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NEUROLOGICAL COMPLICATIONS OF SKELETAL FLUOROSIS
WITH SPECIAL REFERENCE TO LESIONS IN THE CERVICAL REGION

by

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SUMMARY: Seventy cases of skeletal fluorosis observed in the endemic Nalgonda District of Andhra Pradesh, India had neurological manifestations. Special reference is made to lesions in the cervical region.

In two cases surgical decompression of the cord relieved the quadriplegia. The mechanism of the neurological involvement in skeletal fluorosis is discussed.

Involvement of the nervous system in skeletal fluorosis has been reported almost exclusively from India. The earliest description of neurological complications of fluorosis was given by Shortt et al. in 1937, who reported 10 cases from an area of endemic fluorosis in the Nellore District (1). Detailed studies were subsequently made by Siddiqui (2, 3), Rao and Siddiqui (4) and Singh et al. (5-8). In 1965 Sauerbrunn et al. (9) reported the clinical and autopsy findings of the first case of skeletal fluorosis with neurological manifestations in the U.S.A. The patient was a 64 year old white farmer who for 43 years had consumed well water with a F^- concentration varying from 2.4 to 3.5 ppm. The patient had polydipsia for 20 years and renal insufficiency.

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This paper proposes to bring forward a few interesting neurological features of skeletal fluorosis with special reference to the lesions affecting the cervical region.

The Investigation

A series of 70 cases of skeletal fluorosis with neurological manifestations was studied. All were heavy manual laborers. Most of them were residing in a known area of endemic fluorosis of the Nalgonda District, Andhra Pradesh, India (2). The F^- concentration of water consumed by the patients varied between 1.2 and 11.8 ppm. Their ages were between 21 and 70 years. Fifty-two cases were males. In 50 cases there was evidence of involvement of the cervical cord.

The important neurological manifestations were paraesthesiae, weakness of all the limbs, wasting of the small muscles of the hands, spasticity of the lower limbs and loss of sphincter control. Two cases had muscular fibrillation. The physical signs depended upon the anatomical factors of maximum narrowing of the spinal canal or of the vertebral foraminae, furthermore on whether the compression was chiefly at a single or at multiple sites. Fifty-nine of the 70 cases had sensory disturbances though these changes were inclined to be patchy. Four cases had a sensory level and loss of sphincter control. Posterior column sensory loss was common. The tendon reflexes were brisk in all cases except in 4 individuals with paraplegia in flexion where the reflexes were difficult to elicit. In 39 cases the deep reflexes were brisk in the upper limbs and absent in 2. The inverted radial reflex was present in 9 cases. The plantar response was extensor in 58, equivocal in 2 and flexor in the remaining 10 cases.

Cases exhibiting radiological changes in the skull may suffer from a perceptive type of deafness. In the series reported by Rao and Siddiqui (4) hearing loss commenced at 3000 c. p. s. and was marked (up to 60 db) at 8000 c. p. s. Bone conduction was more affected than air conduction. Hearing loss was for the high frequencies, which indicated that hearing loss resulted from pressure on the eighth nerve during its passage through the narrowed and sclerosed internal auditory meatus. Tetaniform convulsions as reported by Waldbott (10) were not observed.

The following few cases deserve special mention. Fig. 1 is the pre-operative myelogram of a case of quadriplegia, who, following laminectomy, made a complete recovery. Two patients, aged 45 and 49, had signs simulating motor-neuron disease. Cysternal myelography revealed a block at C₄ level in both individuals. The neurological findings were attributed to fluorosis. The second patient deteriorated further after laminectomy. He left the Osmania General Hospital in a moribund condition two months later. The fibrillary twitchings and the brisk reflexes in the upper limbs had become more marked.

Fig. 1

Two of the male patients with skeletal fluorosis aged 46 and 50 years respectively, developed sudden quadriplegia following a jerky movement of the cervical spine. The cisternal myelogram in the first case revealed a partial block at the C2-3 level. Laminectomy at the 1 to 3C level was performed. The dura was normal and there was no evidence of an intradural lesion. During the post-operative period he regained sphincter control and is now able to walk about with the aid of a cane.

One case of syringomyelia with Charcot's joints affecting both shoulders and left elbow complicated skeletal fluorosis. The two conditions were not related to each other.

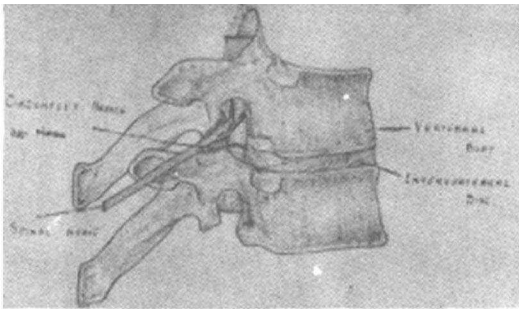
Discussion

Neurological complications usually occur in advanced cases of skeletal fluorosis in which the ingestion of large quantities of F^- has persisted for 20 years or more. However, Siddiqui (2) has reported a few cases where neurological symptoms appeared within 1 to 9 years after arrival in an area of endemic fluorosis where the F^- concentration in water is 11.8 ppm. The severity of the disease is related to the F^- concentration in water. The lowest F^- concentration in water causing skeletal fluorosis and quadriplegia was 1.35 ppm. Singh et al. (7) recorded spastic paraplegia in a 50 year old male who consumed water containing 1.2 ppm F^- .

In skeletal fluorosis the involvement of the cervical vertebrae is relatively less severe than that of the lower portions of the spine. Hence the neurological lesions involving the cervical region are relatively less severe. They resemble the clinical picture of cervical spondylosis. The patho-

Fig. 2

Postero-Lateral View

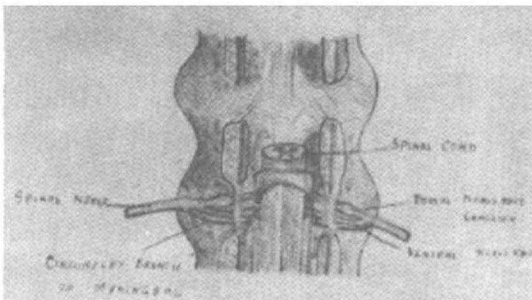


Circumflex Branch
Spinal Nerve

Vertebral Body
Intervertebral Disc

Fig. 3

Posterior Aspect

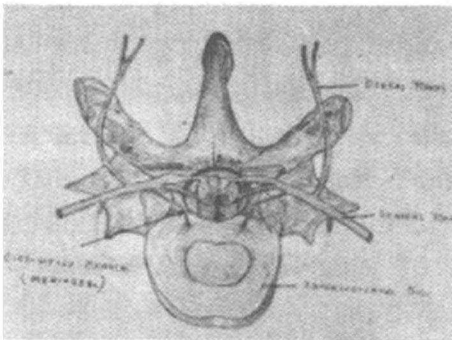


Spinal Nerve
Circumflex Branch of
Meningeal

Spinal Cord Spinal
Dorsal Nerve Root Ganglion
Ventral Nerve Root

Fig. 4

Antero-Superior Aspect



Circumflex Branch
(Meningeal)

Dorsal Ramus
Ventral Ramus
Intervertebral Disc

genic mechanism of cord compression is a more integral part of fluorosis whereas the root compression is much more common in cervical spondylosis.

The pathogenesis of the cord lesion in fluorosis is complex and several factors are concerned. Most important is the compression of the spinal cord by a fluorotic bar or protrusion similar to those reported by Lyth (11), Singh et al. (8) and temporary or permanent interference with the blood supply.

The cervical cord compression is likely to occur when the antero-posterior diameter of the spinal canal is 11 mm. or less, the average diameter of the cord of the cervical enlargement being 10 mm. The diameter of the spinal canal is not constant and varies in its size. Neurological complications are more liable to occur in persons with a narrow spinal canal than in those with a relatively spacious one. This probably explains the discrepancy encountered in the neurological picture in different cases of skeletal fluorosis although the radiological appearances might be identical.

The blood supply of the spinal cord in the cervical region comes through the anterior spinal and the radicular arteries (2). The radicular arteries running along the cervical roots may be compressed if there is gross narrowing of the intervertebral foraminae due to osteophytic growths or if there is marked root sleeve fibrosis.

Sudden compression of the cervical cord, as the result of severe injury, is a well known occurrence. Symonds (12) has discussed in detail the mechanism of cord compression in severe flexion or hyperextension of the neck in cervical spondylosis. A similar mechanism was operative in the two cases reported in the series under review who had fused cervical vertebrae. In one, the quadraplegia followed sudden flexion of the neck when the bus in which he was riding came to a sudden halt. In the other, there was sudden hyperextension of the neck when the patient was charged by a bull. Two cases of Singh et al. (9) developed sudden quadriplegia under similar circumstances.

Very severe pain in the neck is not a frequent feature of fluorosis. Williams (13) gives an interesting and thought provoking explanation for it. After emerging from the intervertebral foramen, each spinal nerve supplies a small slender circumflex branch (meningeal branch) which re-enters the spinal canal through the intervertebral foramen and is distributed to the vertebrae and their ligaments, and to blood vessels of the spinal medulla and its membranes (Fig. 2 to 4). According to Williams, this small nerve is vulnerable in its recurrence to and through the osteophytic bones. When a root has been sufficiently damaged to produce sensory loss the fibres contributing to the root must have also been damaged. If either of these mechanisms is in operation, then the joint will be denervated, neuropathic, painless and behave like a "Charcot joint".

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