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RESEARCH ARTICLE

MR IMAGING OF CHRONIC SPINAL CORD INJURY

Dr. Harneet Narula[1], Dr. Gavinder Singh Bindra[2], Dr. Aditi Vohra[3], Dr. Shallini Mittal[4].

1. MD, DNB, Associate. Prof. Dept of Radiodiagnosis and Imaging- M.M. Institute of Medical Sciences and Research, Mullana, (Dist - Ambala), Haryana, India.
2. MCH, Associate Prof, Dept of Neurosurgery- M.M. Institute of Medical Sciences and Research, Mullana,(Dist- Ambala), Haryana, India.
3. MD, Associate Prof, Dept of Radiodiagnosis - M.M. Institute of Medical Sciences and Research, Mullana, (Dist - Ambala), Haryana, India.
4. DMRD, Senior resident, Dept of Radiodiagnosis and Imaging - M.M. Institute of Medical Sciences and Research, Mullana, (Dist - Ambala), Haryana, India.

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Corresponding Author

Dr. Harneet Narula

Abstract

Spinal cord injury is a devastating trauma resulting in variable degree of neurological deficits, depending upon the severity of cord damage. Neurological outcome depends not only upon the extent of initial cord injury but also on the superimposed chronic sequel and wallerian degeneration. Spinal cord injury has variable appearance on imaging in acute and chronic phase, recognition of which is important not only for adequate and timely treatment but also for prognostication. Recognition of wallerian degeneration is also important as it is another independent cause of delayed neurological deterioration. We report a case of young child with spinal cord injury presenting in chronic phase with neurological deterioration due to development of extreme degree of spinal cord atrophy distal to the site of injury which is very rare and can be attributed to severe degree of distal wallerian degeneration. We also review the literature for chronic spinal cord injuries and wallerian degeneration with emphasis on various causes of delayed neurological deterioration and consequent importance of recognizing them by imaging.

Introduction:-

With the increase in incidence of vehicular accidents, traumatic spinal cord injuries are increasing day by day. Traumatic spinal cord injuries in children are rare and may be unaccompanied by disruption of spinal column called SCIWORA, i.e spinal cord injury without radiographic abnormality.[1] Patient with this entity presents with variable degree of motor and/or sensory neurological deficit.

Injured spinal cord may have spectrum of insult ranging from edema, contusion or hemorrhage, compression or cord transaction and the extent of these findings determines the neurological outcome. Apart from extent of damage to spinal cord at the time of injury the neurological status also depends upon the evolution of injury. Patients may have static neurological deficits for a long period after injury and later may show deterioration in chronic stage by development of wallerian degeneration, syrinx, cystic myelopathy, myelomalacic myelopathy, atrophy or cord adhesions.

Neurological deterioration in chronic phase may improve if the condition is amenable to surgical treatment as in cystic myelopathy, syrinx, cord adhesions or in presence of persistent cord compression. However in other conditions like atrophy the prognosis is poor.

Usually post traumatic cord atrophy begins at the level of cord injury that may extend from the injured site distally. We report a case of a young child with old cervical cord injury presenting with further neurological deterioration in chronic phase, showing proximal wallerian degeneration and severe distal spinal cord atrophy, with the cord being barely perceptible, while there was mild myelomalacic changes at the site of injury.

We report this case in view of rarity of occurrence of such severe degree of post traumatic atrophy distal to the site of injury without atrophy at the injured site along with expected changes of wallerian degeneration proximally. We also review the literature of chronic spinal cord injury and its radiological appearances.

Case report:-

A young 4 year old child was run over by a tractor while sleeping in field and presented to the neurosurgery department of our hospital with total loss of movement of all the four limbs, i.e. quadriplegia and spinal shock. Child was evaluated clinically and stabilized and then sent for radiological investigations to our department of radiodiagnosis. X-rays and CT of cervical spine were normal with no features of spinal disruption suggesting SCIWORA. MRI of cervical spine with whole spine screening was done which revealed long segment of altered signal in cervical cord extending from C4 to C7 level, appearing mildly hypointense on T1 and showing large central hyointensity with peripheral hyperintensity on T2W images, corresponding to hemorrhage (Image 1a). Child was managed conservatively with high dose methylprednisolone. After a period of about one week, the patient came out of spinal shock with minimal improvement of neurological status with muscle power improving to 1 to 2/5 over next six months of rehabilitation. However after a period of one year post injury patient presented with marked spasticity with history of neurological deterioration over a period of last four months with progression to quadriplegia. MRI of cervico-dorsal spine with whole spine screening was performed to know the status of chronic spinal cord injury. MRI revealed mild T2 hyperintense signal in cervical cord at the site of previous injury, C4-C7, with normal T1 signal suggesting mild changes of myelomalacia (Image 1 b). Cord proximal to C4 level showed hyperintense signal in dorsal column on axial images suggesting wallerian degeneration (Image 1c). Cord distal to C7 level was markedly atrophic till the distal end and appeared like a thin strand of hair (Image 1d). No evidence of syrinx or cyst formation was seen.

In view of the presence of severe cord atrophy the patient's family was explained about the poor prognosis of recovery and physiotherapy and nursing care were suggested.

Discussion and review of literature:-

Spinal cord injury is a devastating injury leaving the afflicted person with functional loss and dependence on others. Spinal cord injury in pediatric age group is uncommon.

In pediatric age group cervical dorsal spine is the most common site of injury in adolescents while upper cervical spine is more common in the very young.[2, 3]

The spinal cord injury may present as variable degree of motor or sensory neurological loss which may or may not be accompanied by radiographic disruption of spinal column on X-rays or CT scan. In the latter condition the entity is termed as SCIWORA, spinal cord injury without radiographic abnormality, term being coined by Pang and Wilberger in 1982 and occurs due to the mismatch of elasticity of spinal cord and spinal column.[1, 2]

The mechanism of cord injury may be direct cord traction, direct compression by ligamentous/discal bulges and epidural hematoma or subluxation. Injury may also occur by transmission of external kinetic energy. Even ischaemia and impaired perfusion may contribute to the injury.[1]

MRI remains the mainstay of diagnosing spinal cord injury. MR will reveal not only the site and length of cord injury but also the type of injury, i.e. cord edema, contusion, hemorrhage or compression, which is important in view of different levels of neurological recovery and prognosis. [4, 5, 6]

MR may be normal in few patients despite neurological deficits.

Cord edema has the best chances of recovery and is seen on MRI as intramedullary T2W hyperintense signal with no signal changes on T1W (Image 2a). However involvement of lengthier segment and more rostral extent may have

poor motor outcome. Cord edema significantly increases during its evolution in acute phase while hemorrhage is usually static [7].

Cord contusion is associated with intermediate prognosis and appears on MRI as focal area of altered intramedullary signal isointense on T1W images and shows central iso to hypo intensity with thick peripheral rim of hyperintensity on T2W images (Image 2b).

Cord hemorrhage has the worst prognosis and is usually associated with complete and irreversible neurological deficit. It is perceived on MR as heterogeneous signal on T1 while on T2 it shows large central area of low signal with thin peripheral rim of hyperintensity (Image 1a). Length of involvement has significant correlation with extent of neurological deficit as well as future recovery. Sizeable hemorrhages of size more than 1cm is associated with marked edema and have poorer prognosis with significantly more chances of retaining complete injury at follow up.[7]

Cord compression is seen as severe obliteration of the spinal cord with significant alteration of its morphology which prevents detection of signal alterations and such cases usually require surgery. Compression can be caused by hematoma, disc bulge or vertebral fracture fragments (Image 2c).

Cord transection is the most severe of all the cord injuries with no scope of any neurological recovery. Radiological appearance of transection on MRI is the sagittal discontinuity of cord (Image 2d).

Patient after initial stabilization may deteriorate further in subacute or chronic phase.

In subacute phase further deterioration can be caused by a very rare complication known as subacute progressive ascending myelopathy. This is a syndrome of ascending neurological deficits presenting within four weeks of initial injury by four or more segments following traumatic spinal cord injury which is not related to mechanical compression, instability, or syrinx formation at the level of injury or above but may be related to ischemic and inflammatory changes triggered by cord injury.[8,9] MRI feature are of cord expansion and an increased intramedullary signal on T2 -weighted sequences for a long segment beyond the extent of initial injury.

In chronic phase patient may remain neurologically stable or may deteriorate. The patient in whom deterioration occur are said to be affected by post traumatic progressive myelopathy. The cause of this type of myelopathy may be the development of atrophy or evolution into conditions like myelomalacia (progressive post traumatic myelomalacic myelopathy), spinal cord cyst (progressive post traumatic cystic myelopathy), syrinx or formation of intradural adhesions.

Spinal cord cysts may form at the site of previous trauma and may be a cause of progressive neurological deficit with the condition known as post traumatic cystic myelopathy (PTCM). On imaging the posttraumatic spinal cord cyst are hypointense to the cord on T1W images, and markedly hyperintense on the heavily T2-weighted images with signal paralleling that of CSF (Image 2e). These patients may benefit from shunting of the cyst into subarachnoid space. [10,11,12,13]

Posttraumatic syringomyelia (PTS) refers to the formation and progression of a CSF filled cystic cleft within the spinal cord. PTS is an uncommon complication in which patient may present many years after the initial spinal cord injury by the insidious progression of pain and loss of sensorimotor function. Patients of PTS may benefit from shunting of the syrinx especially in patients with cysts larger than 2cm and in symptomatic cysts. [13, 14]

Radiologically syrinx is seen as a very well defined elliptical area of intramedullary CSF intensity on T2 weighted images (Image 2f). It has to be differentiated from cystic myelomalacia in which a rim of hyperintensity is present in relation to the cystic component on T2 weighted images, and the cystic component is usually small.

Spinal cord myelomalacia or progressive post traumatic myelomalacic myelopathy (PTMM) can also cause delayed and progressive neurological deterioration in the form of progressive motor loss or changes in sensory level, increased spasticity or loss of bowel or bladder control. Differentiation from post traumatic cystic myelopathy is important, as PTMM may be caused from extramedullary cord adhesions/tethering and might benefit from lysis or release of adhesions while PTCM may benefit from shunting of the cyst.[15,16] PTMM appears as a lesion with low signal intensity within the cord on the T1 -weighted images and slightly more intense than the surrounding cord on

the moderately T2-weighted images while on heavily T2-weighted images shows signal which do not parallel the signal from the cerebrospinal fluid in the adjacent subarachnoid space (Image 2g).

Spinal cord atrophy represents axonal loss and invariably reflects poor prognosis. Cord atrophy occurs as consequence to a complex phenomenon related to the effects of dynamic inflammation which leads to demyelination, axonal injury, neuronal loss and Wallerian degeneration.[16, 17] Spinal cord atrophy is defined as an abnormal narrowing of the spinal cord in the sagittal plane in two or more segments beyond the limits of vertebral injury. Cord atrophy on MRI is seen as cord thinning without accompanying signal changes (Image 1d). Normal cord measures approx 7mm in cervical region, 6mm in upper dorsal and 5mm in lower dorsal region.

Cord tethering can also be a cause of delayed myelopathy in patients with traumatic cord injury. Release of the tethered cord should be considered in a patient presenting with delayed myelopathy when a posttraumatic syrinx is not found. [18] On MRI adhesions are seen as focal area of cord appearing confluent with the dura.

Due to the injury to the axons by trauma, antegrade degeneration of axons and their myelin sheath begins to occur and is labelled as Wallerian degeneration. It starts as early as 8 days on histological studies and is usually evident as early as 7 weeks on MR. It is seen as altered signal, appearing hyperintense on T2W images in dorsal column above the level of injury (Image 1c), such changes are better appreciated on axial images and such changes becoming more pronounced and discrete with progression of time. Similar altered signal can be seen in lateral columns below the level of injury. [19, 20, 21, 22, 23]

Identification of changes of Wallerian degeneration are important as its presence above the site of injury indicates that there was partial or total interruption of axons of dorsal column and Wallerian degeneration itself can be a cause of further neurological deterioration in chronic phase. Diffusion tensor imaging can detect Wallerian degeneration even when conventional MR fails to do so and shows that axonal degeneration is a continuous process occurring for years after cord injury.

Diffusion tensor imaging nowadays is a new emerging tool that may be used in the clinical assessment of patients with Spinal cord injury. Diffusion tensor imaging reveals that the changes occurring remote from the site of injury occur most likely secondary to the degeneration of white matter tracts.[24] Decreased fractional anisotropy is associated with poorer motor and sensory function.

Incidence of various types of chronic cord injuries varied in different studies. According to Siberstein, cord atrophy (43%) and syrinx (41%) were the most frequent imaging findings with cord compression seen in 24%, cystic myelomalacia in 15%, myelomalacia in 11% and normal spinal cord in 18% of patient respectively. [16]

While in study by Curati, the incidence of various chronic cord injuries were as - myelomalacia in 37%, syrinx in 40%, persistent cord compression in 32% and atrophy in 18%. [25] Neurological changes after initial neurological stabilisation are seen in patients with extended atrophy, malacia or a syrinx, not in those with only a cyst or cord disruption. Tethering is usually always associated with other lesions.

According to study be Yamashita, patients with normal signal on both T1 and T2 had mild neurological impairment with prognosis being excellent. [26] Patients who had normal T1 signal and high T2 signal had mild degree of neurological deficit and was frequently associated with compression while patients with low intramedullary signal on T1 images and high signal on T2 images had worst prognosis. They also observed that cord atrophy occurs with long standing myelopathy.

Unlike injuries in adults, pediatric spinal cord injuries show significant functional improvement irrespective of the completeness of injury. However, some pediatric patients may not recover and might show progression of neurological deficit wherein chronic spinal cord injury sequelae must be considered. [1] Children should be followed up with MRI examinations to detect chronic spinal cord sequelae at an early stage so that if condition is amenable to surgery it can be treated early.

MR imaging appearance of various types of cord injuries in different phases is summarized in table 1.

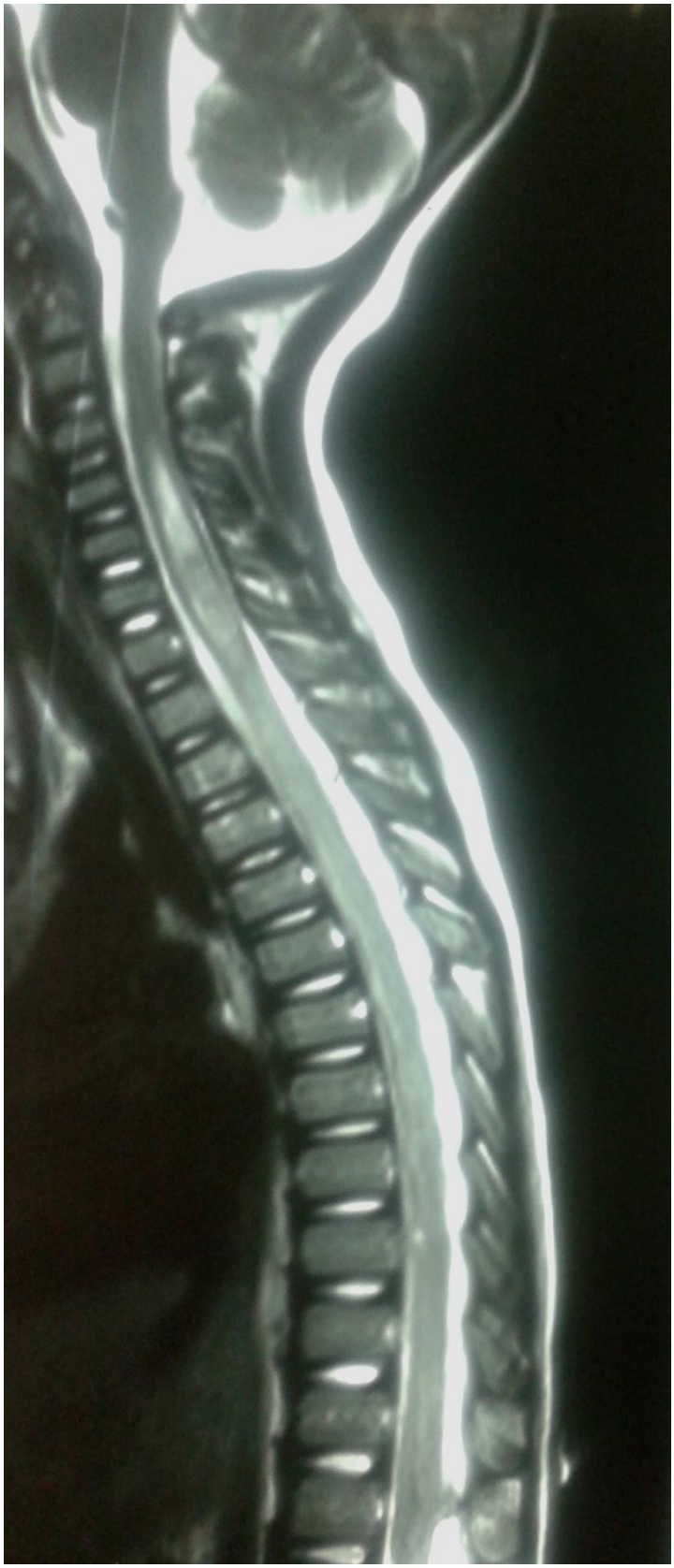

Figure1a: Sagittal T2W image shows cord hemorrhage as large central intramedullary hypointense area surrounded by thin peripheral hyperintensity at C4-C7 level.

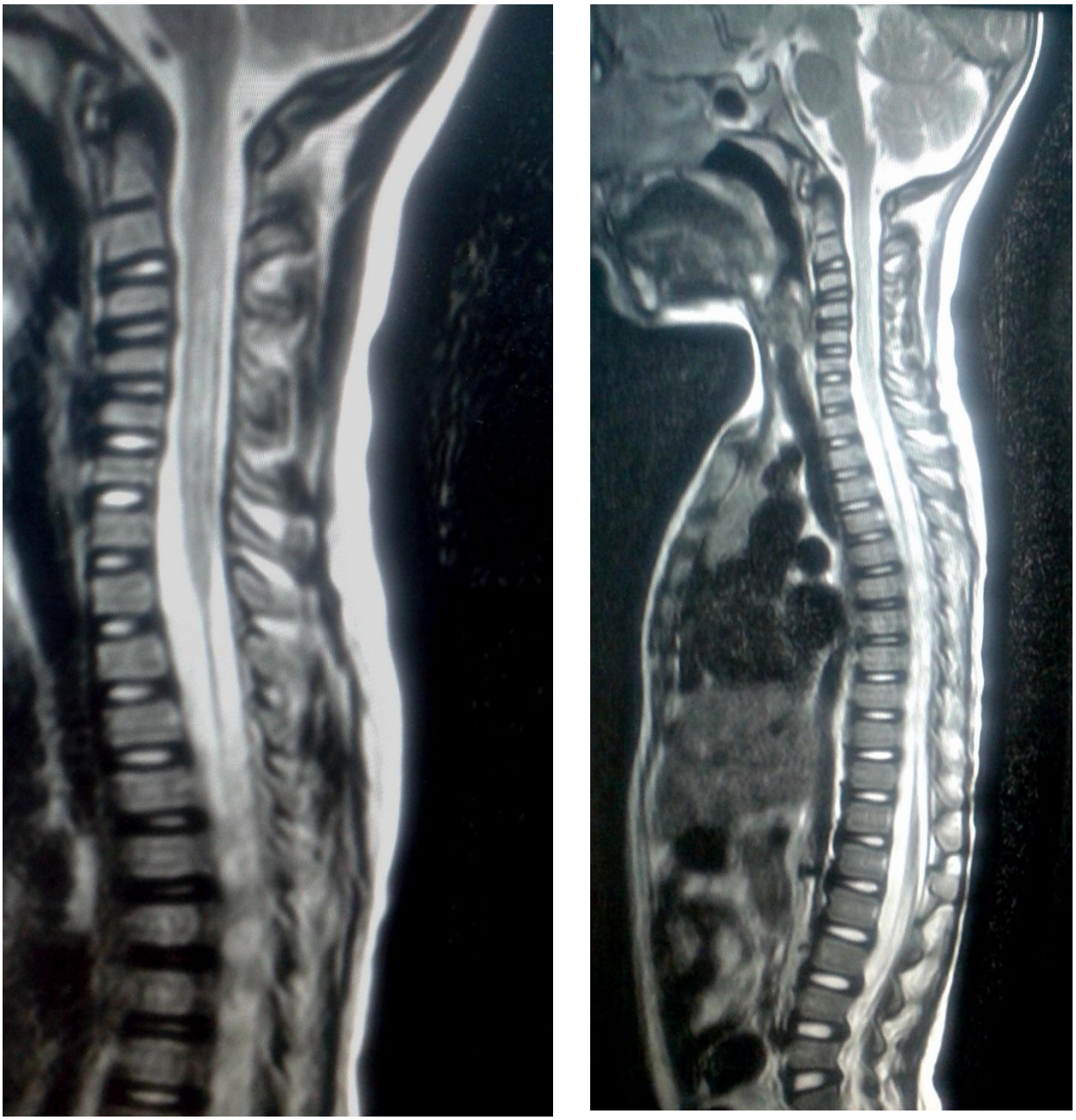

Figure1b-1d: Follow up MRI after 16months of initial spinal cord injury.

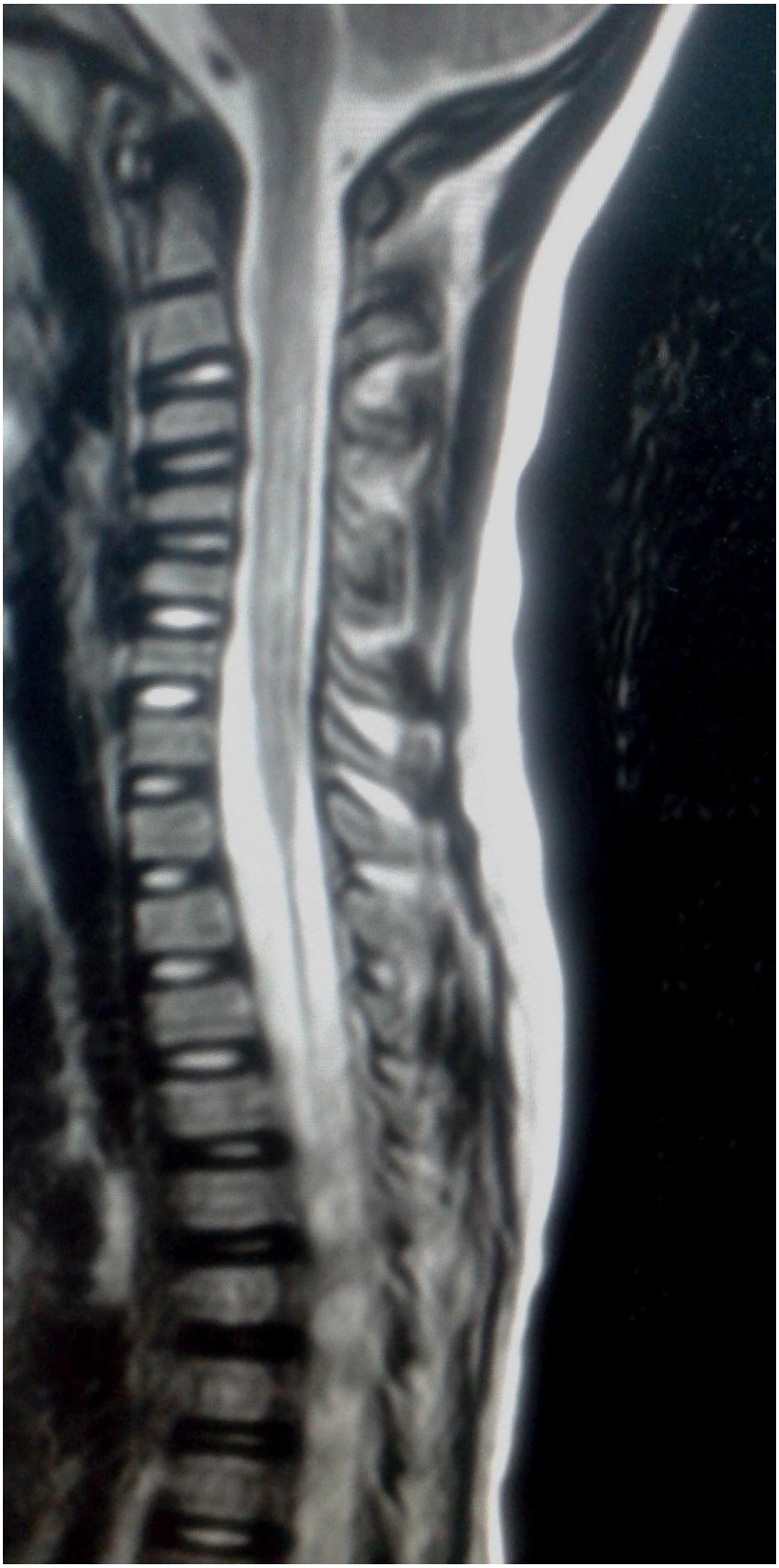

Figure1b: Sagittal T2 image showing myelomalacia at the site of previous injury as intramedullary linear area of mild T2 hyperintensity.

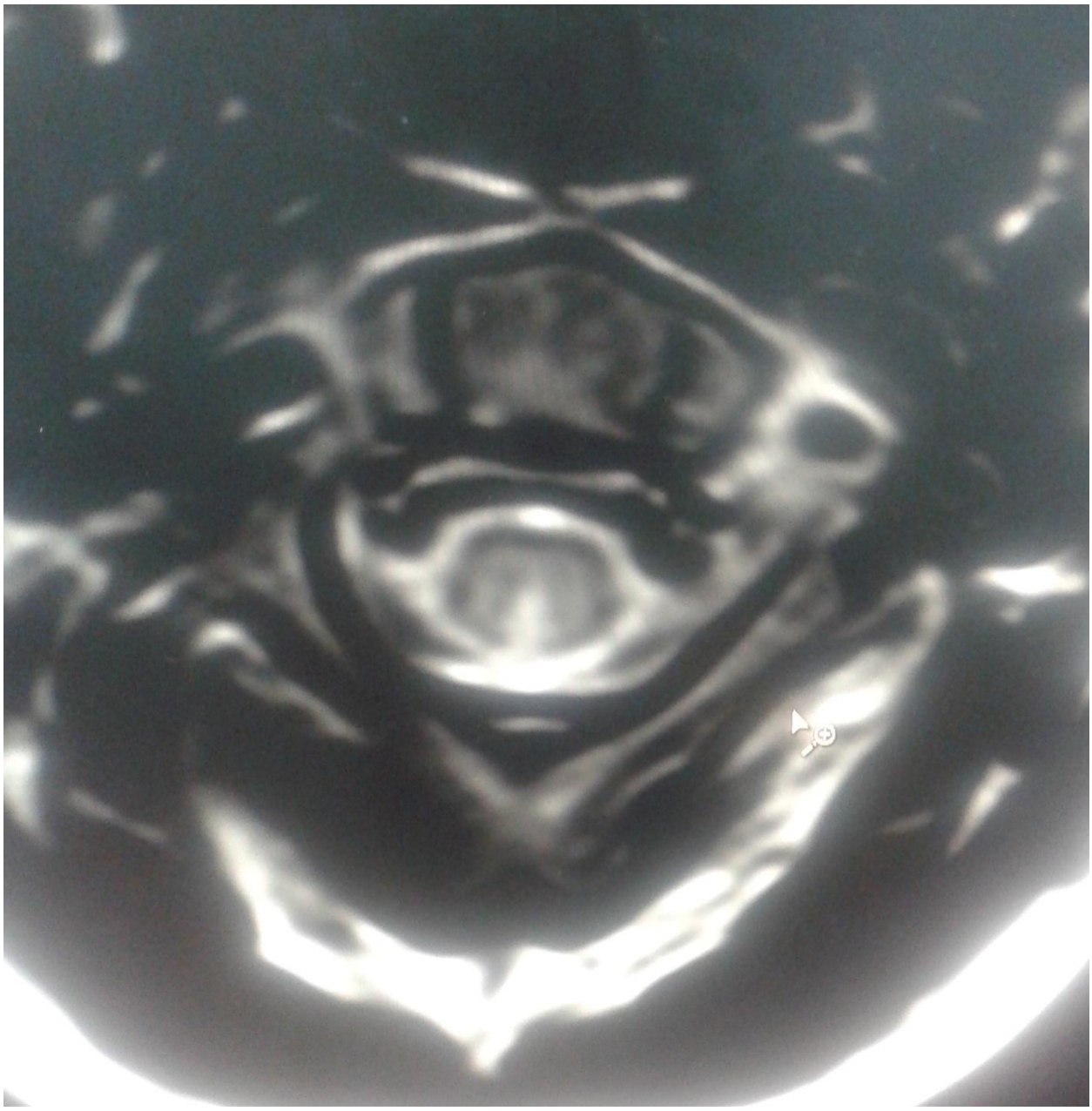

Figure1c: Axial T2 images above the site of previous injury showing hyperintense signal in dorsal columns indicating wallerian degeneration.

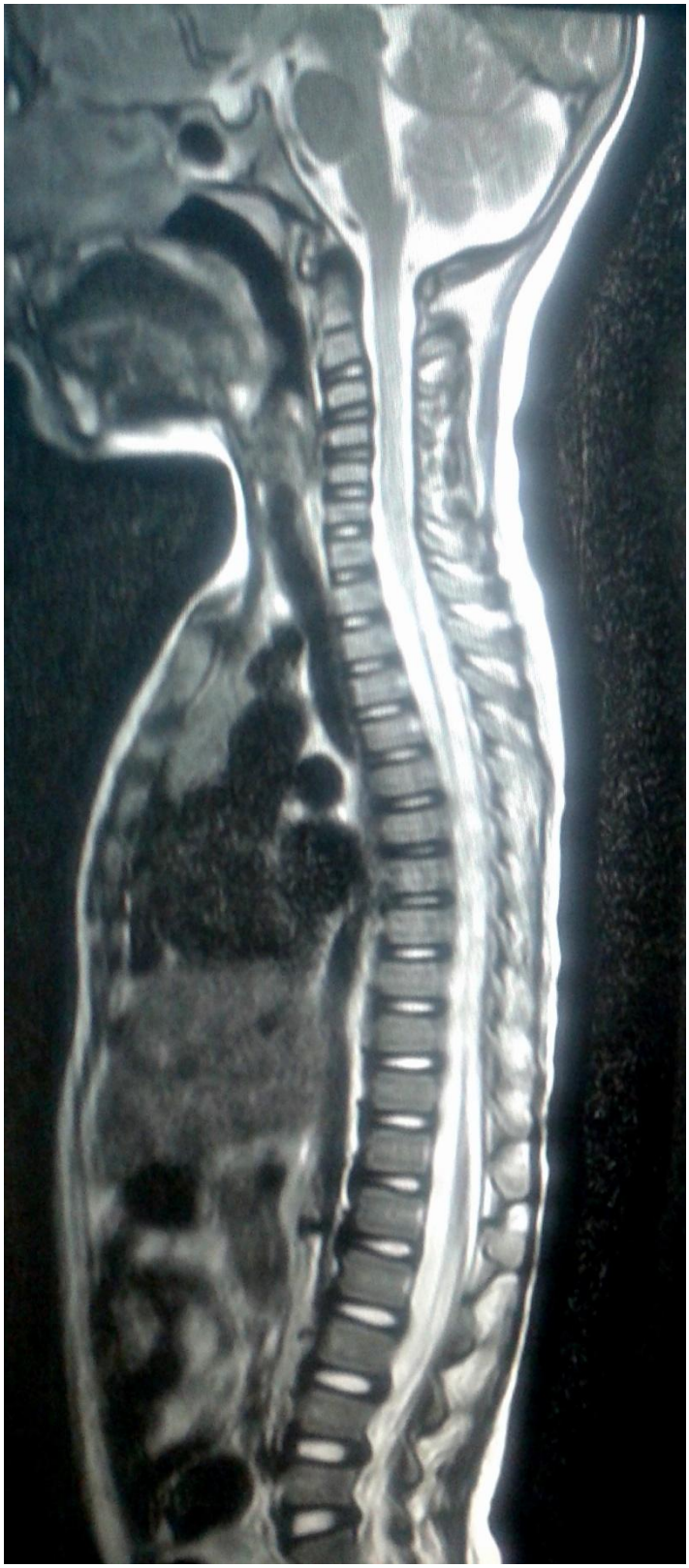

Figure1d: T2W sagittal image showing severe diffuse thinning of spinal cord distal to C7 level.

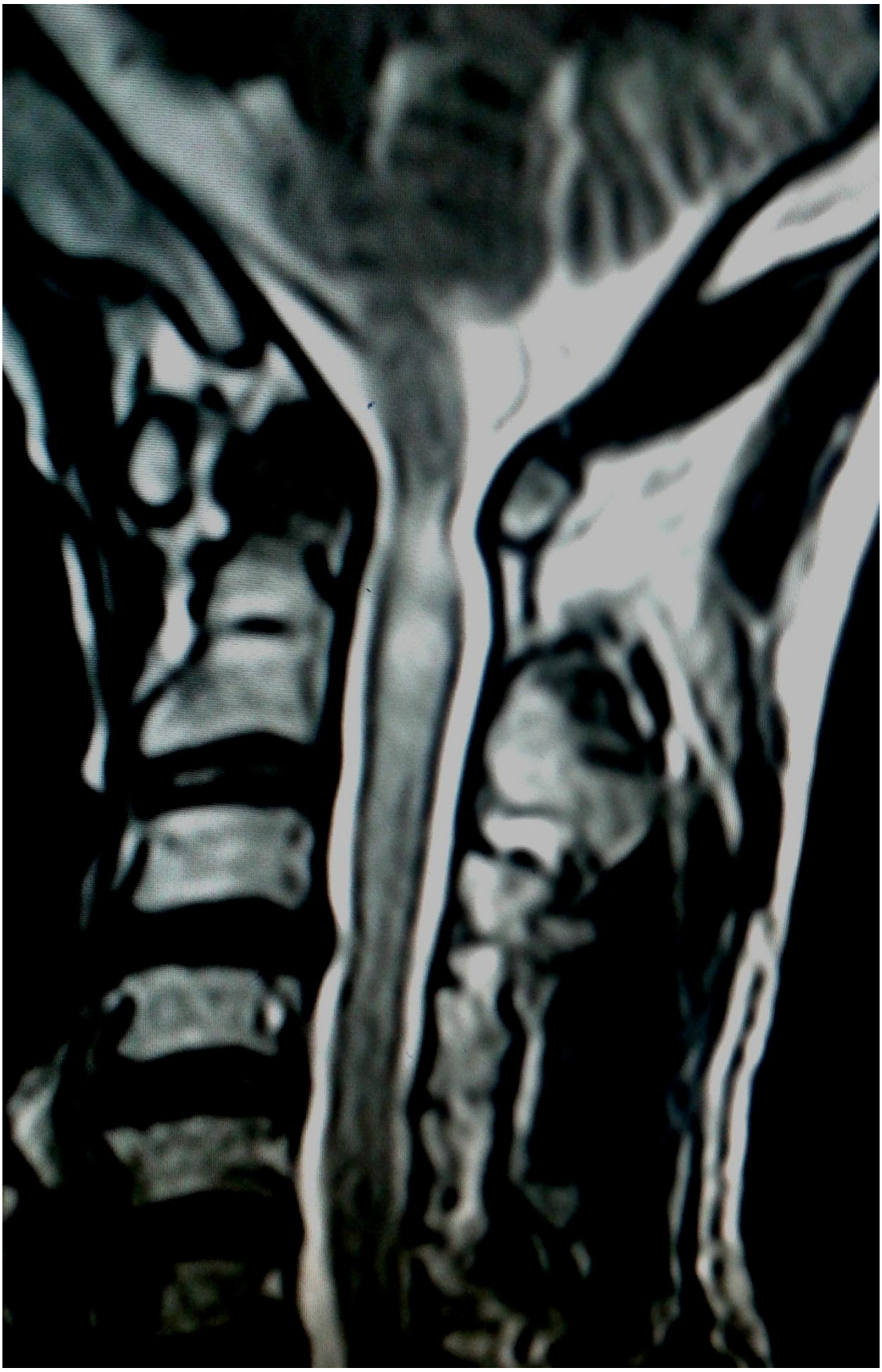

Figure 2a: Cord edema- diffuse T2 hyperintense signal.

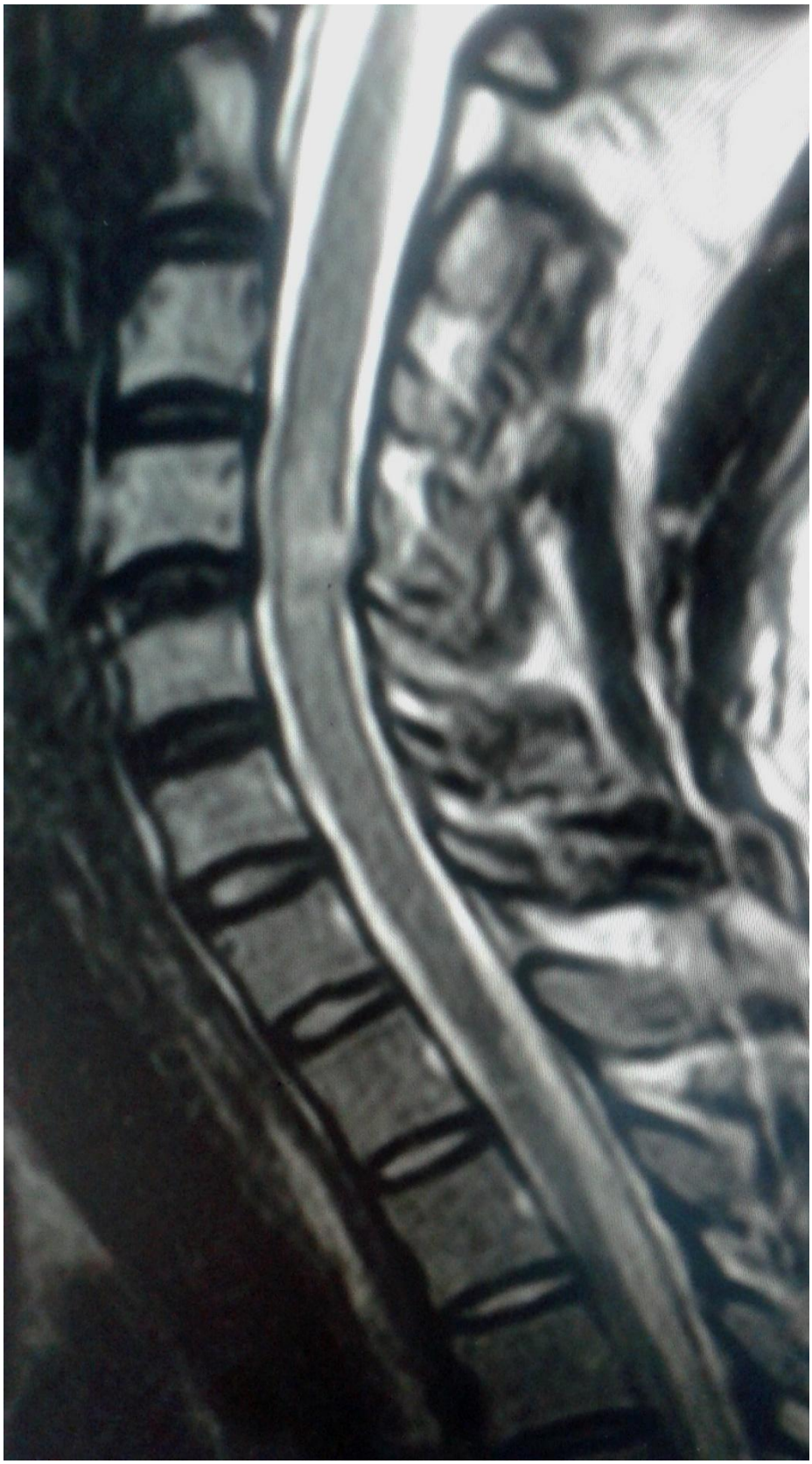

Figure 2b: Cord contusion- small central T2 hypointense signal surrounded by large peripheral T2 hyperintensity.

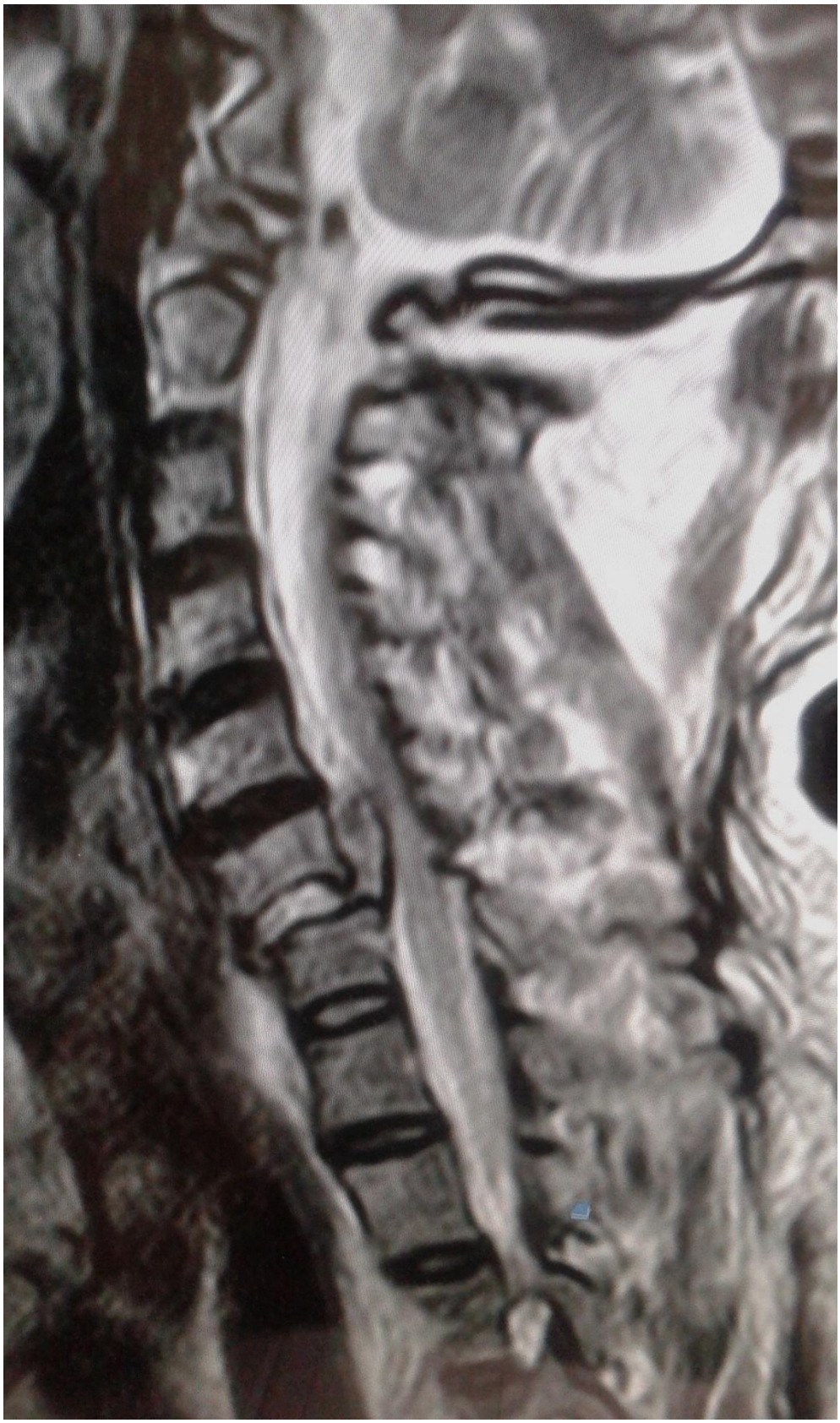

Figure 2c: Cord Compression associated with epidural hematoma and traumatic spondylolisthesis.

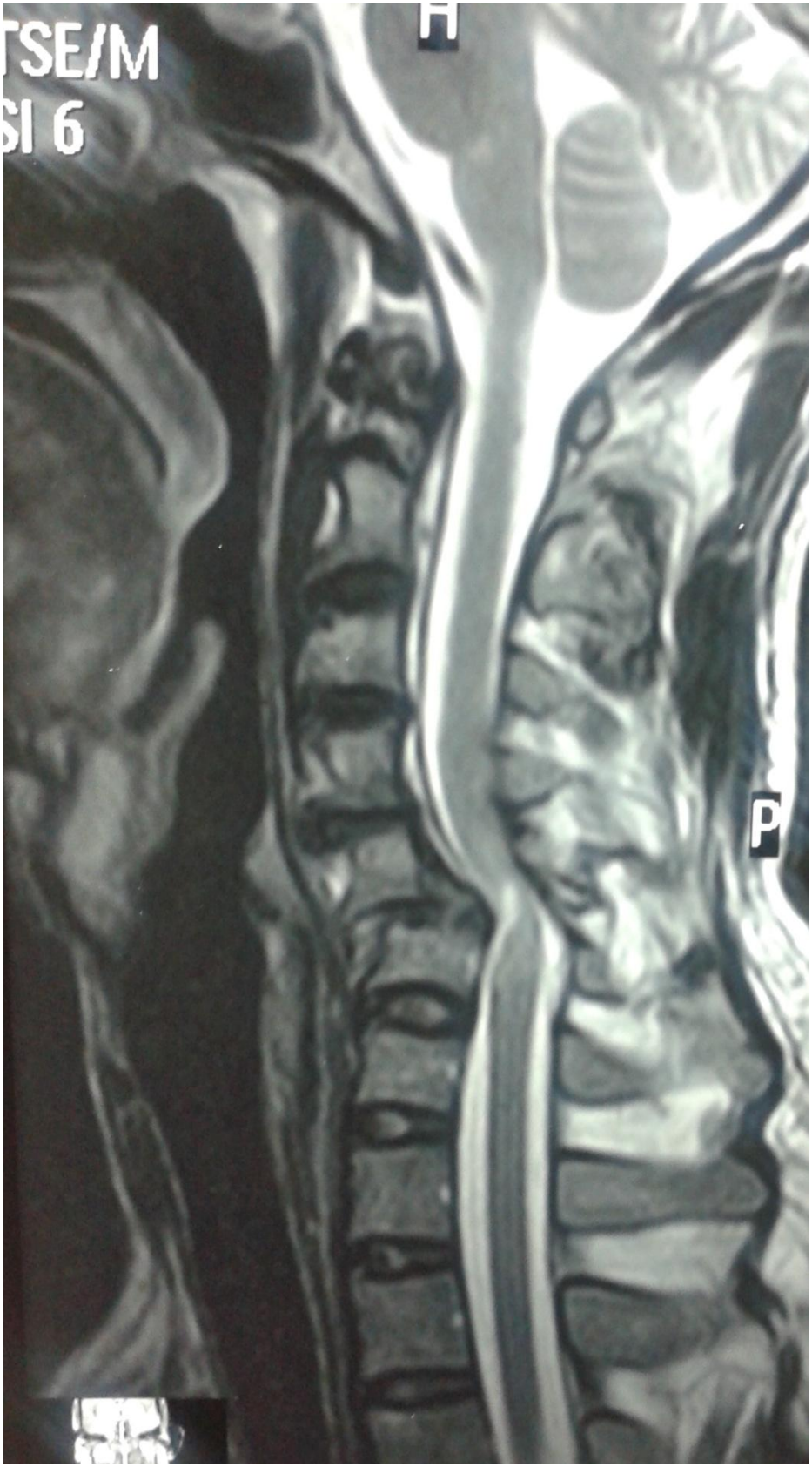

Figure 2d: Near complete cord transaction - sagittal discontinuity of cord.

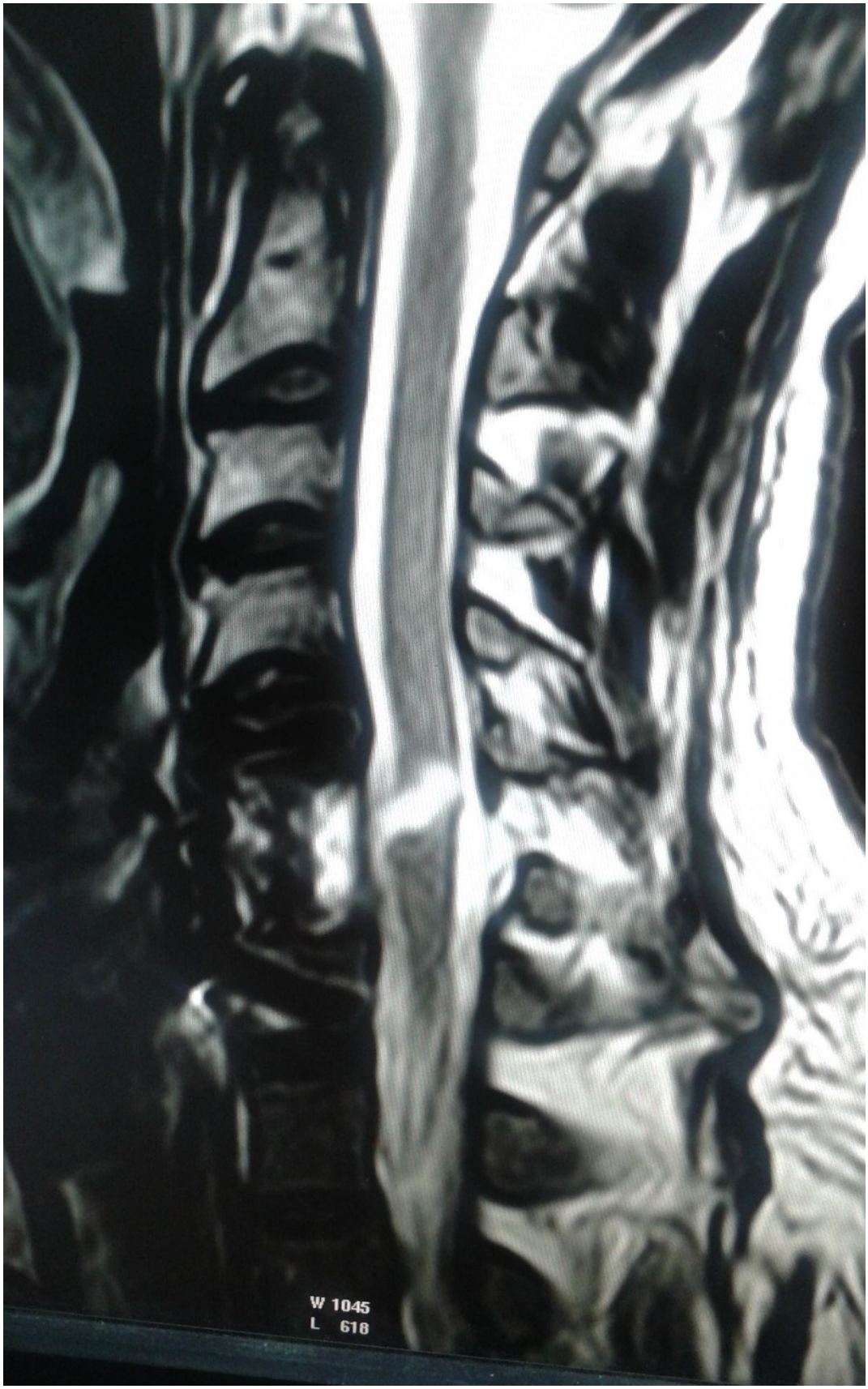

Figure 2e: Spinal cord cyst – focal rounded area of CSF signal at the site of previous injury.

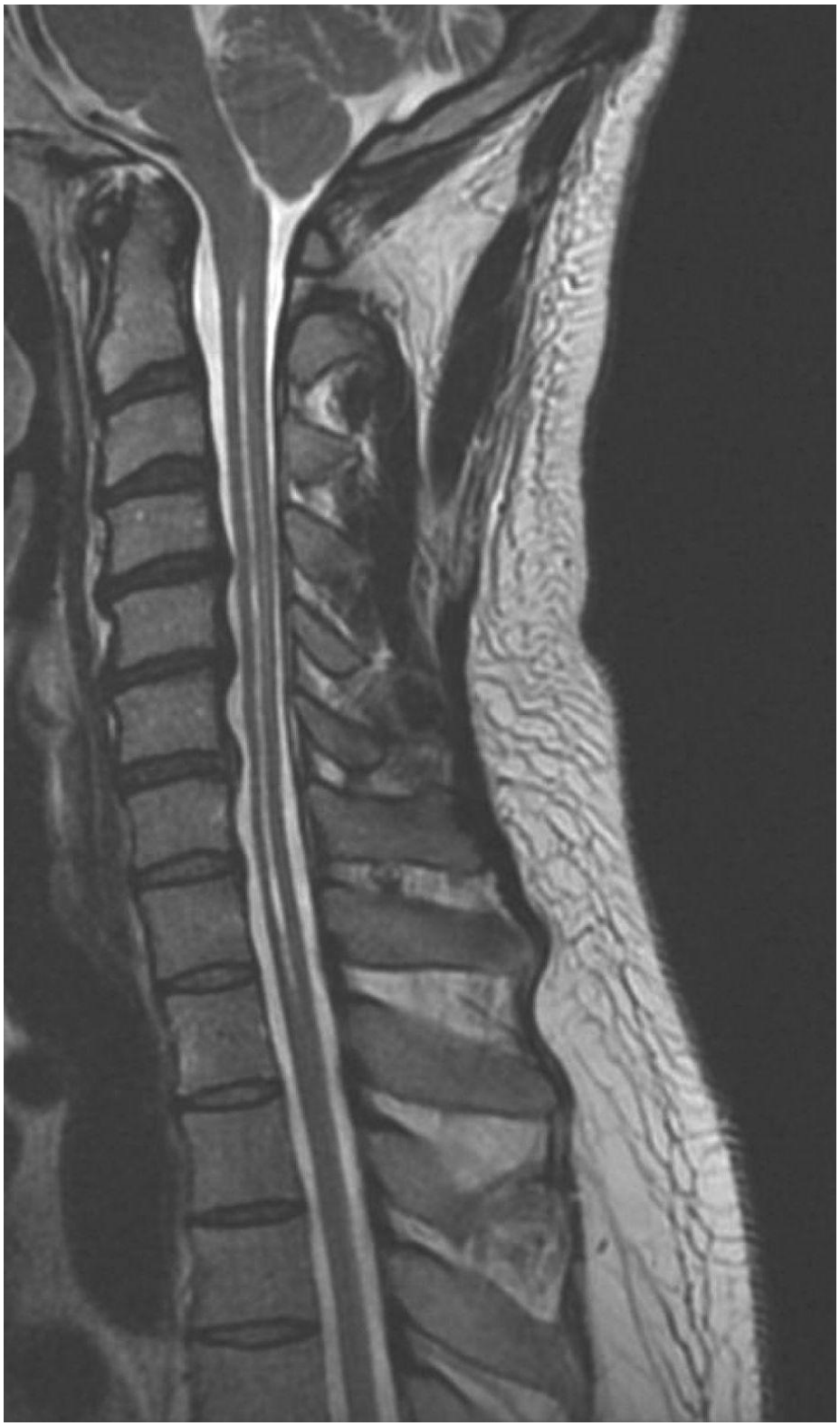

Figure 2f: Syrinx - Linear area of intramedullary CSF signal.

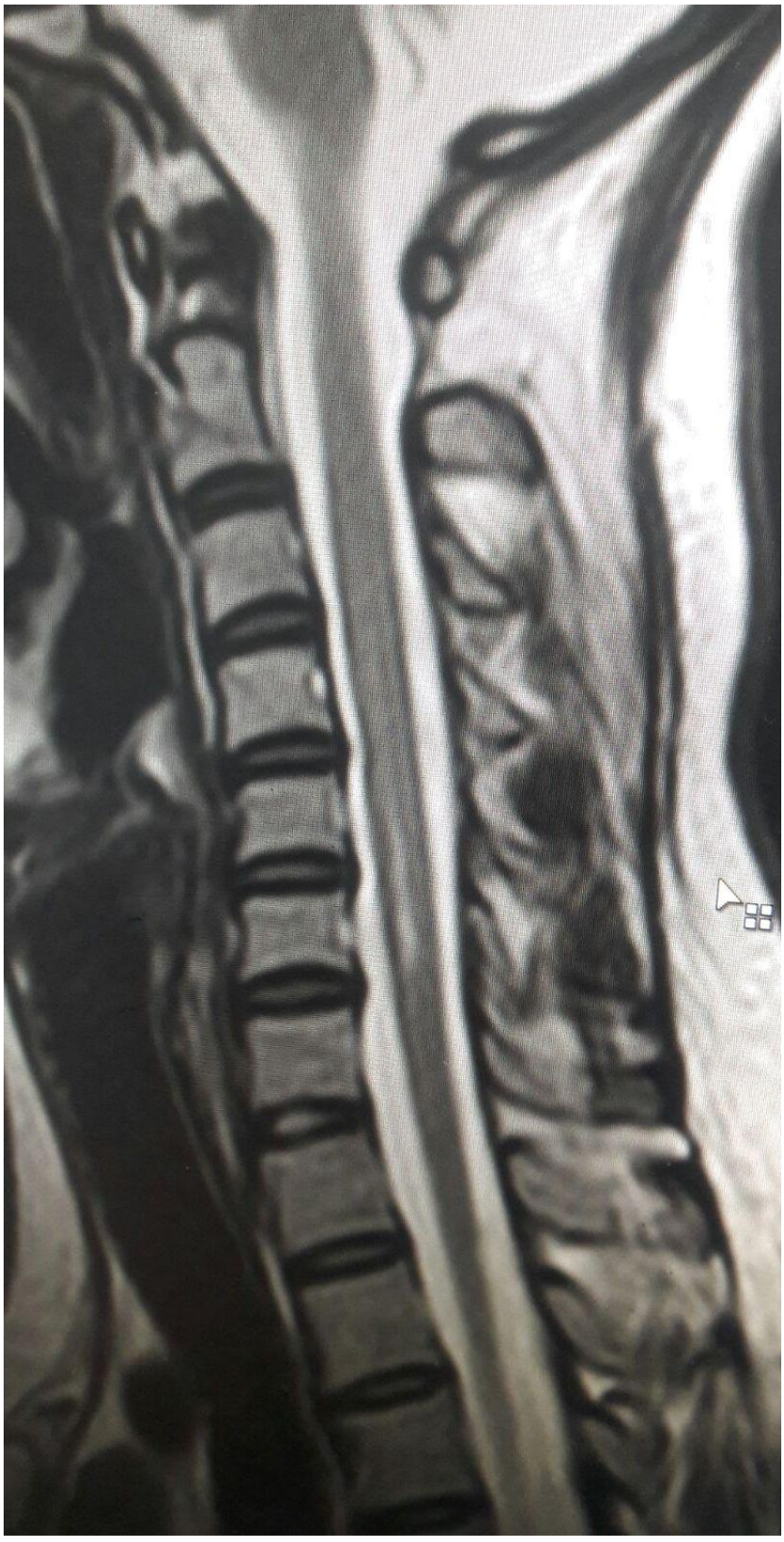

Figure 2g: Myelomalacia- Mild intramedullary T2 hyperintense signal, not paralleling signal of CSF.

Conclusion:-

Spinal cord injury is a devastating injury the neurological outcome of which is dependent not only upon the extent of initial damage but also upon evolution into various forms of chronic sequelae such as myelomalacia, syrinx, atrophy as well as Wallerian degeneration which itself is another independent cause of neurological deterioration. Cord atrophy usually occurs at the site of initial injury but may occur distal to the site of injury as in our case and can be attributed to severe distal wallerian degeneration.

Table 1

Acute phase	
1. Cord edema	T1 – isointense T2- hyperintense
2. Cord contusion	T1- isointense T2-small central hypointensity, large peripheral hyperintensity
3. Cord hemorrhage	T1- heterogeneous hyperintensity T2 – Large central hypointensity with small peripheral hyperintensity
4. Cord compression	T2- severe obliteration of cord obscuring the internal medullary signals
5. Cord transection	T2- sagittal discontinuity of spinal cord
Subacute phase (1-4weeks)	
Sub acute ascending progressive myelopathy	T2- expansion and hyperintense signal extending beyond confines of acute injury
Chronic phase	
1. Post traumatic syrinx	T2- very well defined linear intramedullary CSF intensity extending beyond the confines of initial injury
2. Post traumatic cysts	Small focal well defined lesion appearing at site of previous injury : T1W - hypointense T2 weighted – markedly hyperintense with signal of CSF
3. Cystic myelomalacia	T2 - small well defined rounded or elliptical area of intramedullary CSF intensity representing small cyst at the site of previous injury surrounded with a hyperintense rim
4. Myelomalacia	T1 – low signal, T2 weighted images - slightly more intense than the surrounding cord, but less than that of CSF
5. Atrophy	Cord thinning without accompanying signal changes
6. Adhesion	Focal area of cord appearing confluent with the dura
7. Persistent compression	Focal narrowing with adjacent persisting compressive cause
Wallerian degeneration(>7 weeks)	T2- hyperintense signal in dorsal column above the injury Hyperintense signal in lateral column below the injury

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