

MANAGEMENT OF FLUOROTIC MYELOPATHY (PRELIMINARY REPORT)

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Involvement of the nervous system in skeletal fluorosis has been exclusively reported from India. The credit for the earliest description of neurological cases must be given to Short, Pandit and Raghavachari (1937) who described ten such cases from the Nellore district of Madras Province. A few sporadic cases have since been described from different parts of India. Siddiqui (1955) reported 32 neurological cases and Singh, Jolly et al (1963) have reported another 42 cases.

In order to elucidate the cause of the myelopathy we studied the skeleton of a patient who was suffering from fluorosis with quadriplegia and died in the hospital. The changes in the vertebral column were striking and have already been reported by Singh et al. The vertebral bodies from the cervical, thoracic and lumbar regions were larger than normal and showed lipping of the margins. Calcification of various ligaments, particularly the ligamenta flava, intertransverse and interspinous ligaments, had produced large numbers of osteophytes. The anterior and posterior longitudinal ligaments were thickened and calcified, and the membrana tectoria formed a process similar to the dens, lying behind and parallel to it.

The spinal canal was narrowest at the lower end of the third cervical vertebra, where it was semilunar in shape and only 2mm wide, the normal being about 8mm. Below this it gradually expanded until it became reduced again at the junction of first and second thoracic vertebra. There was less narrowing in the lumbar region. The reduction in size of the spinal canal was seen in the antero-posterior measurements only. The transverse measurements were normal except from the seventh cervical to the third thoracic vertebra. The second and third cervical, and second,

third and fourth thoracic vertebrae were fused with one another by calcification in their articular capsules.

Some of the intervertebral foramina were narrowed and sometimes distorted by osteophytes. There was more narrowing antero-posteriorly than vertically.

In short the study of the skeleton leads us to think that the fluorotic myelopathy may be due to compression of the cord.

As far as we know nobody has attempted any treatment for those afflicted with fluorotic myelopathy. We therefore performed the operation of extensive cervical laminectomy rather hesitantly on advanced cases of myelopathy which were thought to be hopeless. Encouraging results have prompted us to present this preliminary report.

CASE REPORTS

Case No. I.H.S. a 43 year old male coming from Bhatinda District (an endemic area for fluorosis in Punjab) noticed stiffness of the back in the early part of 1957. This stiffness progressed gradually so that a year later (1958) he started feeling weakness in all the four limbs, more marked on the left side.

He gradually went downhill and in 1962 (about 6 months before admission) he was unable to move and was bed ridden. He was also unable to feed himself. There was slight constipation but no urinary complaint.

The patient had been consuming water having high proportions of fluorine. The elder brother was a proven case of fluorosis.

CLINICAL EXAMINATION

He was completely paralysed and was lying helplessly in bed. There was no active

movement at any joint though the joints could be moved through a fair range passively. The back was stiff like a board and the patient had to be turned in bed like a log of wood.

NEUROLOGICAL EXAMINATION

LOWER LIMBS. There was complete loss of power in all the muscles. Muscle tone was markedly exaggerated, the knee and ankle jerks were brisk, and patellar and ankle clonus was present. The plantar response was extensor on both sides. The cremasteric and abdominal reflexes were absent.

UPPER LIMBS. Except for slight contraction of the deltoid and biceps there was no power in any muscles. There was wasting of the muscles of the hand and of the extensor muscles in the forearms. The biceps and triceps jerks were brisk and the supinator jerk showed inversion.

The sensations were blunt.

The cranial nerves were normal.

The other systems were normal.

INVESTIGATIONS

Routine investigations and blood chemistry were normal. VDRL & Kahn's test were negative.

The fluorine content of:

the urine was 0.34 mgm%

the blood was 0.67 mgm%

the bone was 180.2 mgmg%

Radiological examination showed grade III advanced changes of fluorosis with osteosclerosis and marked osteophytosis of the spine.

CSF:

Proteins: 168 mgms %

Chlorides: 700 mg %

Sugar: 60 mgm %

Cytologically — No abnormality.

Myelography could not be done, as attempts at subsequent cisternal and lumbar punctures did not succeed.

TREATMENT

Extensive laminectomy of the cervical spine was done through a midline longitudinal incision extending from the occiput to the second thoracic spine. The laminae of the upper thoracic and the lower four cervical vertebrae were exposed and removed. The spinal cord did not show any pulsation. One by one the laminae of the upper cervical vertebrae were removed. The moment the lamina of the second cervical vertebra was nibbled away, the cord started pulsating and appeared to bulge out of the spinal canal. The posterior arch of the atlas was also removed. The wound was closed in layers.

POST OPERATIVE COURSE

The patient started showing gradual recovery from the tenth day onwards and showed a striking improvement at three weeks. At present, eight months after operation, the patient is able to walk without any support, can sit up and can feed himself. There is no instability of the cervical spine. A fair range of cervical movement is possible. The neurological examination revealed the following state of affairs.

Sensations: There is slight blunting of sensation over the right lower limb and right half of abdomen. These are normal over the rest of the body.

There is slight wasting of the intrinsic muscles of the left hand and the muscles of posterior compartment of the left forearm.

The reflexes are slightly exaggerated on the left side. The plantar response is equivocal on both sides. The abdominal reflexes have reappeared and have been present for the last three months.

COMMENT: — Recovery in the left upper limb is rather poor. This is probably due to compression of the nerve roots at the intervertebral foramina which are narrowed.

CASE II.

C.S. a 40 year old male coming from an endemic area for fluorosis, started feeling stiffness in the back about six years before admission. This gradually progressed and in about five years time the back became rigid

like a board. One year prior to admission he started feeling numbness of the right half of the body, more marked in the upper limbs. About six months later he developed weakness of all the four limbs.

This was more on the right side than on the left. He started having great difficulty in walking. He had slight constipation but there was no urinary trouble. There was no history of venereal disease.

He has been taking a relatively poor diet consisting mainly of carbohydrates. The water consumed contained a high proportion of fluorine.

GENERAL PHYSICAL EXAMINATION

He was of moderate build with slight anaemia. The spine was stiff as a whole and no movement was possible.

NEUROLOGICAL EXAMINATION

Muscle tone was increased; it was more marked in the right upper and the right lower limbs. The biceps, triceps, knee and ankle jerks were brisk. The supinator jerk was inverted. The plantar response was extensor on both sides. The abdominal reflexes were absent.

Muscle power was grade 2-3 in all the limb and trunk muscles.

He was able to walk but with a markedly stiff gait. Sensation was intact all over the body, though there was a subjective feeling of numbness of the right lower limb and trunk.

INVESTIGATIONS

X-ray of the spine revealed increased density of bones with calcification of the ligaments.

C.S.F.:—

Protein: 65 mgs.

Globulin: ++

Chlorides: 750 mgs.

Urine Fluorides: 0.40 mgm. %

Serum Fluorides: 0.23 mgm. %

Bone Fluorides: 175.60 mgm. %

VDRL and Kahn Test: negative.

TREATMENT

Extensive laminectomy of the cervical spine was done. Operative findings were the same as in case No. 1. The cord did not show any pulsation till the lamina of C2 was removed, when it bulged backwards.

POST OPERATIVE COURSE

The patient has made a gradual recovery. The neurological examination on 9.4.63 (4 months after the operation) was as follows:

SENSATION: Intact all over the body.

GAIT: The patient is able to walk much better.

REFLEXES: Slightly exaggerated.

The plantar response is equivocal and the abdominal reflexes have reappeared.

DISCUSSION

There are some problems presented to the surgeon. It would be logical to remove the protuberant exostosis or projections which narrow the spinal canal, but any attempt at their extirpation is so fraught with danger to the cord that it is better avoided and an alternative procedure sought. Since the changes in fluorosis are most marked in the cervical and the upper thoracic regions, it was thought that de-roofing the cord over this area by extensive laminectomy would be useful at least to relieve the myelopathy. The results were better than expected. We may have to do more extensive operations in future cases or we may have to utilize the technique of Lambert Rogers to mobilize the cord if this simpler operation which can be done in one stage fails to relieve the condition. We feel that decompression of the cord in cases of fluorotic myelopathy is a useful procedure.

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