

1 **Diagnostic approach, therapeutic strategies and surgical indications in**  
2 **Intradural Thoracic Disc Herniation associated with CSF Leak,**  
3 **Intracranial Hypotension and CNS Superficial Siderosis**  
4

5 **ABSTRACT**

6 **Background and purpose:** Intradural disc herniation (IDH) can manifest with radicular or medullary  
7 syndrome. In about 15% of cases, IDH may be responsible, through a dural laceration, for a CSF leak,  
8 determining spontaneous intracranial hypotension (SIH) and CNS superficial siderosis (CNSs). This  
9 paper attempts to present an overview on IDH as the cause for both CSF leak, and subsequent SIH,  
10 and CNSs, and to describe a peculiar clinical and neuroradiological scenario related to this condition.

11 **Methods:** Clinical examination, brain and spinal imaging and lumbar puncture were performed. IDH  
12 was treated through a posterior transdural herniectomy via a monolateral laminectomy and the  
13 placement of muscle and fibrin glue to repair the anterior dural fistula

14 **Results:** A 69-year-old woman was referred to our hospital for slowly progressive gait disturbances  
15 and hearing impairment. Brain imaging revealed diffuse bilateral supratentorial and infratentorial  
16 superficial siderosis, mostly of the cerebellum, the eighth cranial nerves and the brainstem. Spinal  
17 imaging disclosed a posterior disc herniation determining a dural tear at D6-D7. Lumbar puncture  
18 revealed low opening pressure and hemorrhagic CSF with siderophages. Herniectomy and dural  
19 sealing determined a stabilization of hearing and a significant improvement in both gait and balance.

20 **Conclusions:** The diagnostic workup of IDH associated with CNS leak demands whole neuraxis  
21 imaging, especially in cases presenting brain radiological signs of CNSs. This may avoid delays in  
22 detection and treatment of spinal dural leaks. The different forms of treatment available depend on  
23 the type and severity of the clinical picture.

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## 27 **INTRODUCTION**

28 An intradural disc herniation (IDH) consists of a nucleus pulposus fragment of the intervertebral disc  
29 violating the dural sheath and penetrating the thecal space. The first report of an IDH was made by  
30 Dandy in 1942. [1] IDH has an incidence rate of about 0.27% to 0.33% of all cases of disc herniation,  
31 and its incidence peaks in the fifth and sixth decades of life. [2] Approximately 92% of IDHs occur  
32 in the lumbar region, most commonly in the L4-L5 segment (55%), followed by L3-L4 segment  
33 (16%), and L5-S1 segment (10%). [3] By contrast, less than 5% occur in either the cervical or thoracic  
34 segments [4]. The main clinical sign of thoracic IDH is represented by radicular pain. [4] Thoracic  
35 IDH may also manifest with walking ataxia or pyramidal tract syndrome, raising the suspect of  
36 medullary compression. Other less common signs include axial pain (cervical above T5 level and  
37 lumbar below T10 level), scapulargia for T1–T2 locations, radiating pain in the T1 territory and  
38 Bernard Horner’s syndrome due to T1 root’s sympathetic component compression. IDH may also  
39 result in a dural laceration, which, in turn, can promote a CSF leak, whose incidence has been reported  
40 to be up to 15%. [5] CSF leak may be easily responsible for spontaneous intracranial hypotension  
41 (SIH) and CNS superficial siderosis (CNSss). While SIH mainly manifests primarily with headache,  
42 CNSss becomes explicit with hearing and gait impairments. Both SIH and CNSss are also  
43 characterized by typical radiological findings.

44 In this paper, we review the literature concerning thoracic IDH as the cause for both CSF leak, and  
45 subsequent SIH, and CNSss. We also present a case exposing the peculiar clinical picture, discussing  
46 the possible underlying pathophysiological mechanisms and examining the therapeutic strategies that  
47 were implemented.

48

## 49 **CASE PRESENTATION**

50 We present the case of a 69-year-old, right-handed, woman who suffered from a 1-year history of  
51 slowly progressive gait disturbances and hearing impairment. Neurological examination revealed an  
52 ataxic gait and bilateral sensorineural auditory alteration. No previous history of surgery or trauma of

53 the CNS were present, and no major comorbidities were reported. Therefore, the patient underwent  
54 brain magnetic resonance imaging (MRI) (**Fig.1**) which disclosed diffuse bilateral supratentorial and  
55 infratentorial superficial siderosis, mostly of the cerebellum, the eighth cranial nerves and the  
56 brainstem. Hemosiderin coating was specifically accentuated at the level of the cerebellar folia. This  
57 appearance was combined with a prominent and symmetrical secondary degenerative atrophy of the  
58 vermis and superior cerebellar peduncles. Spinal MRI and computed tomography (CT) (**Fig.1**)  
59 showed an anterior dural detachment with a ventral epidural fluid collection extending from D6-D7  
60 up to D1. There was also an osteoporotic fracture with anterior wedge deformation of the D6 and D7  
61 somae with superimposed posterior marginal-somatic osteophytosis and herniation of disc material  
62 with osteocalcific signal that determined a dural tear at D6-D7. Moreover, fibrotic reactive tissue was  
63 recognizable at the level of dural defect. Lumbar puncture showed a low opening pressure (3 cm  
64 H<sub>2</sub>O) and hemorrhagic CSF with 40/mm<sup>3</sup> red blood cells and siderophages. In October 2021, the  
65 case was discussed by a multidisciplinary committee which set the indication for surgical repair of  
66 the dural fistula. The patient gave written informed consent for the procedure. The day of surgery,  
67 the patient was positioned prone and, under microscopic guidance, a D6-D7 herniectomy through a  
68 right hemilaminectomy combined with an intradural approach was performed (**Fig.2**). A muscle patch  
69 and fibrin glue were used to seal the ventral dural defect. Histology confirmed degenerative disc  
70 material. Postoperative course was uneventful and characterized by early autonomous mobilization.  
71 The patient was discharged from our Institute two days after the surgical procedure. She exhibited no  
72 signs or symptoms of intracranial hypotension at the time of the discharge and at two months follow-  
73 up. In addition, she reported a stabilization of the hearing impairment but also a significant  
74 improvement in both gait and balance in the following two months after surgery.

75

## 76 **LITERATURE REVIEW**

77 In 2014, Toro et al. described the case of a patient with a four-year history of leg weakness and  
78 progressive gait impairment, besides other symptoms such as bilateral hearing loss and urinary

79 incontinence. T2-weighted brain MRI showed extensive hemosiderin deposits around the brainstem  
80 and along the cerebellar folia, while spinal MRI demonstrated a disc herniation at the T8-T9 level,  
81 located where a previous dynamic CT myelogram highlighted a ventral dural defect. A surgical dural  
82 repair was offered but declined by the patient. [6] Reviewing this case, we believe that in cases of  
83 thoracic IDH with progressive myelopathy and/or CNSs, a more invasive treatment is needed  
84 through a total microdiscectomy.

85 In 2018, Wipplinger et al. described a case of CNSs secondary to a thoracic IDH causing CSF leak.  
86 Their patient was successfully treated with lateral T6-T8 transpedicular partial corpectomy, as well  
87 as discectomy with decompression and fusion, followed by watertight closure of the CSF leak. No  
88 postoperative complications occurred, and, at three months' follow-up, the patient displayed stability  
89 of preoperative symptoms, such as mild bilateral hypoacusis and mild difficulty in tandem gait [7].

90 In 2020, Cornips et al. presented two patients with a thoracic IDH, secondary SIH and, in one of them,  
91 CNSs [8]. The first patient, without CNSs, presented with frontal headache, mild cognitive  
92 dysfunction and gait impairment. Cranial MRI showed bilateral subdural effusions and sagging of the  
93 midbrain. On the suspicion of a spinal CSF leak, MRI of the entire neuraxis was also performed,  
94 demonstrating a left paramedian thoracic IDH at the T9-T10 level. After refractoriness to both  
95 thoracic and lumbar EBP, a left-sided thoracoscopic microdiscectomy was performed, resulting in  
96 clinical improvement [8]. The second patient, with CNSs, presented with a two-year history of pain  
97 in the occipital region. Cranial MRI indicated subarachnoid susceptibility artifacts, particularly in the  
98 posterior fossa. As both history and MR angiography were not indicative for subarachnoid bleeding,  
99 a diagnosis of CNSs was formulated. Spinal MRI revealed a large central thoracic IDH at the T7-T8  
100 level. Hence, a left-sided tubular microscopic discectomy was performed. A few days after surgery,  
101 the patient became dyspneic and a CT-thorax demonstrated a large fluid collection in the left  
102 hemithorax, thus posing an indication for an external lumbar drainage, which allowed significant  
103 improvement of the clinical picture [8]. In our opinion, in the presence of a thoracic IDH with  
104 symptomatic SIH and CNSs, the conservative EBP does not represent the most effective therapeutic

105 strategy. At the same time, in the presence of mild SIH and/or CNSss symptoms, a thoracoscopic  
106 approach for microdiscectomy represents a far too invasive procedure.

107

## 108 **DISCUSSION**

109 The migration of a disc into the intradural region requires perforation of the annulus fibrosus, and  
110 then the laceration of both the posterior longitudinal ligament and the dura mater. Various  
111 predisposing anatomical factors have been proposed, of which the most relevant one is the presence  
112 of adhesions between the annulus fibrosus, the posterior longitudinal ligament and the dura mater.  
113 These adhesions are likely to be congenitally present or occur secondary to chronic disc protrusion  
114 [9]. In this regard, over the long term, a herniated disc does not represent an inert tissue, but rather an  
115 active area of proliferation, neovascularisation, and inflammation. The inflammatory response  
116 (expressed by the production of proinflammatory cytokines) leads to calcification of the disc, which,  
117 in turn, causes intradural erosion, and subsequently, tears [10]. Lesions of the dura mater, in turn,  
118 may lead to SIH [11]. CSF leak is also associated with CNSss [12, 13], a rare disorder characterized  
119 by the deposition of hemosiderin in the leptomeninges and the subpial layer of the cerebellum, VIII  
120 cranial nerve, brainstem and spinal cord, resulting in severe neurological deficits, such as ataxia and  
121 hearing impairment [14].

122 MRI yields pathognomonic findings, which may be revealed in the pre-symptomatic phase of the  
123 disease. They result from the deposits of hemosiderin, ferritin and ionic iron. In gradient-echo T2-  
124 weighted images, the hallmark is a dark band surrounding the intracranial structures, such as the  
125 cerebellar hemispheres and the VIII cranial nerve, while other cranial nerves are less frequently  
126 affected. Following gadolinium administration, the leptomeninges are enhanced. On the other hand,  
127 CT imaging is not sufficient for the diagnosis of CNSss [15].

128 The main differential diagnosis of CNSss are neoplasms (21%), cranial or spine trauma (13%), and  
129 vascular malformations (9%), neurosurgical procedures, (7%), brachial plexus injury (6%), and  
130 cerebral amyloid angiopathy (3%). Nevertheless, most cases remain idiopathic (35%) [16].

131 The association between CSF leak and CNSss has been explained by two theories. The first theory  
132 states that brain sagging due to intracranial hypotension may lead to bleeding from bridging veins on  
133 the surface of the cerebellum [17]. While the second theory affirms that the bleeding source may be  
134 localised around the dural defect [13, 18, 19]. In fact, regarding the latter theory, when CSF leaks  
135 through a dural defect, the epidural space enlarges, stretching the epidural venous plexus and making  
136 it more prone to laceration. Therefore, when bleeding from this plexus occurs, it may not stop  
137 spontaneously because of CSF's continuous flow. This, in turn, can lead to subarachnoid bleeding.

138 Preoperative imaging-based diagnosis of thoracic IDH is challenging. Considering the literature [20-  
139 30], in only a few cases the presence of thoracic IDH was suspected following preoperative  
140 investigation with CT myelography. In our case, preoperative diagnosis through spine MRI was  
141 possible by understanding the underlying pathophysiological mechanism that binds CNSss, SIH and  
142 IDH. However, when preoperative imaging-based diagnosis of IDH is non-discerning, an alternative  
143 route must be undertaken and therefore diagnostic workup for SIH should be implemented. SIH can  
144 be identified through neuroradiological exams such as brain MRI. Through brain MRI, five typical  
145 features can be identified: subdural fluid collections (50%), diffuse non-nodular enhancement of the  
146 pachymeninges (due to dilated blood vessels in the subdural area), engorgement of venous structures,  
147 pituitary hyperemia, to the point of mimicking a pituitary tumor [31] and sagging of the brain [32].

148 Both the dural enhancement and pituitary enlargement are consistent with the Monro–Kellie doctrine:  
149 in the presence of a significant decline in CSF flow, intracranial blood volume must increase to keep  
150 the total intracranial volume constant. In line with this theory, subdural hematomas, could be the  
151 expression of an underlying CSF leakage and they usually can be managed by treating the spinal CSF  
152 leak. [33] In addition to MRI imaging, brain CT scan can suggest the diagnosis by highlighting  
153 subdural fluid collections or obliteration of subarachnoid cisterns. [34] Even myelography with  
154 iodinated contrast followed by spine CT can accurately define the location of a CSF leak. [35]

155 Surgical treatment of thoracic IDH is needed in the presence of radicular symptoms, myelopathy  
156 clinical and radiological signs (highlighted by a medullary T2 hyperintensity area on spine MRI) and

157 symptomatic SIH. [36] In our case, symptoms were ascribed to CNSs, thus posing a new and rare  
158 surgical indication in the case of an IDH. Three types of surgical approaches are now used in the  
159 treatment of thoracic IDH: 1) posterolateral with pedicular-transfacet and transfacet variations that  
160 spare the pedicle; 2) lateral such as costotransversectomy; 3) anterior such as transpleural  
161 thoracotomy, thoracoscopy and mini-thoracotomy. The choice of approach depends on patient's  
162 characteristics (weight) and on the location (central or lateral), size and type (soft or calcified) of  
163 herniation. On the other hand, treatment of SIH begins with conservative management, including bed  
164 rest, intravenous administration of fluids and steroids. If the patient fails to respond to medical  
165 therapy, epidural blood patch (EBP) is adopted [37], either by percutaneous or open surgical  
166 approaches. The mechanism of action of EBP is based on its initial tamponade effect over the dural  
167 tear and subsequent scar formation. [38]

168 In our case, the clinical picture was mainly ascribed to CNSs, which, undoubtedly, set the indication  
169 to invasive treatment as to resolve both the CSF fistula and the SIH. Given the absence of myelopathy  
170 and significant SIH symptoms, our surgical strategy was primarily based solely on repairing the  
171 ventral dural defect to handle the progression of frank CNSs. Therefore, we proceeded with a  
172 posterior transdural herniectomy through a monolateral laminectomy and the placement of muscle  
173 and fibrin glue to repair the anterior dural fistula, rather than performing a more invasive total  
174 microdiscectomy.

175

## 176 **CONCLUSIONS**

177 In the case of thoracic IDH associated with suspected CSF leak, diagnostic workup should include  
178 MRI of the whole spine, particularly in patients having radiological signs of CNSs. This may avoid  
179 delays in detection and treatment of spinal dural CSF leaks. With no apparent myelopathy, a less  
180 demolitive surgery is recommended, proceeding with the repair of the fistula with sealants and the  
181 excision of the intradural herniated disc material.

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297

## 298 **FIGURE LEGEND**

299 **Fig. 1 (a)** Axial T2-weighted Brain Magnetic Resonance imaging (MRI), **(b)** Axial Constructive  
300 Interference in Steady State (CISS) sequences of Brain Magnetic Resonance imaging (MRI) and **(c)**  
301 Axial Gradient-echo T2-weighted Brain Magnetic Resonance imaging (MRI) showing infratentorial  
302 superficial siderosis mostly of the cerebellum (specifically at the level of the folia), the eighth cranial  
303 nerves and the brainstem; **(d)** Axial T2-weighted Spinal Magnetic Resonance imaging (MRI), **(e)**  
304 Sagittal T2-weighted Spinal Magnetic Resonance imaging (MRI) and **(f)** Non-contrast sagittal Spinal  
305 Computed Tomography (CT) images demonstrating an anterior wedge deformation of the D6 and D7  
306 somae with superimposed posterior herniation of disc material (white arrow) with osteocalcific signal  
307 that determined a dural tear at D6-D7

308 **Fig. 2** Intraoperative picture illustrating the ventral intradural disc herniation after posterior durotomy