Article *NIMHANS Journal*

Stray Thoughts on Cervical Spine

Volume: 06 Issue: 2S December 1988 Page: 21-23

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(a)

Ossified posterior longitudinal ligament (OPLL):

OPLL was first described in 1960, by Odda from Japan. I have found that in Hawaii 10 years earlier. In plain X-rays it will be seen as a ridge of bone at the anterior wall of the spinal canal, attached to the back of the body of the vertebrae. Usually it is of 5 - 6 mm in thickness. It narrows the anterio-posterior diameter of the spinal canal. It is often limited to C2 vertebra region. It can cross and grow upto C5. In the CT scan extreme narrowing of the canal can be visualised. The cord will have half moon appearance. About 5000 cases have been reported in Japanese literature. They divide these into two groups namely :

(1) Segmental type;
(2) Mixed type.

It can be a continuous strip which can extend all the way down the spine or be localised type opposite the disc spaces. According to Krandel OPLL may result in various clinical syndromes. They are transverse myelopathy, motor system syndromes, central cord syndrome, Brownsequard syndrome and brachialgia.

Average age of presentation is 51 years in males and 48 years in females. There is no sex predeliction. But it can be seen in all ages especially over 40 years. Location wise C3 - 4 and C5 - 6 are commonly affected. Very thick lesion can produce spinal canal obstructions.

Microscopically calcification starts like a cap, in the center of the vertebral body, in the posterior longitudinal ligament. It is infact ossification as trabeculation can not be demonstrated.

I have followed a patient with OPLL from 1951 to 1986. In this case OPLL started at C4 - 5 and ascended up rapidly. He underwent a laminectomy which did not help him clinically. The antigravity ascent of this calcification is interesting, for which no explanation is available. In some patients there was no progression. One had a similar lesion at T2, who had improved following removal of the lesion anteriorly.

This lesion can be mistaken for a tumour like osteoblastoma or an osteoma. But it covers the disc space and at surgery it is very hard and ivory like. Since Laminectomy was not found to be useful, I started removing the lesion anteriorly. If required discogram used to be done to demonstrate the disc prolapse on either side of the OPLL. Hard bone need to be drilled. Roungers will be used later. Subsequently a

bone graft should be placed. With this the results are satisfactory and significant improvement in myelopathy was noticed.

There are three ligaments in cervical spine that can turn into bone. Anterior long ligament, post long ligament and ligamentum nuchae. The latter is not symptomatic but the other two may be. The calcified anterior long ligament is called intercallary bone. During follow-up it has been observed that it grow and enlarges and gets attached to the vertebral body. Following spinal fusion and stabilization of spine the osteophyte melt away, where as OPLL once starts, it persists producing canal narrowing. Sometimes ossification can start as a tiny bit and progressively enlarge like a tumour. As the anterior long ligament get ossified anteriorly the size of the vertebrae will increase. This is called "growing vertebrae". The exact mechanism of this is not known. I believe that there are congenital foetal rests of cells of ossification in these ligaments which stay dormant until the 4th or 5th decade of life. Later they expand and grow and can attain formidable sizes. Intercallory bone seldom gives rise to symptoms.

(b)

Vertebral artery syndrome:

This is a rare condition. A lateral osteophyte from the joint can compress the vertebral artery and gives rise to the syndrome. This manifests as syncope. In one of the patients who had recurrent syncopal attacks X-rays showed osteophyte at C5-6. Discogram revealed ruptured disc. Angiography demonstrated compression when the neck was extended. This osteophyte can be easily removed by anterior approach. Body is drilled, with gauze and patteys. The artery will be displaced from osteophyte. Then the osteophyte will be roungered so that artery is decompressed.

(c)

Locked facets:

Locking of facets will prevent reduction of a dislocation. Often in such situations laminectomy and removal of the facet joint is the common practice. But this will add to the instability. I have found that this locked facet can be reduced by anterior approach. After removing the disc with skull traction we can pull up the upper vertebrae and get to the facet. Then it can be deduced by pushing the upper vertebra with the thumb into an alignment. If it does not reduce I have an instrument called dislocation reducer which can be used to reduce the locking and dislocation

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(d)

Anterior horn cell involvement in cervical myelopathy:

A 74 year old man presented with wasting and weakness of muscles of shoulder girdle and arm and hand. X-ray showed body of vertebra projecting into the canal. Myelogram showed indentation of the cord. The body was excised and a bone graft was placed. After 3 years all the muscles became normal. Ant. bony fusion was confirmed by X-ray. This is a classical example of reversible myelopathy.

I do three level fusion anteriorly through a single incision. Generally C5-6 disc space is opened. Disc is removed. Before putting bone graft, lower level disc is removed and bone graft is placed. Then the disc one level above will be dealt in the same way and the graft is placed. The graft at the central place will be done at the end.

(e)

Cervical myeloradiculopathy due to spondylosis:

In case cervical spondylosis spinal compression occurred due to disc herniation, narrow canal, hypermobility of space and malalignment. Narrow foramen, soft disc are bony spur in the foramen are required to produce radiculopathy. Thickened ligamentum flavor can also produce radiculopathy.

Narrow canal can be congenital and remain asymptomatic till 3rd or 4th decade. This is not an indication for surgery. Then he gets symptoms due to osteophytes and disc prolapse. The osteophytes can indent the cord and produce myelopathy and anterior hern cell damage. Osteophytes are associated with disc degenerations and the disc space is usually narrow. To get large osteophytes with normal disc space is very rare. If such osteophyte exists with a normal disc space it is called "Osteophytosis".

Angulation of spine following trauma and anterolisthesis and retrolisthesis can produce myelopathy following laminectomy for trauma or other causes. Especially extensive laminectomy results in anterior angulation which can lead to progressive myelopathy. This needs excision of osteophytes and correction of angulation by placing appropriate wedge graft. The canal should be made free for the cord to prevent progressive damage.

I use a special technique to prevent angulation following extensive laminectomy. All the bone pieces removed where made into small pieces by a bone mill. Gelform is placed over the dura and as the gelform all these small bits of bone will be placed. It fuses and forms a rigid piece of bone and prevents angulation.

In 1966 Turnbull a Canadian did an interesting experiment. He fixed 20 cadavers with a cervical spondylosis, ten in flexion and ten in neural and in extension. He fixed the spines in formalin and did microangiographic studies. He found in flexion spinal cord is oval and compressed antero-posteriorly where as in neutral or extension cord was round and nice. In flexion, the spinal arteries which run in the transverse direction, close down their lumen and the vessels running anterioposteriorly had wide open lumens. In cervical myelopathy two things are involved in upper extremities, weakness and wasting of arm hands and shoulder. There are anterior horn cell deficits. Normally blood vessels of anterior horn cells run in transverse direction. The inter commissural blood vessels go upto the anterior commisural and turn laterally and goes horizontally to supply blood to anterior horn cells. Up to pyramidal tract all blood vessels run horizontally from the post spinal artery, so there is an internal pathology in the form of ischemia of tissue due to compression of cord in AP direction. If the pathology persists, the blood vessels might thrombose and the deficits become permanent. But it occurs only in small percentage, if the canal is widened around the cord to make it round, the rocking movement of the cord over the osteophytes is not there, the blood supply is restored, and myelopathy improves.