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AIM AND SCOPE

World Journal of Radiology (World J Radiol, WJR, online ISSN 1949-8470, DOI: 10.4329) is a peer-reviewed open access academic journal that aims to guide clinical practice and improve diagnostic and therapeutic skills of clinicians.

WJR covers topics concerning diagnostic radiology, radiation oncology, radiologic physics, neuroradiology, nuclear radiology, pediatric radiology, vascular/interventional radiology, medical imaging achieved by various modalities and related methods analysis. The current columns of WJR include editorial, frontier, diagnostic advances, therapeutics advances, field of vision, mini-reviews, review, topic highlight, medical ethics, original articles, case report, clinical case conference (clinicopathological conference), and autobiography.

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INDEXING/ABSTRACTING

World Journal of Radiology is now indexed in PubMed, PubMed Central, and Emerging Sources Citation Index (Web of Science).

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PUBLICATION DATE

April 28, 2017

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http://www.f6publishing.com
Imaging spectrum of spinal dysraphism on magnetic resonance: A pictorial review

Jyoti Kumar, Muhammed Afsal, Anju Garg

Abstract
Congenital malformations of spine and spinal cord are collectively termed as spinal dysraphism. It includes a heterogeneous group of anomalies which result from faulty closure of midline structures during development.

INTRODUCTION
Congenital malformations of spine and spinal cord are collectively termed as spinal dysraphism. It includes
Kumar J et al. MRI of spinal dysraphism

a heterogeneous group of anomalies resulting from incomplete midline closure of osseous, mesenchymal and nervous tissue. Most of these conditions are diagnosed at or soon after birth, but some are discovered late in childhood or in adulthood because of absence of clinical manifestations. Magnetic resonance imaging (MRI) is the imaging modality of choice for diagnosing spinal dysraphism because of its superior soft tissue characterisation and multiparametric imaging capabilities. The purpose of this article is to review the normal development of spinal cord and MRI features of spinal dysraphism with clinico-embryologic and radiological correlation.

EMBRYOLOGY

Development of spinal cord occurs during early embryogenesis (between 2-6 wk of gestation) in three main stages - gastrulation, primary neurulation and secondary neurulation. During the stage of gastrulation, the bilaminar embryonic disc composed of ectoderm and endoderm is converted to a trilaminar disc by the formation of mesoderm.

Figure 1 Gastrulation. During gastrulation, the bilaminar embryonic disc is converted to a trilaminar disc by the formation of mesoderm. There is rapid division of cells in embryonic disc which then detach and migrate between endoderm and ectoderm to form mesoderm. Notochord later develops from mesoderm.

Notochord which is formed from midline mesoderm interacts with overlying ectoderm resulting in the formation of neuroectoderm and neural plate. Primary neurulation begins with the formation of neural plate and ends with the closure of caudal end of neural plate (Figure 2). A small depression develops along the central axis of neural plate to form neural groove and neural folds are formed on both sides of the neural groove. The neural plate then bends and neural folds fuse together converting the linear neural plate into a cylindrical neural tube. Closure of neural groove proceeds bidirectionally with the cephalic end (anterior or rostral neuropore) closing on day 25 and the caudal end (posterior or caudal neuropore) closing on day 27 or 28.

During secondary neurulation there is formation of caudal cell mass composed of undifferentiated pluripotent cells from the caudal end of neural tube and notochord distal to caudal neuropore. Neurons and vacuoles develop within the caudal cell mass. Vacuoles then coalesce and eventually connect to the central canal by the process called cavitation. Finally the cells of caudal cell mass undergo retrograde differentiation during which cells undergo programmed cell death or apoptosis to form conus medullaris, filum terminale and ventriculus terminalis.

Various disorders due to defective primary neurulation include open spinal dysraphism (OSD), closed spinal dysraphism (CSD) and dorsal dermal sinus while defects during secondary neurulation results in filar lipoma, tight filum terminale, caudal agenesis and sacrococcygeal teratoma. Defective development of notochord results in diastematomyelia, neurenteric cyst, caudal agenesis and segmental spinal dysgenesis.

CLASSIFICATION

Table 1 Classification of spinal dysraphisms

| Open spinal dysraphisms          |
|---------------------------------|
| Myelomeningocele               |
| Myelocele                       |
| Hemimyelomeningocele           |
| Hemimyelocele                   |
| Closed spinal dysraphisms       |
| With subcutaneous mass          |
| Lipomyelomeningocele            |
| Lipomyelocele                   |
| Terminal myelocystocele         |
| Meningocele                     |
| Myelocystocele                  |
| Without subcutaneous mass       |
| Simple dysraphic states         |
| Intradural lipoma               |
| Filar lipoma                    |
| Tight filum terminale           |
| Persistent terminal ventricle   |
| Dermal sinus                    |
| Complex dysraphic states        |
| Dorsal enteric fistula          |
| Neuroenteric cyst               |
| Diastematomyelia                |
| Caudal agenesis                 |
| Segmental spinal dysgenesis     |

OSDs

Myelomeningocele and myelocele

OSDs result from faulty primary neurulation due to defective closure of the neural tube. About 98.8% of
all OSDs are constituted by Myelomeningocele (MMC) where both the neural placode and meningeal lining protrude through the bony and cutaneous defect in the midline[13]. OSDs are commonly diagnosed clinically as the neonate presents with a midline reddish exposed neural placode and immediate surgical repair is usually done, so imaging studies are not always performed. It usually involves the lower lumbar and sacral regions (98%) and is rare in cervical and upper thoracic spine, probably because the lesion in these areas are more severe leading to fetal demise[4,13].

In MMC, the protruding neural placode extends beyond the skin surface as there is enlargement of the adjacent subarachnoid space (Figure 5)[9]. This help to distinguish MMC from the far rarer myelocele, where the placode is flush with the skin surface (Figures 6 and 7). About 80% of myelocoele and MMC have associated hydrocephalus and 100% patients have Chiari II malformation which involve cerebellum, brainstem, skull base, spine and spinal column (Figure 8)[14,15]. Studies have shown that defective neural tube closure resulting in abnormal drainage of CSF and hence decompression of the primitive ventricular system resulting in various manifestations of Chiari II malformation[14,15].

Advancements in prenatal diagnosis permit diagnosis of neural tube defects in fetus as early as first trimester, and now most of the cases of MMC are diagnosed prenatally during screening sonography. Although sonography is the modality of choice for screening fetus for any gross congenital anomalies, MRI is being increasingly used for prenatal evaluation of CNS anomalies with the advent of faster MR sequences (Figure 9)[16,17].

Prenatal diagnosis and developments in fetal surgery has made possible inutero repair of the neural tube defects which can arrest the development of other malformations developing secondary to abnormal tube closure. The Management of Myelomeningocele Study trial, a prospective randomized study done in United States has shown that fetal surgery for MMC in second trimester preserves neurologic function, reverses the changes of Chiari II malformation and reduces the need for postnatal ventriculoperitoneal shunt[18-21].

Hemimyelomingingocele and hemimyelocele
These conditions are extremely rare and are caused by defective gastrulation and primary neurulation[22]. Diastematomyelia is a common association with OSDs but only when one of the hemicords shows defective neurulation, the malformation is labelled hemimyel(meningo)cele[23]. Here one of the two hemicords exhibits a myelomeningocele or myelocele while the other hemicord can be normal or is tethered.

CSDS WITH SUBCUTANEOUS MASS

Lipomas with dural defect - lipomyelocele and lipomyelomingingocele
Lipomyelocele and Lipomyelomingingocele result from defective primary neurulation where there is premature focal disjunction of cutaneous ectoderm and neuroectoderm allowing mesenchyme to enter the neural tube. This mesenchyme later forms the lipo-matous tissue for unknown reasons[23,24]. Clinically they are characterized by the presence of subcutaneous fatty mass lesion above the intergluteal line which may extend to buttocks. Sagittal T1 WI images show high intensity fat on the dorsal aspect of the placode which is continuous with the adjacent subcutaneous fat. T1 weighted fat saturated images show suppression of fat signal. Lipomyelocele and Lipomyelomingingocele are differentiated based on the position of neural placode - lipoma interface (Figure 10). It lies within or at the edge of the spinal canal in lipomyelocele and outside the spinal canal in lipomyelomingingocele (Figure 11)[21].
In lipomyelomeningocele there is expansion of subarachnoid space anterior to the cord pushing the neural placode posteriorly to lie outside the boundaries of spinal canal. In hemilipomyelomeningocele or hemilipomyelocele there is associated diastematomyelia with one of the hemicord showing lipomyelocoele or lipomyelomeningocele respectively (Figure 12).

**Meningocele**

Meningocele refers to herniation of CSF filled sac lined by dura and arachnoid mater. The exact embryogenesis is unknown but is thought to be caused by ballooning of meninges due to CSF pulsation. By definition, spinal cord should not be seen within the meningocele but may be seen tethered to its neck. Meningocele may contain nerve roots and or filum terminale which usually appear hypertrophied.

Posterior meningocele is due to herniation of meningeal lining through posterior spina bifida (Figure 13). It is usually lumbosacral in location but can be seen in other locations also. Anterior meningocele is almost always presacral in location.²⁵

**Terminal myelocystocele**

Terminal myelocystocele involves herniation of a
dilated terminal central canal forming terminal syringohydronephomyelia (syringocele) through a posterior vertebral defect into an expanded CSF filled dural sheath (meningocele). It results from defective secondary neurulation which affects the CSF flow dynamics. The inner terminal syrinx communicates with the central canal of the spinal cord and the outer meningocele is continuous with the spinal subarachnoid space. The syringocele and meningocele usually do not communicate with each other.

**CSDS WITHOUT SUBCUTANEOUS MASS**

**Simple dysraphic states**

These are a heterogeneous group of conditions which arise due to abnormalities of primary and secondary neurulation and are the most common type of spinal dysraphism seen in older children. Simple dysraphic states include intradural lipoma, filar lipoma, tight filum terminale, dermal sinus and persistent terminal ventricle.

**Intradural lipoma**

It is a midline lipoma located in the groove of unopposed neural placode in its dorsal surface within an intact dural sac. The intact dura help to differentiate this from lipomyelomeningocele and lipomyelomeningocele. They are usually seen in lumbosacral region and are associated with tethered cord syndrome. Large lipomas may cause cord displacement. On MRI lipomas follow the signal intensity of subcutaneous fat on all sequences (Figure 14).  

**Filar lipoma**

Filar lipoma is an abnormality of secondary neurulation which shows fibrolipomatous thickening of the filum terminale. On imaging filar lipoma appears hyperintense on T1 and T2 weighted images within a thickened filum terminale (Figure 15). One point five percent to 5% of normal adult population may show fat within filum terminale on MRI and hence the finding is considered a normal variant (Figure 16) unless it is associated with tethered cord syndrome. Tethered cord syndrome is characterized clinically by progressive neurological deficit and on imaging, there is low lying conus medullaris with a short thick filum terminale which is tethered to dural sac.

**Tight filum terminale**

It is characterized by shortening and hypertrophy of filum terminale which cause tethering of cord and impairs the ascent of conus medullaris. Embryologically the defect lies in retrogressive differentiation during secondary neurulation.

On imaging it is characterized by a thick filum terminale (thickness measuring > 2 mm) and a low lying conus medullaris - below L2 vertebral body (Figure 17). It is usually seen in association with other malformations and isolated cases are rare.

**Dermal sinus**

It is an epithelial lined fistulous communication between CNS or its meningeal covering and skin. It results from focal incomplete disjunction between neuroectoderm and cutaneous ectoderm. Clinically a midline dimple or ostium is found on the cutaneous surface and is commonly associated with cutaneous stigmata of underlying occult spinal dysraphism like hairy nevus, hemangioma or hyperpigmentation. The tract then ascends and opens into spinal canal (Figure 18). Dermal sinus may be associated with intraspinal dermoids or epidermoids which show variable imaging findings depending on their contents. Dermoids usually appear hyperintense on both T1 and T2 weighted images while epidermoids are hypointense on T1 weighted and hyperintense on T2 weighted images. CNS infection is a common complication because of fistulous communication and hence these cases require early surgical repair (Figure 19).

**Persistent terminal ventricle**

Terminal ventricle is a small, ependyma lined cavity within conus medullaris (Figures 13 and 17). Embryologically incomplete regression of terminal ventricle during the stage of secondary neurulation is responsible for the condition. Location just above filum terminale helps to differentiate it from hydromyelia and lack of enhancement is the differentiating feature from intramedullary tumors.

**COMPLEX DYRAPHIC STATES**

Any abnormality occurring at the time of gastrulation affects the spinal cord and various other structures which are derived from notochord resulting in complex anomalies. Most of these abnormalities are covered by skin and subcutaneous masses are absent. On the basis of their embryogenesis complex dysraphic states are divided into two subtypes - disorders of midline
notochordal integration and disorders of notochordal formation.

**Disorders of midline notochordal integration**
The process of fusion of paired notochordal anlagen to form a single midline notochordal process is called midline notochordal integration\(^3\). Any abnormality at this stage results in longitudinal splitting of spinal cord. Most important entities in this group are neurenteric cyst and diastematomyelia.

**Neurenteric cyst:** Most severe form of disorder of midline notochordal integration is dorsal enteric fistula - a fistulous communication between skin surface and bowel - which is an extremely rare condition. Neurenteric cyst is a localized form of dorsal enteric fistula and is seen anterior to spinal cord with adjacent vertebral anomalies. These cysts are typically seen in extramedullary intradural compartment of cervicothoracic spine, however may be seen in other locations too\(^37\). On MRI, neurenteric cysts usually appear iso- to hyperintense to CSF on both T1 and T2 weighted images due to high protein content and show absent contrast enhancement (Figure 20)\(^38,39\).

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**Figure 8** Cervico-dorsal myelomeningocele with Chiari II malformation. T2 weighted sagittal (A), T1 weighted sagittal (B) and T2 weighted axial (C) images of cervicodorsal spine showing myelomeningocele. There is posterior herniation of a CSF filled sac (dashed arrows) containing nerve fibers (black arrow). Spinal cord show kinking at the same level and dilated central canal (white arrow) suggestive of syringohydromyelia. There is no overlying skin cover. Sagittal T1 weighted (D) and axial T2 weighted (E and F) images of brain shows herniation of cerebellar tonsils (black arrow) with tectal beaking (white arrow). Fourth ventricle is elongated and tubular shaped. There is colpocephaly suggestive of corpus callosal agenesis (in E) and interdigitating gyri (dashed arrow in F) suggestive of fenestrated falx.

**Figure 9** Fetal Myelomeningocele with Chiari II malformation. Axial (A) and sagittal (B) T2 weighted images of lumbar spine of fetus showing splaying of posterior elements of lumbar vertebrae with herniation of spinal canal contents (black arrow) suggestive of MMC. Axial T2 weighted image of brain (C) shows associated hydrocephalus. Hydrocephalus in the setting of MMC in a fetus is considered highly suggestive of Chiari II malformation. MMC: Myelomeningocele.
Diastematomyelia: It is the most common form of defective midline notochordal integration. Due to defective midline integration there are two notochordal processes each of which induces formation of separate neural plate with intervening primitive streak tissue. The development of the primitive streak tissue decides the type of diastematomyelia. In type I diastematomyelia the intervening primitive streak develops into bone.
or cartilage, resulting in two hemicords in different dural sacs separated by an osteocartilaginous septum (Figure 12). In type II diastematomyelia, the primitive streak is reabsorbed or forms a fibrous septum with the hemicords lying within the same dural sac (Figure 21)\(^\text{[40]}\). Diastematomyelia is commonly associated with vertebral anomalies and hydromyelia. A high lying hairy tuft over a child’s back is a reliable indicator for underlying diastematomyelia\(^\text{[41]}\).

**Disorders of notochordal formation**

Apoptosis or programmed cell death is an important process occurring during different steps of embryogenesis. Abnormal apoptosis results in disorders of notochord formation and these disorders include caudal agenesis and segmental spinal dysgenesis\(^\text{[29]}\).

**Caudal agenesis:** It is characterized by partial or total agenesis of spinal column and is commonly associated with genital anomalies, anal imperforation, pulmonary hypoplasia, renal aplasia or dysplasia and limb abnormalities. Caudal agenesis (CA) is broadly divided into two types.

In type I CA both caudal cell mass and notochord formation is affected, resulting in high position (most commonly at the level of D12 vertebra) and abnormal termination of conus medullaris. There is accompanying varying degree of vertebral aplasia, with the last vertebra as L5 through S2 in majority of patients.

In type II CA there is abnormal development of

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**Figure 13** Posterior meningocele with persistent terminal ventricle. T2 weighted sagittal (A) and axial (B) images of lumbosacral spine showing posterior meningocele and persistent terminal ventricle. There is herniation of a CSF filled sac (arrowhead) through a posterior spin a bifida (dashed arrow) consistent with posterior meningocele. No neural tissue is noted within the CSF sac. There is another CSF attenuation lesion in conus medullaris (black arrow) suggestive of persistent terminal ventricle. The filum terminale is short and thick (white arrow) with low lying spinal cord suggestive of tight filum terminale. CSF: Cerebrospinal fluid.

**Figure 14** Dural lipoma. T2 weighted sagittal (A), T1 weighted sagittal (B) and T2 weighted axial (C) images of cervico-dorsal spine showing a T1 and T2 hyperintense lesion in the spinal canal (black arrow) causing compression and anterior displacement of the spinal cord (white arrow). The lesion is isointense to subcutaneous fat on all sequences.

**Figure 15** Filar lipoma. T2 weighted sagittal (A) and T1 weighted axial images of lumbosacral spine in a case of filar lipoma. The lesion (black arrow) appears hyperintense on both T1 and T2 weighted images and follows the signal of subcutaneous fat on all sequences. Filum terminale is thickened and is tethered (white arrow).
only caudal cell mass with unaffected true notochord formation. Hence, there is defective secondary neurulation with normal primary neurulation. As a result only the most caudal part of conus medullaris is absent in type II CA (Figure 22). Vertebral dysgenesis is less severe in these cases and these patients present with tethered cord syndrome as the conus in these cases is stretched and tethered\[42,43\].

**Segmental spinal dysgenesis:** This is an extremely rare condition characterized by: (1) segmental agenesis or dysgenesis of lumbar or thoacolumbar spine; (2) segmental abnormality of spinal cord or nerve roots; (3) congenital paresis or paraplegia; and (4) congenital lower limb deformities. This occurs due to notochordal abnormality occurring during gastrulation which involves an intermediate segment of notochord\[29,44\].

**SACROCOCCYGEAL TERATOMA**

Sacrococcygeal teratoma, although a tumor, needs special mentioning as it develops from the pleuripotent cells of caudal cell mass. It is the most common tumor of fetus and new born and commonly present as a large complex solid cystic mass caudal to coccyx. Most of the teratomas are benign and contains derivatives from all three germ layers. Based on the presence of external and internal components they are divided into four types - type I: Primarily external, type II: Equal external and internal portions, type III: Primarily internal and type IV: Entirely internal.

On MRI, sacrococcygeal teratoma has variable signal on T1 and T2 weighted images depending on the internal contents (fat, soft tissue, fluid, calcium). On post contrast images, there is heterogeneous enhancement of the solid portion (Figure 23)\[45,46\]. This may also be picked up on antenatal scan where MRI can accurately depict its extension into the pelvis and its mass effect on pelvic organs (Figure 24).

**POST-OPERATIVE IMAGING**

Surgery is the treatment of choice for spinal dysraphism and surgery includes closure of the neural tube defect and detethering followed by lifelong supportive care and follow up. During follow-up, worsening of symptoms
should be looked for and MRI should be done at the earliest suspicion. During post-operative imaging, various immediate and late complications of spinal surgery like wound infection, shunt infection, wound dehiscence, cerebrospinal fluid leak, problems related to kyphectomy, adhesions with retethering or dermoid formation should be looked for (Figure 25). Retethering is due to post-surgical fibrosis resulting in tethering of cauda equine fibres. MRI may be done in prone position to look for CSF dorsal to the conus for ruling out retethering\textsuperscript{47}.

**CONCLUSION**

Imaging of spinal dysraphism may appear complicated as it is a group of diverse conditions which can have

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**Figure 19** Infected dermal sinus with intraspinal dermoid. T1 weighted sagittal image (A) showing a dermal sinus (white arrow) with an intraspinal lesion (black arrow) showing mildly hyperintense signal with expansion of spinal cord. On T2 weighted sagittal (B) and STIR coronal (C) images the intraspinal lesion appears hyperintense with well defined hypointense rim. On post contrast image (D) there is peripheral enhancement of the intraspinal lesion and the tract.

**Figure 20** Neuroenteric cyst. T2 weighted images of cervicodorsal spine in sagittal (A), axial (B) and coronal (C) planes show a bilobed lesion with both extraspinal (black arrow) and intraspinal extramedullary component (white arrow). The communication between them (arrowhead) is better appreciated in the coronal plane (C).

**Figure 21** Diastematomyelia II. Axial T2 weighted (A) and T1 weighted (B) scan of lumbar spine showing two hemicords. No intervening bony septum was noted. The hemicord on the right shows dilated central canal or syringohydromyelia (arrow).
variable imaging appearance. A systematic approach and correlation with neuroradiological, clinical and develop-
mental data helps in making the correct diagnosis.

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