Cervical spondylotic myelopathy

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Cervical spondylotic myelopathy is a condition in which there is progressive degeneration of the intervertebral joints of the cervical spine. It is widespread throughout the adult population such that it has been estimated that on the basis of plain X-rays 50% over the age of 50 years and 75% over the age of 65 suffer from the condition. It is indeed fortunate that only a small proportion suffer from the most serious complication of Cervical Spondylotic myelopathy (CSM); however when this occurs it can lead to devastating and crippling neurological deficit. A discussion needs to evaluate 3 main areas: aetiology and pathogenesis, natural history and treatment. In order to discuss and illustrate these and other points I will be using material from my own series of 170 consecutive patients with CSM, and course comparing this with data from other major series.

Aetiology and pathogenesis

The aetiology of cervical spondylotic myelopathy is poorly understood in the majority of cases. There are some specific agents which lead to early spondylotic such as trauma either a single major episode or minor repetitive episodes, and arthritis either chrystalline or rheumatoid.

The pathology seems to start in the central disc joints and in the apophyseal joints. The central disc tends to dessication as the patient ages. This narrows the disc space and leads to reduction in movement and loss of alignment which in turn leads to abnormal strains on other joints. Ligamentous hypertrophy occurs followed by osteophyte formation. The spinal cord and/or spinal nerve roots become compressed as do the reinforcing radicular arteries from the vertebral artery which itself can occasionally become involved. The posterior articulation can also involved in particular the ligamentum flavum. Once started the process gathers pace though at varying speeds for differing patients.

There are three main theories as to the cause of myelopathy: spinal cord compression, ischaemia and spinal cord and nerve root traction. Occlusion of the anterior spinal artery is rare. If the reinforcing arteries are to be invoked one might expect many more cases of nerve root compression to rapidly proceed to a myelopathy, which is not the case; this cause is unlikely. Traction of the spinal cord against osteophytes can be incriminated and in support there is the evidence from some workers that deterioration is more common in cases with more mobile necks; however if this were the case then one would expect wearing a firm cervical collar to benefit many patients which is in fact not the case. There is much more evidence in favour of compression. Nurick found that the constitutional and acquired sagittal diameters of the cervical canal are narrower in patients with CSM. Compression can be demonstrated by myelography or MRI, being anterior in 75%, posterior in 21% and both anterior/posterior in 4%. Such patients are very sensitive to trauma and the myelopathy may advance more rapidly in some 15% patients who already have myelopathy and then suffer an injury. Finally there is the evidence which I will show later that the vast majority of patients improve following decompressive surgery. It would seem the most likely explanation is compression provided one allows that in any compression there must be an element of squashing of the vascular bed at the site of compression as well as distortion/compression of neural tissues.

Natural history of CSM

The natural history of CSM is variable; it may be slow and insidious, progressive and unrelenting or intermittent and associated with remissions. To some extent the natural history in differing series is dependent on the pattern of referral and the expectations of patients and referring physicians. Although more common in the elderly one must point out that in my series many patients presented with severe myelopathy in the
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ages 45-55 years. 50% of patients had suffered from other major illnesses such as diabetes mellitus, myocardial ischaemia or infarction and the like, thus suggesting that their physiological age was more than their chronological age. The mean duration of symptoms varies in differing series between 1.2 years and 2.3 years; however the mean obscures the fact that although in my series the mean duration of symptoms was 1.69 years 50% patients experienced their symptoms for less than 1 years before coming to treatment. Trauma played a part; 30% has an injury to the cervical spine in the past and 27% had an injury which accelerated their neurological decline.

Neurological deficit of CSM

Patients who present with neurological symptoms of CSM do not often complain of much pain in the neck, perhaps because the cervical spine is becoming rigid and most pain seems to come from movement. Undoubtedly some patients present with both myelopathy and wit radiculopathy, but the majority who present with myelopathy have a myelopathy as the major problem.

The myelopathy can take one of 4 main forms:
1. Ataxia of gait with loss of fine function of hands 34%
2. Ataxia of gait with spasticity of legs and weakness of hands 38%
3. Partial Brown-Sequards syndrome 6%
4. Tetraparesis both motor and sensory 22%

Many of the patients give good descriptions of how they are disabled and some are very disabled before they present for treatment. A simple functional grading system has been used for both pre and post-operative assessment:
1. Totally disabled dependent on others for daily living.
2. Disabled living at home but capable eating and personal hygiene.
3. Capable of light employment or light household duties.
4. Capable of full employment and sport or heavy household duties.

In my series 50% patients were in Grade 1 and 50% in Grade 2 pre-operatively.

Diagnosis and investigations

In the first instance one investigates a cervical myelopathy, to exclude other pathological processes such as a neoplasm and then to confirm a compressive CSM; it is vital to manage patients this way since Campbell & Phillips found that 17% of patients presumed to have CSM were subsequently shown to have another disease process. Plain X-rays of the cervical spine are useful to assess the degree of spondylosis and need to be taken in extension and flexion to demonstrate any possible subluxation. They may also suggest other pathologies such as metastases. For those neurosurgeons with MRI available this is the next investigation. However for many only myelography is available and this is still an excellent investigation with the only drawback being that it is invasive. I have not had MRI available until very recently and all the patients have had myelography with the following results:

<table>
<thead>
<tr>
<th>Anterior indentation</th>
<th>59%</th>
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<tbody>
<tr>
<td>Posterior indentation</td>
<td>4%</td>
</tr>
<tr>
<td>Partial block in one position</td>
<td>26%</td>
</tr>
<tr>
<td>Complete block in any position</td>
<td>11%</td>
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</tbody>
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Treatment

Cervical spondylosis was first described as a clinical problem in the 19th century (Wilkinson), and gradually it was recognized that it could lead to myelopathy, but the picture was clouded by similar findings occurring in some patients with motor neuron disease (Amyotrophic lateral sclerosis). The first neurosurgical operations involved decompressive cervical laminectomy which produced variable results, and overall these were only slightly better than those for non-operative treatment; though it did seem that withing the operative group were some patients who were definitely improved. In 1958 the anterior approach was first described simultaneously by Cloward and by Smith & Robinson. The principles were similar, namely decompression of the cervical spinal cord followed by fusion with a free bone graft. Philips (1973) showed that the anterior approach produced much better result than laminectomy or a cervical collar. But not all patients were suitable for the anterior approach and in 1975 I started on a prospective trial which continues and the results of which I present to you.

The over-riding aim has been to free the spinal cord from compression. The specific indications for the approach have been:

Cloward's operation.

To remove any anterior protrusion extending 4 mm
or more beyond the level of the vertebral body, even if this meant multiple level operations. Any protrusion 3 mm or less was left alone.

Cervical laminectomy

a. Patients with multiple anterior protrusions of 3 mm less and with narrowing of the theco-peritoneal diameter to 12 mm or less.
b. Patients with no anterior indentation but multiple posterior indentations and a narrow canal.

Using this protocol 137 patients have undergone Cloward’s operation and 33 decompressive laminectomy. In the Cloward’s group 72% had a single level operation, 26% 2 levels and 2% 3 levels. The pathology of the compressing lesion was recorded with the result that 40% had osteophytes as the main compressing lesion, 22% had discs and 38% had both disc and osteophytes. The surprising fact is that so many patients had disc material causing compression even though the preoperative plain X-rays showed osteophytes.

The technique used was essentially that described by Cloward with a few modifications:
1. Skull traction, using the Garner-Wells tong, was used and this obviated the need for disc space spreaders.
2. The drill was set to 20 mm depth in men and 18 mm in women rather than measuring off the X-rays which is inaccurate; this avoids the disaster of drilling into the spinal cord.
3. Osteophytes were chipped away with small curettes; the high-speed air-drill was not used since it was felt that the heat generated would conduct through onto the already diseased spinal cord.

The laminectomy group all had a laminectomy from C3 to C7 inclusive in order to allow the spinal cord to gently bow backwards rather than angulate acutely as can occur with a small laminectomy of only a few levels. However it must be pointed out that some neurosurgeons favour a short segment laminectomy.

Complications

There were no post-operative deaths and 141 patients suffered no post-operative complications. Of the 15 (9%) patients who suffered complications the more common problems were:

- Painful shoulders 2
- Worse temporarily post-op 2
- Donor site infection 2
- Wound infection neck 1

Electrolyte problems 2
Oedeme legs 2
Other complication occurred singularly and included psychosis, tear of vertebral artery, bleeding peptic ulcer and bronchopneumonia.

Il patients required further operations on their cervical spine for recurrence of symptoms, 7 who had previously undergone a Cloward’s operation needed a laminectomy and 4 required a second Cloward’s operation at another level. All patients recovered well though 2 patients did not make as full a recovery the second time.

Results

Patients were assessed at 6 weeks, 4 months and 8 months and thereafter as necessary. The same grading system was used as mentioned above. Although patients started to improve immediately they continued to improve for another 6-8 months.

<table>
<thead>
<tr>
<th>Grade 1</th>
<th>4 months</th>
<th>8 months</th>
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<tbody>
<tr>
<td>Grade 2</td>
<td>44</td>
<td>22</td>
</tr>
<tr>
<td>Grade 3</td>
<td>112</td>
<td>70</td>
</tr>
<tr>
<td>Grade 4</td>
<td>13</td>
<td>78</td>
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If one takes Grade 3 or 4 as success then overall 87% did very well and 13% were left still disabled though slightly improved. Various factors were assessed for their possible affect on outcome; the only one that had a statistically significant (0.005) effect was the pre-operative function. Those that had no statistical effect included age, duration of symptoms, pre-operative neurological syndrome and the myelographic features. However one must also point out that 9 (5%) patients, although improving greatly in function, continued to suffer pain, which was of a burning nature, in their hands and which was very difficult to treat although carbamazepine helped a little.

Results reported in the literature vary greatly. Phillips showed that out of 102 patients 73% improved and 58% returned to work. Lunsford & al found that of 32 patients only 50% improved and 50% were unchanged or worse; however one must point out that in this series the operations were performed by 8 different surgeons of varying experience. Though it may seem presumptuous to state there should be no reason why results comparable to this series and that of Phillips cannot be achieved provided the right operation is chosen and scrupulous attention is given to operative technique.
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The question of whether or not to insert a dowel graft is difficult to answer; some neurosurgeons favour this, as I do, and some do not. Certainly the curvature of the cervical spine can be better aligned with a dowel and this may have an advantageous effect on other disc space but only very long term follow-up will give a valid answer.

Conclusion

It would seem for the foreseeable future patients will develop CSM and it will be important for at least one neurosurgeon in a department to develop expertise in the management and surgical treatment of these patients. In Europe with an increasing number of ageing patients, as life expectancy lengthens, more patients with this problem will present with CSM. In this day and age we should be treating such patients earlier before they are profoundly disabled.

References and some suggested reading


