improved her paralysis. She subsequently recovered to a minimal level of paralysis.

Anterior decompression was performed via a left thoracotomy through a rib bed. Findings at operation were recorded in detail in terms of activity of disease, levels of active involvement, and the presence of pus, an abscess cavity, granulation tissue, and sequestra. The internal kyphus was then exposed and excised, and the exact cause of the compression identified.

In general, the excision was technically simpler in patients with active disease, as the abscess cavity often led to the front of the spinal cord, and tissue at the internal kyphus was softer, allowing simple, atraumatic removal. In healed disease the cord was first exposed laterally through an intervertebral foramen by tracing a segmental nerve root. The bone in front of the cord was then carefully excised. At least 2.5 cm of the anterior
dura was exposed and thick fibrous tissue in front of the cord (pachymeningitis externe) was removed as far as possible. In five of the 22 patients, the dura was seen to be pulsating at the end of the decompression. All five patients had soft-tissue compression of the cord.

Spinal fusion was performed in all cases, either at the time of the decompression or, in cases having staged procedures, after satisfactory correction of the deformity by halo-pelvic traction. Rib grafts were usually used and inserted into bleeding cancellous bone in the upper and lower limbs of the kyphosis.

All patients with active disease received a full course of anti-tuberculous drugs for nine to 15 months. Streptomycin, isoniazid and para-aminosalicylic acid were used in 13 patients. Rifampicin was substituted for para-aminosalicylic acid in one patient, while in five patients pyrazinamide and ethanbutox were added because sensitivity tests showed resistance to the first-line drugs. Patients with healed disease received antituberculous drugs for two to three months as prophylaxis against recurrence.

All patients wore either a plaster body jacket or a halo-pelvic distraction device for at least six months after their last operation. Follow-up averaged seven years (range two to 14 years).

RESULTS

Complications. Significant complications from the decompression included two cases of cerebrospinal fluid fistulae caused by damage to the dura. These closed spontaneously after draining for about four weeks. Our previous experience with this complication has shown that attempts at closing such fistulae are usually unsuccessful.

In two patients, moderate paraplegia became worse after decompression and did not recover. One of these had active and the other healed disease, and they deteriorated to severe and complete levels respectively. The probable cause was mechanical injury to a spinal cord that was already under tension. In four other patients, three of them with healed disease, mechanical injury caused neurapraxia of the cord. The varying degrees of increased paralysis all recovered and further improvement then followed.

Cause of compression. The operative findings are summarised in Table II. Of the 22 patients reviewed, eight had disease that was completely healed while in 14, activity was still present. In 12 of the 14 patients with active disease, this was at the apex of the internal kyphus. In the other two, a soft bony ridge had developed over the apex of the internal kyphus, giving rise to cord compression, but disease activity was present only in the upper and lower limbs of the kyphus beyond the apex. In all eight healed cases, a similar bony ridge was the cause of compression.

In patients with active disease, pressure from pus or caseous material was felt to be responsible for compression in six. In five of these, the pus was of the classical thick caseous type while in one, it was more fluid. The latter patient presented with fairly acute pain over the kyphus before the onset of paraplegia, suggesting that there had been reactivation of the disease. In four patients sequestra larger than 5 mm were indenting the cord and in two the cause of the compression was probably a combination of pus and sequestra.

Relationship to activity of disease. Table I summarises the severity of the paraplegia at presentation. Patients with active disease had more serious involvement – six of the 14 were unable to walk. In healed disease, this applied to only one of the eight cases. The duration of paraplegia prior to presentation averaged 1.8 months in patients

<table>
<thead>
<tr>
<th>Paraplegia</th>
<th>Active disease</th>
<th>Healed disease</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-op</td>
<td>Post-op</td>
<td>Pre-op</td>
</tr>
<tr>
<td>Complete</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Severe</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Moderate</td>
<td>6</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Minimal</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>8</td>
<td>14</td>
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Table II. Operative findings in 22 patients

<table>
<thead>
<tr>
<th></th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete healing</td>
<td>8</td>
</tr>
<tr>
<td>Active disease</td>
<td>14</td>
</tr>
<tr>
<td>Pus or caseous material</td>
<td>10</td>
</tr>
<tr>
<td>Granulation tissue</td>
<td>7</td>
</tr>
<tr>
<td>Sequestra &gt;5 mm</td>
<td>6</td>
</tr>
<tr>
<td>Healed bony ridge</td>
<td>2</td>
</tr>
<tr>
<td>Dural fibrosis</td>
<td>4</td>
</tr>
</tbody>
</table>
Table III. Degree of recovery after decompression in 22 patients

<table>
<thead>
<tr>
<th></th>
<th>Active disease</th>
<th>Healed disease</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total recovery</td>
<td>9</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>Partial recovery</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Unchanged</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

with active disease (range one to three months) and 9.2 months in healed disease (range six to 18 months).

Recovery after anterior decompression. Levels of paraplegia after anterior decompression are given in Table I. Of the 22 patients, 12 patients recovered fully, four partially, four remained unchanged and two deteriorated (Table III). Decompression was more rewarding in active disease. In this category all six patients who had not been able to walk at the time of presentation recovered normal walking ability. Of the six with moderate paraplegia, four became better, one remained unchanged, and one worsened slightly to become an aided walker. The two patients with minimal involvement recovered completely. The recovery period in patients with active disease averaged 6.8 months (range two to 12 months).

Of those with healed disease, one patient with severe paraplegia recovered completely, but one with moderate signs became totally paralysed. Two other cases of moderate paraplegia were unchanged and two improved slightly. The two minimal cases had total recovery. The recovery time in healed disease was very variable but in the patient with severe paralysis full recovery took 24 months.

DISCUSSION

In Pott's disease, paraplegia usually occurs in the early active phase. This early onset type (Sorrel and Sorrel-Dejerine 1924) is well documented and was described by Hodgson and Yau (1967) as paraplegia of active disease. The late onset type of paraplegia is uncommon. Seddon (1935), describing this variety (his Type III paraplegia) stated that the patient had usually had fairly extensive spinal carries early in life but made an apparent recovery and remained symptomless apart from increasing kyphosis. Then, after a long period of "anything from four to 40 years", paraplegia set in, usually incomplete but sometimes severe. Seddon was uncertain whether this type of paraplegia was caused by reactivation or by a healing bony ridge.

All our patients fit Seddon's description. The operation for anterior decompression gave us the opportunity to study the cause of cord compression. We found that compression could be related to active or healed disease or to a combination of both. In 12 of our 22 patients, the cause was directly related to the infective process; pus, caseous material and sequestra were found causing compression in front of the cord. In one of these 12, the finding of fluid rather than caseous pus suggested reactivation rather than persistent chronic infection.

In our other 10 patients compression was caused by a bony ridge at the internal kyphus. In two of the 10, although a soft bony ridge suggested healing, disease was still active beyond the apex. In the other eight, compression was by a hard bony ridge and the disease had healed completely. This type of paraplegia in healed disease was well described by Hodgson and Yau (1967), and an essential factor in pathogenesis is the increasing kyphosis which gradually squeezes the healing bone backwards on to the cord.

The pathogenesis of the compression in paraplegia of late onset is seen to be variable, but most often it is secondary to persistent low-grade infection with increasing kyphosis. In disease of long standing, reactivation and healing may occur simultaneously at various levels of the spine and one may see compression by a healing bony ridge in the presence of active infection elsewhere. Reactivation of quiescent disease is another possible cause, seen in one case in our series.

In Pott's paraplegia, compression is almost always from the front and decompression should therefore be from the anterior aspect, approached by a thoracotomy or a costo-transversectomy. The view that laminectomy is contra-indicated for Pott's paraplegia was well expressed by Griffiths in 1979. Our results show that when the compression is related to active disease, the prognosis is much better, the response to decompression being faster and more complete than if the compression was due to a bony ridge.

The Medical Research Council's extensive trial on treatment of tuberculosis of the spine (1978), considering relatively early disease, concluded that Pott's paraplegia from active disease could be managed conservatively by anti-tuberculous drugs alone. This trial, however, dealt mainly with limited disease with little kyphotic deformity. The compression in these cases is usually from increasing tension in the abscess and oedema of its wall. Drugs alone can arrest the pathological process, reduce tension and oedema and relieve the compression. The response to conservative management is slow and, if the compression is by a large sequestrum or a soft healing bony ridge, its efficiency is doubtful. Surgical decompression seems preferable, the selection of treatment being helped by detailed radiological studies of the internal kyphus.

The pathogenesis of cord compression in late disease is quite different; involvement is usually extensive and the kyphosis is severe and invariably progressive. Compression is usually caused by thick fibro-caseous pus or large sequestra rather than by tension in the abscess or oedema of its wall. Anti-tuberculous drugs alone will fail to relieve compression because increasing kyphosis will tend to squeeze even a healing abscess backwards and eventually flatten the cord.
We believe that late onset Pott's paraplegia from active disease should be relieved by anterior decompression (and fusion). This gave us rewarding results despite the hazardous surgery resulting, in a few cases, in deterioration of cord function and other serious complications. Patients commonly have severe lung disease and adequate anaesthetic support is mandatory.

In paraplegia from healing disease, our results were not as rewarding. We feel that a distinction should be attempted between the soft compression of early healing and the hard bony ridge of well healed disease. Our two patients with soft compression had good results, whereas only one of the eight with a hard bony ridge improved significantly, and three of them had neurapraxia of the cord. In view of this, we feel that patients with hard ridge compression and only mild or moderate paraplegia should perhaps be treated by stabilisation only, to prevent further progression of the paraplegia, and that decompression should be reserved for those with severe paralysis.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

REFERENCES


