# **Research Paper**

# **Medical Science**



# Fluorotic Spine: Compressive Myelopathy, Cervical/Dorsal and Lumbar Laminectomy In Single Sitting.

Dr. Rohin Bhatia	Associate Professor, Neuro-Surgery Department , Mahatma Gan- dhi Medical College and Hospital, Sitapura , Jaipur , India .
Dr. Anshul Sharma	Associate Professor, Neuro-Surgery Department , Mahatma Gan- dhi Medical College and Hospital, Sitapura , Jaipur , India .
Dr. Abhishek	2nd year P.G. student, M.S. Orthopaedics, Mahatma Gandhi Medical College and Hospital, Sitapura , Jaipur , India.

Background: The common complication of fluorosis is spinal cord compression which occur due to thickening and ossification of posterior longitudinal ligament and ligamentum flavum. The surgical management in spinal cord compression due to fluorosis at multilevel in single sitting is the aim of our study.

Material and Method: We are presenting five(5) cases with similar symptoms of cervical/ dorsal or lumbar cord compression causing cervical or dorso-lumbar fluorotic compressive myelopathy. They all belong to Rajasthan which is an endemic region for fluorosis in India managed surgically at multilevel in single sitting. These patients were included in this study. Results: Surgical outcome was based on improvement in examination findings of patients and recovery rate of patients over the period of five(5) months. At five(5) month follow up; sixty percent(60%) showed excellent outcome and forty percent(40%) showed good outcome. No patient had poor outcome in our study.

Conclusion: The spinal cord compressive myelopathy in the fluorotic spine underwent surgical management with multilevel laminectomy at two regions in single sitting showed good to excellent results.

#### **KEYWORDS**

Endemic skeletal fluorosis , Spinal Cord Compression, Myelopathy , $\operatorname{Spasticity}$ , Magnetic Resonance Imaging , Laminectomy .

#### Introduction:

Fluorosis is disease caused by intake of high concentration of fluoride<sup>[1]</sup>. The optimum upper safe limit for intake of fluorosis is less than 6 milligrams per day<sup>[2]</sup>. Endemic skeletal fluorosis occurs with chronic fluoride intoxication due to prolonged intake of high fluoride intake through water and food. The primary manifestations of fluorosis are osteosclerosis, calcification of soft tissue, mottling of teeth and bony overgrowth at margins. The secondary effects involves the nervous system damaging. Initially patients can be asymptomatic, predominantly dental and skeletal and in advance stage patients present as crippling deformities which is due to skeletal involvement and neurological compression. Symptoms can be mild motor and sensory loss or severe like spastic paraplegia and spastic quadriplegia with bladder and bowel incontinence<sup>[3]</sup>. Fluorosis is more predominant in endemic areas like Southern Rajasthan, Uttar Pradesh and Andhra Pradesh in India and many countries all around the world like China etc.<sup>[4]</sup>. The first report of endemic skeletal fluorosis was revealed in Darsi, Kanigiri and Podili areas of Prakasam district in Andhra Pradesh in the year 1937<sup>[5]</sup>...

# Material and Method:

This study was conducted at our hospital during February 15 to July 15. We are presenting five(5) cases with similar symptoms of cervical/ dorsal or lumbar cord compression causing cervical or dorso-lumbar fluorotic compressive myelopathy. They all belong to Rajasthan which is an endemic region for fluorosis in India and managed surgically at multilevel in single sitting. These five(5) patients were included in this study. They all presented with numbness and weakness in all four limbs, unable to stand (bedridden state), stiffness. There was no associated bladder or bowel dysfunction in three patient but two of them had these problem. All these patient underwent surgical management after initial assessment.

Examination includes higher mental functions and cranial nerves which were intact, motor examination revealed Upper

Motor Neuron findings and decreased power (3/5) and two with the power grade (1/5) in all four limbs without any atrophy/fasciculation, reflexes were exaggerated, spasticity was present, sensory examination revealed pain, temperature, and joint position sense were impaired below C4 dermatomes areas, Hoffman's sign was positive.

Investigations includes Laboratory tests values of serum fluoride and urinary fluoride levels which were raised, complete blood count, erythrocyte sedimentation rate, electrolytes values of calcium, magnesium and phosphorus were normal. The patient had X-Ray, CT scan and magnetic resonance imaging (MRI) of the cervical, dorsal and lumbar spine respectively which showed multilevel hypertrophy of ligamentum flavum with spinal cord compression resulting in compressive myelopathy with lumbar canal stenosis(Figure 1 and Figure 2). CT scan revealed thickening and ossification of the posterior longitudinal ligament at the cervical region, thickening and ossification of the residual ligamentum flavum at cervical and dorsal region, and dural calcification at the cervical region, leading to dorsal thecal sac indentation with a residual canal space measuring





Figure 2: MRI

Figure 1: X-Ray showing fluorosis

ISSN - 2250-1991

#### Surgical Management:

Compressive mylopathy due to fluorosis in five(5) cases presenting with similar feature treated by laminectomy at multi-level in two regions in single sitting. Surgery has showed satisfactory results and outcome with improvement in symptoms of numbness and weakness and motor examination showed increase in power . The incision site is shown in figure 3 and figure 4.

During surgery, surgical drill is very important to thin out the bone. The Dura was adherent with ligamentum flavum and is very commonly torn with CSF leak. It is very important to protect and separate the Dura from ligamentum flavum. Separate with Dissector and use punches after c thinning out the bone. Try to avoid Nebuler.



Figure 3



Figure 4

#### **Results and Discussion:**

These five patients were operated and after five (5) months of follow up the results were good to excellent. Out of those; Three patient (60%) had no numbness and weakness with power 5/5 in both upper limb and lower limb with slight spasticity in lower limbs, bladder and bowel were normal and these patients were able to walk without support(Figure 6 and Figure 7). Two patient (40%) improved to grade 3 who had initial power 1/5 in whom spasticity decreased and patient were kept catheterized, reflexes were exaggerated. These two patients are undergoing physiotherapy and are walking with a walker support (figure 8).





Figure 6

Figure 7



Figure 8

### Discussion:

In our study, patients presented with similar complaints and all belong to endemic region of fluorosis. Fluorosis is a disease caused by intake of high concentration of fluoride <sup>[1]</sup>. Fluoride is one of the trace element in human body. Important values regarding fluorosis; Safe limit for intake of fluoride is < 6mg /day<sup>[2]</sup>. Quantity of fluoride; in drinking water is 0.5 to 0.8, in urine of healthy individual is 0.1 to 2.0 ppm ( avg 0.4 ppm), in urine of fluorotic patient is 0.68 to 7.80 ppm ( avg 3.28 ppm ), in bone of healthy individual is 500 to 1000 ppm, in bone of fluorotic patient is 6000 to 8400 ppm<sup>[3]</sup>.

Fluorosis is endemic in twenty five (25) countries worldwide being more widespread in India and China <sup>[4]</sup>. The first report of endemic skeletal fluorosis and neurological manifestation was revealed in Prakasam district in Andhra Pradesh in the year 1937<sup>[5]</sup>. In Rajasthan ,Southern part is affected predominantly. Predisposing factors are high fluoride exposure through water and food, strenuous physical activity, malnutrition, deranged renal function, abnormal concentrations of certain trace elements (Storntium,magnesium,calcium etc.)<sup>[3]</sup>.

Fluorosis in humans is mainly characterized by dental and skeletal changes . In early stage it can be asymptomatic. According to most people fluoride converts hydroxyapatite crystallite into more stable fluoroapatite crystallites in the bone. The bone disease and deformity are more severe in patients with deficiency of calcium<sup>[6]</sup> .

Dental fluorosis is an early , sensitive and easily distinguishable manifestation presenting as white chalky opacities or pitting on the enamel with or without yellow lines. It is seen only if the child has resided in the endemic area during the eruptive period of the teeth if child was in endemic area, the sign of dental fluorosis can be seen and this is taken as index of en-

demicity [6][7].

Skeletal fluorosis is seen in advanced stage which causes neurological complications and crippling deformities. In early phase , it is asymptamatic . Symptoms are neck and back pain , numbness , weakness associated with rigidity. Physical findings are limited spine movement, kyphosis and exostosis [6]

Neurological complications in skeletal fluorosis occurs in 4-10% of cases<sup>[12]</sup>. These complications occurs as osteophytes protrudes along with ligamentum flavum and posterior longitudinal ligament thickening which causes the compression over the spinal cord and nerve roots. It can result in compressive myeloradiculopathy, compressive myelopathy,dorsal radiculopathy and/or compressive radiculopathy with spastic quadriplegia. These features develops if fluoride content is greater than 4 parts per million [PPM] for more than 10 years <sup>[9]</sup>. Spinal cord involvement is more common in the cervical region followed by thoracic and lumbar region but changes are first seen in lumbar region<sup>[10]</sup>. It is also seen in pelvis , forearm bones.

## Diagnosis:

The best indicator of fluoride intake is urinary fluoride level as its excretion is not constant throughout the day so 24 hr urine sample are more reliable as there is a linear relation between fluoride intake and urinary fluoride level<sup>[3]</sup>.

There is a superscan appearance like diffuse linear tracer activity along the ligamentous attachments and joint abnormalities which include osteophyte formation, ligaments calcification, posterior longitudinal ligament ossification and ligament kyphosism flavum and sclerotic bone is seen in bone scanning by technetium labeled methylene diphosphonate (99mTc-MDP) in skeletal fluorosis [11] . In sacroiliac, anterior iliac spine tracer concentration can be noted[12].X-Ray shows chalky white bones, irregular osteophytes and osteosclerosis. Computer tomography (CT) is the best imaging modality than plain skiagrams to visualize about bone pathology. CT visualizes better the direction of the osteophytes, spinal and root canal stenosis and calcified ligament which may help in surgical planning<sup>[8]</sup>. MRI of the cervical ,dorsal and lumbar spine respectively which shows multilevel hypertrophy of ligamentum flavum with spinal cord compression resulting in compressive myelopathy with lumbar canal stenosis. Fluorotic vertebra are hypointense in T1 and T2 imaging. Gupta et al. reported that due to thickening with the ossification of the ligamentum flavum and posterior longitudinal ligament there is compressive myelopathy in fluorosis[12].

Prevention is the best possible approach to fluorosis since no cure at present is possible once disease sets in there is no cure possible at present. Serpentine was used to increase fluoride excretion in human fluorosis. Magnesium intake can reduce endemic fluorosis<sup>[14]</sup>.

Defluoridation decreases fluorine level but not advisable because it increases level of aluminium which itself causes two neurodegenerative diseases ( Parkinsonism and amyotrophic lateral sclerosis)<sup>[15]</sup>.

Posterior approach is indicated in cases of canal stenosis and patients with ossified ligamentum flavum. Major cervical myelopathy precipitated by trivial trauma would also benefit from decompressive laminectomy. OPLL, both long segment and short segment, can be tackled through anterior approach with better results<sup>[15]</sup>. In fluorosis, the occurrence of compressive myelopathy can occur at different levels. The results of surgical management at two region in single sitting is unclear.

This paper presentation is mainly to suggest that compressive myelopathy due to fluorosis can be managed surgically at multilevel in two region at single sitting.

## **REFERENCES**

1. Teotia SPS, Teotia M. Endemic Fluorosis - A challenging national health problem. JAPI 1984; 32: 347-352. | 2. Environmental Health Criteria 227, Fluorides in World Health Organization Geneva: W.H.O; 2002. p. 14-6. | 3. Praveen Kumar, AK Gupta1, Shashank Sood, Ashok Kumar Verma, Fluorotic cervical compressive myelopathy, 20 years after laminectomy: A rare event, Surg Neurol Int 2011, 2:11 | 4. Skeletal fluorosis in India and its relevance to the West. Fluoride Action Network. May 2004. | 5. Shortt HE, McRobert GR, Barnard TW, Nayar AS. Endemic fluorosis in the Madras presidency. Indian J Med Res 1937;25:553-568 | 6. AK Gupta, TP Singh, Prabhat K Agrawal, Dhanveer Singh, Mohit Sachan, Vivek Agarwal Quadriparesis – A Rare Presentation of Skeletal Fluorosis JIACM 2008; 9(3): 201-204 | 7. Teotia SP. Dental fluorosis. Nat Med J India 1999;12:96-98. | 8. Reddy DR, Prasad VS, Reddy JJ. Neuroradiology of skeletal fluorosis. Clin Neurosci Ann Acad Singapore 1993;22:493-500. | 9. Haimanot RT. Neurological complications of endemic skeletal fluorosis, with special emphasis on radiculo-myelopathy. Paraplegia 1990;28:244-251. | 11. Teotia SP, Teotia M. Endemic skeletal fluorosis-clinical and radiological variants (Review of 25 years of personal research). Fluoride 1988;21:39-44 | 12. Gupta RK, Agarwal P, Kumar S, Surana PK, Lal JH, Misra UK. Compressive mylopathy in fluorosis: MRI. Neuroradiology. 1996;38: 338-342 | 13. Marier JR, The importance of dietary magnesium with particular reference to humans. Fluoride 1969; 2:185-187, 141. Reddy DR. Trial of intravenous magnesium hydroxide in fluorosis. Proceedings of the symposium on fluorosis. October 1974. Indian academy of Geoscience, Hyderabad-India: 1977. p. 449-456. | 15. D. Raja Reddy ,Neurology India Jan- Feb 2009, Vol 57, Issue 1