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Cervical spinal stenosis

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The cervical spinal canal bears the shape of a triangular (or more accurately, pentagonal) tube, reducing in size from C1 (av. sagittal diameter 20-25 mm) to C4 (av. 17-18 mm); after this upto C7, its dimension remains more or less unaltered. All along the interpedicular diameter is roughly twice the sagittal diameter. The spinal cord is a round structure nearly 10 mm in diameter. The additional contents in the spinal canal include some fat, the ligaments, dura and the CSF in the subarachnoid space.

The basic sagittal diameter of the spinal canal is fundamental to the ease with which the cord can lie within it. Payne and Spillane [1] reported that cervical myelopathy was more likely to occur when the sagittal diameter was smaller than the average. Hinck et al [2] designated such a condition as developmental narrowing of the spinal canal. several other series feature in the literature, which have aimed at measuring the variations of the canal diameters of the entire cervical spine in normal adults, and, diameters as low as 12 mm have often been cited in normal subjects [1], [3], [4], [5]. Considering that the cord is 10 mm in diameter, this leaves just 2 mm of space for all the extramedullary structures in such canals. A canal less than 12 mm should therefore be definitely considered as developmentally narrow, and a canal diameter under 10 mm would be clearly inconceivable. Barring congenital high cervical anomalies at or near the cranio-vertebral junction it is hard to imagine that anyone could be born with such a stenosed canal, and if that were true there would be myelopathy right from birth and not during adulthood as is normally seen. Pure stenosis is relatively infrequent as a cause of neurologic disorders in itself but is more important as a substrate in the event of other disturbances [6], [7]. The connotation derived is that some people do have a spinal canal which is less wide than what it normally is in the vast majority of individuals - a rather tight fit for the cord which can still remain normal, albeit extremely vulnerable to compromise by acquired factors that add up to the narrowing and consequently compress it, comparable to the colloquial last straw that broke the camel's back. Hence, logically, wider the canal, greater is the tolerance to intraspinal space occupying factors, and narrower the canal, more liable is the pathologic disturbance.

Factors which cause further spinal canal narrowing may be osseus (Osteophyte, ridges or spurs, and sometimes inwardly directed facets) and/or nonosseus (disc herniation or bulge, yellow ligament hypertrophy, or thickening of posterior longitudinal ligament which may occasionally even ossify (OPLL)). Disc disease still remains the prime factor causing intrusion into the spinal canal. Sometimes in a very narrow canal the osteophytic projection is barely a mm or two. That itself may be enough to cause critical narrowing; the presence of more than one such osteophyte seem to exert a cumulative effect on the production of cord compression. This finding has relevance to our operative management. Movements of the neck also have a role to play: in flexion the cord becomes thin and elongated but in extension it becomes short and bulky. The yellow ligaments go into folds and encroach further into the canal. Thus extension is more critical than flexion in patients with canal stenosis. Such 'normal' movements, not infrequently even recommended as a neck exercise, occurring frequently and day after day, are liable to inflict repetitive microtraumas to the spinal cord. Occasionally, abnormal movements of the cervical spine itself may cause injury. The occurrence of myelopathy is thus related to the static factor of mechanical pressure, and as well as the dynamic factor.

Clinical presentation

The main features are those of myelopathy; only occasionally there is associated radiculopathy. Paraesthesia in such patients should not be regarded as symptoms of radiculopathy, but rather a result of cord compression. Similarly, atrophy of hands is again not the result of a peripheral problem but of anterior horn cell fall-out. Myelopathy predominantly presents with spasticity and ataxic gait. The symptoms may progress with reasonable rapidity, or may be so slow that they may be mistaken for other spinal cord ailments(non-compressive myelopathy). Occasionally the symptoms may have a march similar to an intradural tumor involving one upper limb, then the lower limb on that side followed by the opposite side, and finally the other upper limb.

Clinical signs are those of cord compression. This may be territorial due to anterior spinal artery compression, or Brown-Squard-like syndrome. Rarely, following trauma, there may be a central cord syndrome or a picture of total cord transection. Most of these patients have atleast one major osteophyte at the appropriate level in addition to canal stenosis. Pyramidal signs abound. Sensory involvement is less marked compared to lumbar canal stenosis or cervical spondylotic radiculopathy. Posterior column impairment may frequently occur, especially in the lower limbs. Muscle atrophy in upper limbs has already been alluded to.

Material

We encountered 16 cases over a period of 5 years, which accounted for 20% of all patients presenting with cervical radiculomyelopathy. The age of these 16 patients ranged from 18 to 62 years. All had features of myelopathy; half of them (8 patients) had wasting of upper limbs, and as many as seven had difficulty in micturation. Posterior column disturbances were noted in 14 of them. Overall, our observation was that symptoms appeared later than signs and clinical signs were often out of proportion to the symptoms. Some patients appeared to be in no great distress e.g. they could tolerate a mildy spastic gait but reported only when the hands got involved.

Radiological examination

Canal stenosis may occur at just one level, and that may be anywhere from C2 to C7, or at multiple levels, more frequently in the lower half of the cervical spine. In addition to the papers mentioned earlier Epstein [7], Kessler [9] and Stratford [10] have investigated the relationship between the clinical findings and radiographic examinations in detail.

X-rays must be carried out properly and perfectly. The aim is to

(i) rule out other causes of high cervical cord compression such as craniovertebral and other anomalies, and inflammatory and neoplastic processes;
(ii) study the primary spinal canal diameters in the sagittal plane;
(iii) identify other features of degenerative processes in AP, lateral and oblique projections.

It is generally agreed that the canal with a diameter of less than 14 mm put the secondary factors into operation early. When measuring the canal diameters in the dead lateral view the factors in consideration are -

(1) tube patient-film distance (conventionally 1.83 m) and
(2) points of reference for measurement of the canal:

According to Boijsen's method, the points of reference are the mid-line of the posterior surface of vertebral body and the nearest point on ventral line of the cortex at the junction of the laminae and the spinous process. Wolf describes joining (a) the posterior border of vertebral bodies and the ventral line of the cortices of the laminar fusion, and measuring the shortest distance between these 2 lines along the entire canal. This method seems to provide a more realistic measure of canal dimension at its narrowest. Of course, these are diameters of the osseus canal, and non-osseus factors can only be studied at watersoluble myelography and (or combined with) CT scan. The latter provides a different dimension and outlines finer details such as pedicular and laminar hypertrophy, thickness of ligaments and direction of osteophytes and spurs. The round shape in the dorsal surface of the vertebral body, shortening of the pedicle which encroaches right on to the body (converting the pentagon into a triangle) and widening of the angle between the laminae results in the canal assuming the shape of a semi-ellipse. The round cord thus flattens anteroposterially, increasing slightly in its transverse diameter. Myelography may show posterior osteophyte indenting the anterior dye column, and occasionally folds or thickness of ligamentum flavum posteriorly (especially when extension and flexion views are compared). The oft noted finding which needs re-emphasis is the expansion of the cord shadow and consequent thinning and widening of the dye columns laterally in the AP view, simulating an intramedullary SOL. This has been noted over one or two levels in cervical disc disease and may occur similarly and even over a greater length of the cervical canal in patients with canal stenosis, which is after all a diffuse, extradural cord-compressive pathology. Post-myelography CT scan should be done when tumor is suspected. It has been our uniform experience that osteophytes have invariably formed and projected into the pre-existing stenotic canal, which now becomes symptomatic.

Finally I must mention an interesting plain X-ray observation of my own. In normal adults with a normal size canal the cortex of the line of fusion between the laminae and the spinous process lies several mms, behind the facet joint. In patients with developmental canal stenosis the apophyseal joint tends to lie over or nearly over the line of laminar fusion. This indicates that the angle between laminae has become much more obtuse. This finding can, at a glance and reliably tell you if there is canal stenosis. Agreeably, this does not permit one to ignore measuring the canal diameters on plain X-rays and on CT whenever the latter is done.

Treatment

All patients with cervical canal stenosis and evidence of cord compression require surgery. In our series of 16 patients half of them were subjected to laminectomy which was the preferred route by another colleague. The remaining 8 patients were operated by the anterior approach which I favour. If we believe that developmentally narrow canal remained asymptomatic for the first two or more decades of life and became symptomatic only thereafter, and if radiology shows further encroachments from the front, careful removal of these by the anterior route would restore the canal to its original anatomic dimension and again make the patient asymptomatic. At operation, with microtechnique, and using fluoroscopy a complete disc removal with meticulous osteophytectomy is done. If the posterior

longitudinal liege. is thickened, it is also excised. In one instance we found a trans-ligaments disc extrusion. Bilateral foramenotomy is also done. To ensure that all osteophytes have been taken away a blunt dural hook is passed behind the vertebral body. Its passage would be free if all osteophytes are fully excised. The hook is similarly passed to check the patency of the intervertebral foramina. However, if one is not equipped or experienced with this anterior operation it is perhaps better to do a decompressive laminectomy. The other indications for the latter operation are:

(1) Proven posterior (lig. flavum thickening) compression;
(2) Suspicion of tumor;
(3) Extreme ossification of the discs which would prevent safe and easy entry into the disc space from the front.

The anterior operation should not be restricted for only one or two levels of decompression, but if we believe in the fundamental principle that an anterior encroachment must be removed from the front, then it will have to employed even at 3 or 4 levels. The skin incision for one or two consecutive levels in a horizontal 'necklace' incision between the sternomastoid and the traches, but for 3 or 4 levels prefer an oblique vertical incision running along the interior border of the right sternomastoid.

In addition to the microdiscectomy and osteophytectomy we prefer to interpose bone graft in place of the removed disc. The cartilaginous plate is not removed but the surface of adjacent vertebral bodies is made horizontal. The bone graft obtained from patient's right iliac crest is measured to size. Its height is 2-3 mm more than the empty disc space (averagely 6-7 mm); and at its depth it reaches flush with the posterior most limit of the vertebral bodies. The cortex of the anterior border of the graft would be recessed by 1-2 mm, behind the anterior limit of the vertebrae above and below. The dural hook is again passed behind the graft to ensure that it has not encroached into the spinal canal. In a couple of instances we have also used readily prepared done cement and after covering the posterior and lateral sides of the empty disc space with gel foam, injected the liquid cement (about 1 ml) into the disc space until it sets. This acrylic keeps the disc space patent and new bone grows around it later.

Advantages of an interbody graft

1. Restores the height of the spinal canal.
2. Keeps the intervertebral foramina wide open.
3. Affords natural spinal lordosis.
4. Prevents collapse of the disc space and thus prohibits buckling of flaval ligaments.

The two indispensable tools for this operation are:

1. Fine mastoid curets of cup diameter ranging from 2 to 5 mm, with long round handles.
2 Cloward's interbody spreader which helps to open the disc space during discectomy and osteophytectomy, and also to ovardistract the space to be able to impact a slightly oversize keystone bone graft.

Post-Operative

A cervical collar is given to all patients. For those who underwent laminectomy it is for only a few

days to ameliorate the wound pain; but for those with the anterior operation it is mandatory for them to wear it day and night for 3 months until early bone fusion is ensured. Check x'rays (lateral view only) are taken immediate post-op, again between one and three weeks, and then at three months, six months and one year.

Steriods and antibiotics are given for 5 days.

Recovery

Recovery from myelopathy depends on the duration and extent of compression. Spasticity often improved dramatically and even patients with signs of anterior horm cell involvement recovered reasonably well. Patients with extensive involvement required prolonged physiotherapy.

Conclusion

Cervical canal stenosis implies a developmentally narrow spinal canal which is however generally compatible with normal spinal cord function, atleast until early adulthood. This inherent narrowing renders it highly vulnerable to compressions acquired and originating from neighbouring bone and soft tissue structures. This result in myelopathy of varying grades and affects all four limbs, and sometimes even bladder function.

A careful radiological evaluation is done to measure the spinal canal and the nature and extent of compression factors. Disc disease is the most frequent cause; the osteophytic encroachments need not be very large and are yet capable of causing the critical narrowing. Meticulous disscectomy and osteophytectomy by the anterior route and using microtechnique at the offending levels takes away this critically intruding factor and restores the canal diameter to its pre-symptomatic anatomic dimension. Precisely measured and contoured disc -like bone grafts are inserted in the respective disc spaces for its obvious advantages. Technically and anatomically more superior, the results of this operation have been atleast as good as those after laminectomy and has encouraged us to generally give up the latter for its obvious disadvantages.

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