

Split Cord Malformation: Clinical Manifestations and Treatment Outcome

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ABSTRACT

Background: Split Cord Malformation (SCM) is the most common underlying anatomical abnormality of occult spinal dysraphism.^[2,31] Split cord malformations (SCM) are increasingly being recognized as one of the causes of tethered cord syndrome. Renaming them as SCMs has reduced the conflict in terminology, like 'Diastematomyelia' (Olivier) 'Diplomyelia' (Bruce) with poor distinction. Pang et al. have proposed unified theory and coined them as 'Split cord malformations'^[12, 28, 29, 30]. With the advent of MRI, the diagnosis of SCM has become relatively frequent. **Objective:** To study the clinical profiles, radiological findings and surgical outcome of patients having split cord malformation. **Patients and methods:** This study was conducted in neurosurgery department, Assiut university hospital over a three-year period between January 2005 and December 2007. Of 90 patients with spinal dysraphism, 10 (11.1%) patients had symptoms and signs that raised the suspicion of occult spinal dysraphism, which was confirmed on neuro-imaging investigations to be split cord malformation and some associated anomalies. **Results:** There was a total number of 10 patients, their ages ranged from 2 months to 19 years and 7 of them were females. They presented with cutaneous markers in 90%, neurological syndrome in 80%, while neuro-orthopedic syndrome displayed in 60%. They verified into type I SCM in 9/10 of cases while type 2 SCM in one case. Also, pure SCM (without meningocele) was found in 9/10 of cases. The sagittal clefting of the cord was opposite lumbar vertebrae (L1- L4) with bony spur in 9 cases and lower dorsal opposite D10 with fibrous septum in one case. Low lying conus was observed in 9 cases at the level of L5 (8/9 cases) and sacral one level in one patient. No cranial or cranio-spinal junction associated anomalies were present. Results among surgically treated patients were good. CSF leak encountered in 3 patients only and treated surgically in 2 of them, no meningitis at all and no mortality among our patients. **Conclusion:** SCMs are rare complex forms of occult dysraphism. In type I the cord is anchored by an extra-dural bony spur and /or short, thick filum terminale. In type II tethering is caused by fibrous band, which anchors, hemicords to the anterior dura. MRI is the imaging of choice, but high resolution CT can be complimentary. Hypertrichosis is the commonest marker to the clinical diagnosis. Surgery and detethering should be considered prophylactically after diagnosis and in all symptomatic children. Rational decisions are necessary in children who are neurologically compromised and harbor complex anomalies. Outcome is very good in children with isolated split cord malformation without associated complex malformations.^[26]

Keywords: split cord malformations; diastematomyelia; tethered cord syndrome; prophylactic surgery.

INTRODUCTION

Split cord malformations (SCM) are rare and complex conditions. In the recent years magnetic resonance imaging (MRI) has renewed the interest in diagnosis, basic, understanding and management of

these anomalies.^[35,36] Now it has become an established cause of 'Tethered cord syndrome'. Several terminologies were in vogue in literature like 'Diastematomyelia' (Olivier) 'Diplomyelia' (Bruce) with poor distinction. Pang et al. have proposed unified theory and coined

them as 'Split cord malformations'^[12,28,29, 30].

Most widely accepted theory about embryogenesis of this complex malformation was originally proposed by Bremer^[6] and subsequently modified by Pang et al.^[28,30] as 'Unified theory of embryogenesis.' The basic error appears to be development and persistence of 'Accessory Neurenteric Canal' (ANC). In the early weeks of gestation Primitive Neurenteric Canal temporarily connects the yolk sac (Endoderm) with amniotic cavity (Ectoderm). Simultaneously an ANC disappears and its persistence will result in a variety of SCMs^[6,28,30]. The persistence of anterior end of this canal will result in intestinal malformations, posterior end, in cutaneous malformations like angiomas, hypertrichosis, dermal sinus or dermoid. Where as the persistence of intermediate part causes the split in the notocord and the neural placode. Division of notocord then leads to formation of hemiverterbrae, bifid, hypertrophic, hyperplastic vertebrae or fusion of vertebral bodies or posterior elements. The division of placode later on drives the formation of two hemicords^[32]. If the mesenchyme surrounding the ANC contain precursor meningeal cells which results in the formation of dura and bony spur within, leading to SCM type I. If meningeal cells are not incorporated then it is not involved in the formation of dural sac. And once ANC disappears it will be transformed into intradural fibrous band situated between the two hemicords, thus constituting SCM type II. The persistence of ANC could occasionally interfere with neurulation process leading to the formation of meningocele or meningomyelocele, compounding these malformations further.^[29,30]

PATIENTS & METHODS

This study was conducted in neurosurgery department, Assiut university hospital over a three-year period between January 2005 and December 2007. Of 90 patients with spinal dysraphism, 10 patients had symptoms and signs that raised the suspicion of occult spinal dysraphism were studied. A detailed clinical examination of each case was done. In addition to the basal laboratory investigations, plain x-ray of the spine and spinal MRI were done for all cases, while entire spine was screened. Also, cranio-spinal MRI was done to all patients for recognition of the possible associated lesions. One patient came with plain CT of spine. SCM was classified into type I and Type II depending on the presence of bony spur or fibrous septum respectively. This typing was based on radiological and operative findings. Seven out of the 10 patients underwent surgical excision of bony spur/fibrous septum depending on the type of anomaly, and repair of associated spinal dysraphia if present. Postoperatively the patients were examined on first and 7th days and then followed up periodically at one month, 3 and 6 months interval. After an average follow-up of 1.5 year the operative results were clinically assessed.

Surgical procedure

The child is positioned in prone with underlying supports. Midline skin incision is made extending above and below the level of the lesion. Subcutaneous tissue and the paraspinal muscles were carefully separated. Usually, the widened interpedicular distance or thick posterior elements will assist as a landmark to locate the spinal segments involved. One should be careful about bony defects and the scoliotic curves during dissection. It is preferable to expose the normal

laminae one level above and below. The laminae can be hyperplastic or fused. Laminectomy is started at the normal level and continued on both sides close to the pedicles in an encircling fashion, protecting the dura and presenting facet joints. The ligaments are gently removed. The central bony spur with its attachment to the lamina is isolated. The two dural tubes are identified and protected. The bony spur is nibbled gradually till its attachment after careful and adequate separation from dura. There can be profuse bleeding from venous plexuses or occasionally from an arterial source. The dura is then opened in the midline at normal level extending down elliptically on either side of the sleeve of the spur joining in the midline caudally. It is important to open the dura medially as close to the split as possible to prevent stenosis. The arachnoid is also opened. The dural sleeve over the spur is then completely excised. The cords are inspected anteriorly and posteriorly and all the surrounding arachnoidal and fibrous bands are removed. After securing hemostasis, the dura is closed posteriorly in the midline converting into a single dural tube. The anterior defect of dura need not be closed. If the filum cannot be accessed in the same field a separate exposure is made at L5-S1 level to cut the filum (Fig.1, 2).

In type-II Malformations, posterior bony elements almost look normal and hence a marker will be necessary. After laminectomy or laminotomy of the required levels the dura is opened in midline. The hemicords are inspected under magnification for the fibrous bands. The fibrous band is usually located ventrally between the hemicords. The band is released from its dural attachment. Other fibrous bands if present should be released. The filum is untethered and the dura is

closed in the midline. In complex malformations, the other associated anomalies are also dealt with appropriately in the same sitting.

RESULTS

Patient's demographics

10 patients included in this study. Their ages varied from 2 months to 19 years, and the average age was 5.7 years approximately, a striking female predominance of the disease, accounting for 70% (7 out of 10) of cases.

Clinical findings

Out of 10 patients 9 (90%) presented with cutaneous stigmata and the tenth one devoid of cutaneous markers presented with meningomyelocele. Of cutaneous stigmata, hypertrichosis was present in 8/9 (88.9%) cases and in one of them it was the only manifestation, port wine hemangioma in one case, subcutaneous lipoma in 2 cases, and hairy pyriform empty sac in one case. Progressive sensory and motor deficit was found in 8/10 cases (80%) and included motor leg weakness in 7 cases (7/8=87.5%), one of them had complete paraplegia with complete anesthesia, awkward gait and frequent stumbling in one case. five patients (5/10=50%) had sphincteric involvement in the form of infrequent wetness or frank urinary incontinence and one of them had frequent urinary tract infection. Neuro-orthopedic syndrome (NOS) constituted 60 % (6/10) and was observed in the form of scoliosis (4/6=66.7%) and congenital talipes equino varus in 2/6 (33.3%). Backache and leg pain were observed in 2 older patients (14&19 years old). All patients were assigned as pure SCM (not associated with meningomyelocele) apart of one who was associated with meningomyelocele and assigned as compound or composite SCM.

Neuroimaging findings

Plain x-ray spine, plain CT spine (in one patient) and MRI revealed vertebral anomalies in all patients and included fused vertebrae, butterfly vertebra, bifid spines, splaying vertebral arches and wide interpedicular distance (Fig.5A-H.). The MRI revealed type I SCM in 9/10 cases (90%) where the cord was splitted by a bony or osteo-cartilagenous spur attached to posterior surface of vertebral body (Fig.3). The spur was either complete (reached to posterior neural arch) in 6/9 or incomplete in 3/9 cases respectively and type II SCM in one patient only where the cord was splitted by a fibrous septum.

In either types of SCM the two hemicords were a symmetrical in all patients (Fig. 4) except one that belongs to type I (Fig.5L, M, N). The level of splitting was lumbar in 9/10 cases opposite L1—L4 and lower dorsal in one patient opposite D10 (type I anomaly). The splitting involved lower dorsal cord in one patient, conus in 8 patients and conus and filum in one patient. Low lying conus was observed in 9/10 cases where it was at L5 (8/9) and sacral one (1/9) but it was normal (opposite L1---L2 space) in one patient. No associated tethering elements except in 2 patients, one of them belongs to type II where intradural lipoma at lower sacral level was identified and the other one belongs to type I due to thick short filum terminale. No cranial or cranio-spinal junction anomalies could be detected.

Surgical findings and outcome

Apart from one who had complete paraplegia with complete anesthesia and urinary incontinence for long period (3 months), surgery was decided and mediated in all patients who had neurological deficit (7/10=70%). While follow up was decided in the remaining

2 patients as one of them only had tuft of hair and the other had congenital talipes equino varus in addition to hairy empty sac which was excised and was terminated at the dorsolumbar fascia.

SCM was found two vertebral levels rostral to the level of meningomyelocele in the type II anomaly patient (compound SCM) while it was found either beneath the cutaneous marker or in close proximity, coinciding with bony anomalies of spina bifida. There was profuse bleeding from venous plexuses or occasionally from an arterial source, also surrounding arachnoidal and fibrous bands were removed. Various Image findings (as described under radiological findings) were confirmed during surgical procedure.

Backache and leg pain was relieved dramatically in older patients (14&19 years). There was no postoperative deterioration and a stable neurological state was maintained. However, seven of eight patients presenting with variable amount of leg weakness and awkward gait, showed gradual recovery in power, and stumbling became infrequent at follow-up of six months to one year. Patients with pre-operative bladder dysfunction showed improvement during follow-up with increase in their dry periods. Neuro-orthopedic syndrome (NOS) became stabilized, and no further improvement or deterioration was noticed in follow-up.

Postoperative complications

Post-operative CSF leak occurred in three patients. Two required re-exploration and repair of dural defect with fascia lata patch whereas one was treated conservatively with lying prone, pressure dressing, covering antibiotics and acetazolamide therapy and they responded well. However, neither meningitis nor

psedeumeningocele was faced. No mortality among our patients.

DISCUSSION

SCM is not an uncommon finding in spinal dysraphism.^[2,7,10,11,18] In the series of Kumar Raj et al^[24], out of 138 patients with spinal dysraphism SCM constituted 46 of all, reflecting an incidence of SCM to be 33 % (46/138). Campbell et al^[7] found 36 cases of SCM in 100 infants of open spinal dysraphism while Emery & Lendon^[10] reported 78 cases amongst 100 spinal dysraphic patients. In the present study, of 90 patients with spinal dysraphism SCM constituted 10 of all, reflecting an incidence of SCM to be 11.1% which is far less than reported by others and this may be related to sample size of the patients.

Although majority of SCMs are diagnosed in infancy, symptoms can manifest at any age. In Venkataramana N K series which included 38 patients, the majority became symptomatic by second decade^[37]. In the current series 7/10 patients presented in infancy period (2 months to 4 years), while the other 3 patients presented in childhood period (11-19 years) this contradiction may be related to the sample size. SCM is more common in females according to western literature^[3,18,29], likewise in this series (70%) were females but male predominance is seen in Indian subcontinent^[20,21,22,24].

Clinically, The skin manifestations in occult dysraphism represent minor aberrations in the development of the surface ectoderm, brought about by the adverse influences of a dorsal endomesenchymal tract, but these changes are overshadowed by changes in the surface ectoderm in case of an associated meningocele.^[21,25,30,33,34] The skin markers are quite frequent in SCM. In the series of Kumar Raj et al 2005, the incidence of

cutaneous marker was 90% in group II cases (SCM without meningocele (MMC)) as compared to only 10.4% in group I (SCM with MMC) signifying a low incidence of cutaneous markers in SCM presenting with meningocele. It is an important observation because these cases of SCM with MMC have a higher chance of missed diagnosis without awareness^[24]. Amongst these markers the hypertrichosis was the commonest, being present in 50% of the cases of group II. In the series of Kumar Raj et al 2005. The other markers like capillary hemangiomas and dermal sinus were seen in group II only and not in group I. This agrees to the consensus of incidence of cutaneous markers in occult dysraphism varying between 20 to 75%.^[8,9,14,16,17,19,30] In the present series, the incidence of cutaneous stigmata was 90% and the only patient devoid of cutaneous markers was associated with MMC. Hypertrichosis was the hallmark amongst the cutaneous stigmata where was present in 8/9 (88.9%), followed by subcutaneous lipoma, port-wine hemangioma and hairy skin tag (sac like) in this order of descending frequency and thus we are in accordance with others.

The neurological symptoms can vary from pain, bowel and bladder dysfunction, motor or sensory deficits, gait disturbance and trophic ulcers. In the current series the neurological signs included motor weakness in 7/8 cases and ranged from limb/s weakness, foot drooping, awkward gait in 6/8 to frank paralysis with complete anesthesia in 1/8 patient, also bladder and/or bowel dysfunction in 5/10 patients. In older children pain (lumbago or lumbosciatic) and paraesthesias are more common^[1,4,13]. This occurred in 2 of my patients. The symptoms can be precipitated by trivial

fall in an otherwise asymptomatic child. In the series of Kumar Raj et al.^[24], they observed that the incidence of neurological deficit was more significant in group I (SCM with MMC) as compared to group II (SCM without MMC). Following surgery the improvement in neurological functions were more marked in group II cases compared to group I. Also, the onset of neurological deficits seems to be early in type I malformation in comparison to type II^[37]. In the present study I could not do this comparison as only one patient had SCM with MMC. However, the incidence of neurological deficits were far common among type I at the time of diagnosis. In the series of Kumar Raj et al.^[24], the incidence of neuro-orthopedic syndrome was 66.6% in group I and 96% in group II patients, this was in accordance with other reported series^[2,30]. In the current series the incidence of neuro-orthopedic syndrome was 60% that is in the range of literature review.

Plain X-ray, CT scan, CT myelography, and MRI are the imaging modalities used to diagnose this anomaly. The radiological abnormalities have been vividly explained by Neuhauser et al.^[27] Although some of the abnormalities can be recognized on plain X-ray, as occurred in my series where plain X-ray revealed vertebral anomalies in all patients and included fused vertebrae, butterfly vertebra, bifid spines, splaying vertebral arches and wide interpedicular distance, high resolution CT scan is sometimes necessary in identifying the spur^[37], as observed in axial CT of one of my patients. MRI has become the procedure of choice, to demonstrate the type of SCM, dural sac, the spur, location of filum terminale and other associated abnormalities like hydrosyringomyelia, dermal sinus, dermoid, epidermoid and/or lipoma. In my series MRI

verified precisely various issues of SCM in all patients. It is logical to advocate the screening of entire spine with MRI in every spina bifida child. If SCM is found in association with MMC, it should be treated simultaneously to circumvent its deleterious effect on the developing cord^[30].

The type I SCM is reported to have higher incidence of 75% in comparison 25% of type II in literature.^[11,18,20] Also, a higher incidence of type I SCM is reported in cases associated with MMC by Pang et al.^[29] and Ersahin et al.^[11]. Despite the small sample of our patients (10 patients), the same phenomenon was found where Type I SCM was encountered in 9 of 10 (90%), while type II was seen in one case but was associated with MMC. It was interesting in the series of Kumar Raj et al.^[24] as they noted that 14 of 19 split cord malformations were located one or two vertebral levels rostral to myelomeningocele and remaining 5 were found at the level of meningomyelocele itself, but It was obvious that no SCM was found below the level of MMC. In my series where MMC was associated in one patient, SCM was found two vertebral levels rostral to the level of meningomyelocele while it was found either beneath the cutaneous marker or in close proximity, coinciding with bony anomalies of spina bifida in the other patients. The spur can be antero-posterior usually attached to posterior surface of the vertebral body. Rarely it can have dorsal attachment to the spine^[37]. However, in my series the spur was complete (attached to the posterior neural arche) in 6/9 cases and incomplete in 3/9 cases. The spur can divide the canal into two symmetrical halves or can be slanting in the axial plane dividing the canal asymmetrically. In such situations one of the hemicords can be hypoplastic^[37].

Harwood-Nash et al.^[15] have reported such asymmetry in 50%. In my series the two hemicords were asymmetrical in all cases except in one patient. In the majority spur is located at lumbar and less frequently in lower thoracic, cervical and upper thoracic regions, respectively. Double level spurs have also been reported occasionally. Venkataramana NK^[37] has encountered two level spurs in one child at lumbar and lower thoracic regions. In my series the spur was located at lumbar region in 9/10 cases (opposite L1-L4) and lower dorsal in one patient (opposite D10) and the involved cord segment was lower dorsal cord in one patient, conus in 8/10 cases and conus and filum in one patient that is in accordance with what reported in literature. Incidence of low lying cord in SCM with MMC was reported 30% by Iskander BJ et al.^[18] whereas in pure SCM it was found in 40.5% of cases as reported by Ersahin et al.^[11]. In the series of Kumar Raj et al.^[24], it was noticed in 47.3%, and 66.6% of group I (SCM with MMC) and group II (SCM without MMC) patients respectively, and Intraspinial lipoma was the significant finding in the their study, both in group I, 4/19 (21.0%), and group II 7/27 (25.9%). Barring two patients in group I, in whom lipoma was rostral to the split cord; all the patients had distal lipomas in both the groups. This coincides with the reported incidence of terminal lipomas by Pang^[30] who encountered 6 lipomas in total 37 cases of SCM. Other associated lesions with SCM are dermoid, epidermoid, arachnoid cyst and neurenteric cyst, as reported in literature^[20,22,23]. In my series the same phenomenon has been documented where low lying cord was observed in 9/10 cases (90%), one of them had MMC and the only associated tethering element was terminal lipoma which was distal to the split cord in one

patient and thick filum in another patient.

In the literature the reported range of associated cranial and craniospinal junction anomalies with SCMs is 25 to 40%, amongst those are hydrosyrinx, chiari malformation, and hydrocephalus^[17,18,24,34]. No cranial or cranio-spinal junction anomalies could be detected in the present study which may be related to the small sample of the patients.

Time of surgery for SCM is a matter of debate, while there is universal acceptance for surgery as long as progressive neurological deficits are present at least to stabilize the clinical status notably in pure anomaly and rational decisions are necessary in children who are neurologically compromised and harbor complex anomalies; a lot of controversy in non symptomatic patients. Since majority become symptomatic by second decade, and eventually deteriorate in their neurological function; as the neurological deficits once occur are not completely reversible, and the children with no neurological deficits maintain steady neurological state postoperatively, several authors recommend prophylactic surgery. On the other hand, Jamil et al. consider that the deficit is not always progressive and surgery can only be indicated in patients with progressive neurological deficits.^[1,5,28,29,30,31,37] In my series Surgery was decided and mediated in all patients who had neurological deficit (8/10=80%) except one who had complete paraplegia with complete anesthesia and urinary incontinence for long period (3 months). Prophylactic surgery not applied in the other 2 patients as their parents prefer to follow –up and no deterioration was noticed in follow-up.

Venkataramana NK^[37] and I encountered improvement of pain, no

postoperative deterioration and a stable neurological state was maintained however, in the present study gradual recovery in motor deficit and bladder function at follow-up of six months to one year. Neuro-orthopedic syndrome (NOS) became stabilized, and no further improvement or deterioration was noticed in follow-up. No mortality, and CSF leak was manageable.

CONCLUSION

The SCMs are rare complex forms of occult dysraphism. In type I cord is anchored by an extra-dural bony spur

and/or short, thick filum terminale. In type II tethering is caused by fibrous band, which anchors, hemicords to the anterior dura. The MRI is the choice of investigation, but high resolution CT can be complimentary. Hypertrichosis is the commonest marker to the clinical diagnosis. Surgery and detethering should be considered prophylactically after diagnosis and in all symptomatic children. Rational decisions are necessary in children who are neurologically compromised and harbor complex anomalies. Outcome is very good in children with isolated split cord malformation without associated complex malformations^[26].



Fig.1: Hypertrichosis in the lumbar region in a female baby of 2 years. It was the only manifestation. MRI and operative findings proved to be SCM type I.

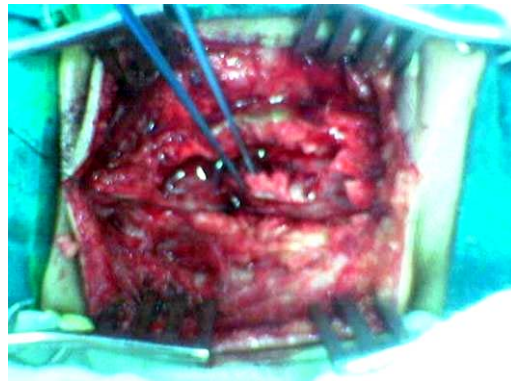


Fig. 2: intra operative photo showing Type I SCM where the cord is splitted and then reunited with the bipolar forceps grasping the bony spur within the cleft. Note the two hemicord are asymmetrical. (The same patient in fig. 1)

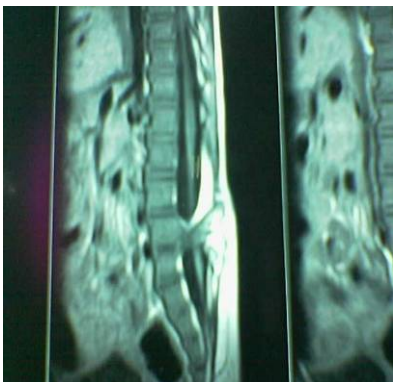


Fig.3: MRI T1W saggital cut revealing a bony spur attached to the posterior surface of L4, note it is complete (reaching posterior neural arch). The same patient in fig.1 and 2.

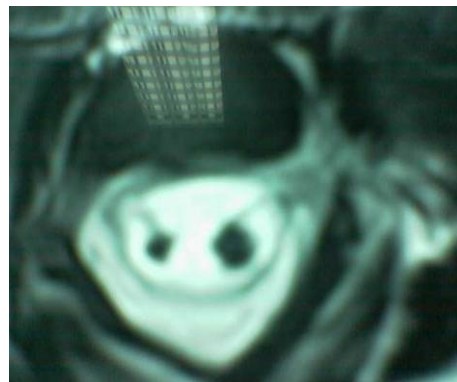


Fig. 4: the same patient in Fig.1, 2, 3 axial cut T2W. type I SCM with a symmetrical two hemicords.

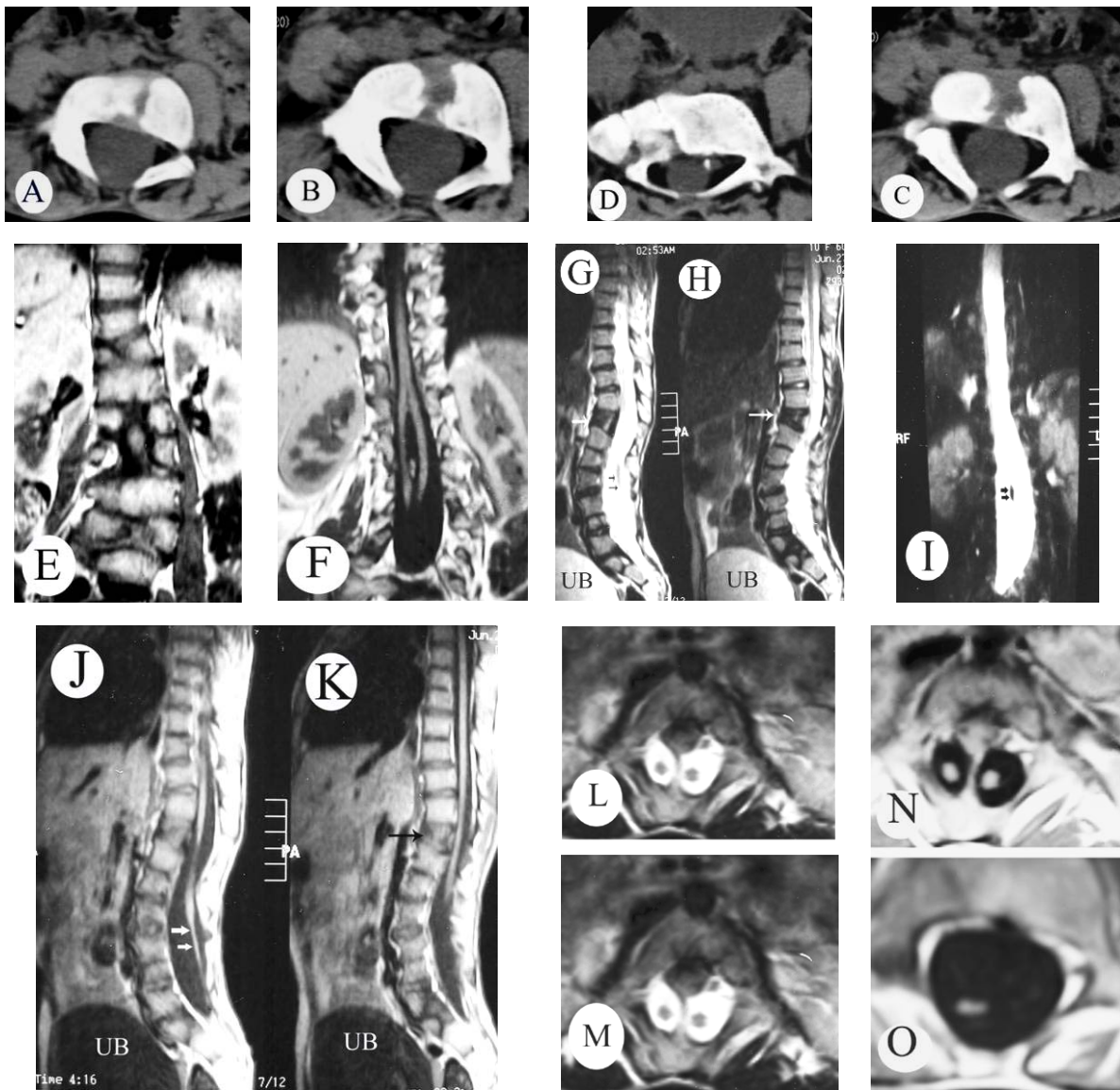


Fig. (5): Eleven years old female presenting with lower limbs weakness, mild scoliosis. Figs [A,B,C,D] axial CT scans at L4 & L5 levels, butterfly vertebra and small bony spur seen. [E-F] Coronal T1 MRI, & [G&H] Sagittal T2 images revealed the vertebral anomalies (arrows) and the scoliosis, also showing the splitted cord with reunion after passing the small centric spur. [I] coronal MR myelography revealed the tethered cord and bony spur. [J&K] Sagittal T1 showing well the tethered cord (white arrows). The two hemicords seen well delineated at axial T1 & T2 W sequences [L,M,N,O] and reunion seen in the lower levels.

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