Magnetic resonance imaging in the differential diagnosis of infectious and inflammatory conus medullaris lesions

The conus medullaris is frequently affected by inflammatory and infectious lesions which many times are hardly differentiated because of the similarity of their clinical history and physical examination among the different etiologies. Magnetic resonance imaging presents high sensitivity in the detection of these lesions and plays a relevant role in the diagnosis as well as in the effective control of the condition. The present pictorial essay with selected cases from the archives of the authors’ institution is aimed at demonstrating imaging findings which might help in the diagnosis of a specific etiology amongst inflammatory an infectious conditions and in the differentiation with diseases of neoplastic and vascular etiologies, for example. Findings such as enhancement pattern, presence of cysts, edema and involvement of other regions of the central nervous system are important for this differentiation, and may define a specific etiology as associated with clinical and laboratory tests findings.

Keywords: Magnetic resonance imaging; Myelitis; Transverse myelitis; Schistosomiasis.

INTRODUCTION

The conus medullaris is a site of frequent involvement by lesions of inflammatory or infectious etiology, generally established by hematogenous spread, but also disseminated through the cerebrospinal fluid or by meningeal or vertebral extension. Cases of neoplastic and vascular etiologies may present similar clinical signs. Many times, the clinical history and physical examination may be not sufficient to determine a specific cause because of interposition of symptoms. Magnetic resonance imaging (MRI) plays a relevant role in the differentiation as well as in the evolutive follow-up of such lesions. Such imaging method is highly sensitive and many times allows the differentiation with of other etiologies such as neoplasms and vascular diseases. However the specificity of this method is limited for differentiating between inflammatory etiologies. But some imaging findings in association with clinical and laboratory findings may lead to a more accurate diagnosis. The knowledge of such conditions and their presentations is fundamental for radiologists.

The present study is aimed at approaching the findings related to conus medullaris lesions of inflammatory and infectious etiologies. For such purpose, the authors have selected images of individual cases in the files of their institution, demonstrating findings which may be useful to differentiate among the several etiologies.

MEDULLARY SCHISTOSOMIASIS

Schistosomiasis is endemic in Brazil. Its most frequent ectopic presentation – medullary schistosomiasis — is considered the most common cause of non-traumatic and
non-neoplastic myelitis\textsuperscript{4}). It is believed that the involvement of the central nervous system (CNS) occurs by means of retrograde flow through the Batson venous plexus, which is valveless. \textit{Schistosoma mansoni} eggs are large and spiculated, impairing their progression through the CNS. For that reason, the conus medullaris is the most frequent presentation site and encephalic involvement is rare with this species\textsuperscript{5}). The clinical picture is that of acute/subacute myelopathy, characterized by pain and weakness of the lower limbs, loss of bowel and bladder sphincter control, besides changes in sensitive and sexual reflexes\textsuperscript{4–6}). MRI generally identifies a conus medullaris expansion, with hypersignal on T1-weighted, hypopersignal on T2-weighted images and contrast-enhancement at the granuloma site. This finding may simulate a neoplastic lesion. A linear and nodular enhancement pattern with an “arborized” appearance is considered to be characteristic, and although it is not present in all cases, is a strong indication for the diagnosis of neuroschistosomiasis (Figure 1)\textsuperscript{6}).

**NEUROCYSTICERCOSIS**

Cysticercosis is the most common parasitic infection of the CNS. The intracranial presentation is most frequently found. Verterbral canal involvement is rare, representing only 2\% to 5\% of total neurocysticercosis cases. Leptomeningal neurocysticercosis is the most common presentation, affecting the subarachnoid space by means of intracranial migration. Intramedullary involvement is rarely observed and occurs generally in the thoracic segment\textsuperscript{5,7}).

In medullary neurocysticercosis, the clinical picture depends on the compression and edema mechanisms, as well as on the inflammatory response. It may be either asymptomatic or present signs and symptoms of myelopathy, including radiating pain, flaccid or spastic paresthesia, and neurogenic bladder\textsuperscript{5,7,8}).

MRI is the method of choice for the study of medullary neurocysticercosis and shows cysts with similar signal to that of the cephalorachidian fluid, with hypersignal on T1-weighted and hypopersignal on T2-weighted images (Figure 2). The scolex is represented by an eccentric nodule near the cyst wall, and its identification is diagnostic. In cysts with some degree of degeneration, peripheral postcontrast enhancement can be observed\textsuperscript{7,8}).

**TUBERCULOSIS**

Involvement of the CNS occurs in approximately 10–15\% of all infections by tuberculosis\textsuperscript{9}). Intramedullary tuberculoma is a rare finding, occurring in approximately 2\% of cases of neutrotuberculosis, with or without association with pulmonary involvement. The clinical presentation is generally subacute, with a variable picture of muscle weakness, paraparesis or quadriparesis. Constitutional symptoms such as fever and weight loss may not be present\textsuperscript{9,10}). At the disease onset, the inflammatory process with ill defined edema predominates. After formation of the granuloma, the lesions become more defined, generally with
hyposignal on T1-weighted images and iso- to hypersignal on T2-weighted images. There is postcontrast enhancement, being such enhancement nodular and heterogeneous or annular and peripheral depending on the presence or not of caseous necrosis (Figure 3)\(^5,10\). In immunocompromised patients, the finding of nodular lesions with nodular or peripheral enhancement may also be observed in cases of toxoplasmosis or fungal infections, but such conditions are extremely rare\(^{11-13}\).

### ACUTE TRANSVERSE MYELITIS

Idiopathic acute transverse myelitis generally refers to a single phased, acute or subacute inflammatory process, without defined etiology, which develops with bilateral motor and sensitive deficit, sometimes with autonomic dysfunction. Approximately one third of the patients recover completely, one third remain with moderate sequel, and one third maintain severe dysfunction\(^5,14\).

Several conditions may present similar clinical signs, such as infectious, vascular and demyelinating diseases and actinic lesions. Therefore, the final diagnosis of idiopathic acute transverse myelitis depends on the investigation and ruling out of a possible identifiable etiology\(^1,14\). The inclusion criteria for such diagnosis comprise motor/sensitive deficits or autonomic dysfunction attributed to the spinal cord, identifiable level of sensitivity, absence of compressive cause, medullary inflammatory process demonstrated by pleocytosis or increase in IgG in the cephalorachidian fluid, or post-gadolinium injection enhancement at MRI, with worsening peak in 4 hours to 21 days after the symptoms onset. The exclusion criteria are history of local radiation, evidence of collagen disease, syphilis, Lyme disease, HIV, HTLV and other viral etiologies, vascular diseases, lesions suggestive of multiple sclerosis or optic neuritis\(^{15}\).

Findings at MRI are variable, most frequently affecting thoracic segments, and least frequently the conus medullaris. A centrally located hypersignal is observed on T2-weighted images, with or without postcontrast enhancement (Figure 4). Increase in the spinal cord caliber, mimicking neoplastic lesion, may be observed\(^2,5,16\).

In the suspicion of a myelinating etiology, further investigation should be undertaken in search of other lesions in the re-
remaining medullary and intracranial segments, besides investigation for optical neuritis, in suspected cases. Approximately 90% of the patients with medullary lesions associated to multiple sclerosis present intracranial foci of demyelination [16]. Usually, medullary lesions related to multiple sclerosis are peripherally located and with an extent of less than two vertebral bodies, while optic neuritis presents lesions that extend for more than three vertebral bodies, with sharp hypersignal on T1-weighted images and greater association with medullary atrophy [17].

VIRAL MYELITIS

Herpes virus, enterovirus and retrovirus (HIV) are some of the viral agents most frequently causing myelitis. The presentations are very similar to each other and the etiological diagnosis is only confirmed by means of laboratory tests.

The clinical diagnosis of the infection by herpes zoster is many times difficult, as only one third of the patients present the typical skin lesion. Myelitis is a rare manifestation of the infection by herpes zoster and generally occurs after reactivation of a latent infection. In cases where a concomitant skin lesion is present, the medullary level corresponds to the affected dermatome [2,18].

The presentation at MRI varies, ranging from single or multiple lesions, with or without postcontrast enhancement (Figure 5) [18]. Myelitis related to HIV occurs in 5% to 8% of patients with acquired immunodeficiency syndrome, generally in association with a severe encephalic involvement [5].

The HTLV-1 is associated to a progressive presentation of spastic paraparesis. The most affected medullary segment is the thoracic one and there may be association with medullary atrophy, besides lesions in the encephalic white matter [19].

The infection by cytomegalovirus causes polyradiculomyelitis which frequently involves the conus medullaris and cauda equina roots, represented at MRI by contrast-enhanced, clumped and thickened nerve roots, a finding compatible with arachnoiditis [20].

SARCOIDOSIS

Neurosarcoidosis is a rare condition which occurs in approximately 5% of the cases of systemic sarcoidosis [21] and may affect any part of the CNS [22]. Medullary involvement affection is rarely observed, and generally presents a subacute or chronic course. The symptoms are varied and generally are followed by pain, with possible occurrence of motor and sensitive deficits. The diagnosis of neurosarcoidosis represents a challenge in the clinical practice. Correlation with laboratory tests and imaging findings is essential [21,23]. At MRI, frequently multiple and centrally located, heterogeneously contrast-enhanced lesions are observed with hypersignal on T2-weighted sequences. There may be fusiform medullary expansion. For patients with a compatible clinical condition, in the presence of pial enhancement beyond the intramedullary lesions, medullary sarcoidosis must be included among the differential diagnoses (Figure 6). The lesions tend to regress with the utilization of corticoids, generally after clinical improvement [22,24,25].

CONCLUSION

MRI is the method of choice in the approach to myelitis. In the present study, the authors have attempted to describe some relevant imaging findings of inflammatory conus medullaris diseases which can differentiate them from neoplastic or vascular lesions, while suggesting a specific etiologic agent.

The type of contrast-enhancement must be carefully evaluated. The “arborized” pattern is typical of neuroschistosomiasis, while a peripheral annular enhancement may be present in cases of granulomatous lesions such as in tuberculosis and toxoplasmosis. In the presence of cysts, the diagnosis of cysticercosis should be considered, and may be confirmed by the identification of the scolex. The differentiation amongst the several viral etiologies by MRI alone is difficult, and association of imaging findings with clinical and laboratory findings is necessary. Finally, a comprehensive evaluation of the whole neuroaxis may be useful in the definition of a specific eti-
ogy, since in many cases there is concomitant intracranial involvement.

REFERENCES