

1 Pseudo-meningocele secondary to obstetrical brachial paralysis

2 **Key words** : Pseudo-meningocele- obstetrical paralysis – brachial plexus .

3 **Introduction** :

4 Obstetrical Brachial Plexus Palsy (OBPP) is a severe paralysis of an upper limb present at  
5 birth. The therapeutic measures for brachial plexus palsy depend on the pathologic condition  
6 and the location of the injury. Imaging studies mainly MRI are used for evaluation of the  
7 brachial plexus. We report a case of a 2-year-old female patient who presents to pediatric  
8 department for weakness of the upper left limb since birth

9 **Case report**

10 We report the case of a 2-year-old female patient who presents to paediatric constltation for  
11 weakness of the upper left limb since birth. The interrogation found a history of vaginal  
12 delivery with macrosomia in the newborn. Traduction en Anglais.

13 As an exploration of the brachial plexus, a cervical MRI was performed. On the T2 STIR axial  
14 sequence with maximal intensity projection ( MIP), we find a foraminal and extraforminal  
15 cystic formation spoiled in T2 hypersignal without clear individualization of the nerve roots  
16 (Figure 1) revealing a pseudo-meningocele. Coronal sections of the same sequence with  
17 MIP confirm the diagnosis (Figure 2).

18 **Discussion**

19 Obstetrical Brachial Plexus Palsy (OBPP) is a paralysis of an upper limb present at birth. Its  
20 frequency is 0.04 to 0.4% of births , affecting up to 3 per 1000 live births. (1)

21 The brachial plexus is located in the lateral neck and extending posterior to the clavicle  
22 into the axilla . It is a complex sensory and motor neural network formed by the 5 nerve  
23 roots destined for the upper limb and pectoral girdle : C5, C6, C7, C8, T1 .

24 The origin nerves are the anterior primary roots of C5 to T1, located at the level of the lateral  
25 cervical foramina. The trunks derived from these roots are situated in the antero-inferior  
26 portion of the interscalene triangle of the neck, comprising the upper trunk (C5-C6), middle  
27 trunk (C7), and lower trunk (C8-T1). Each trunk splits into six divisions at the costoclavicular  
28 triangle, joining at the lateral margin of the first rib to form three cords based on their  
29 relationship with the subclavian-axillary artery: the medial cord (continuation of the anterior  
30 division of the lower trunk), the lateral cord (anterior divisions from the upper and middle  
31 trunks), and the posterior cord (formed by the three posterior divisions of the trunks). The  
32 branches derived from these cords give rise to the musculocutaneous, axillary, radial,  
33 median, and ulnar nerves, which originate lateral to the pectoralis minor muscle.

34 The risk factors for the occurrence of POPB are mainly macrosomia and shoulder dystocia  
35 in the newborn. Gestational diabetes , multiparity and excessive weight gain during  
36 pregnancy, were also identified in literature. (2, 3).

37 Clinical manifestations of permanent nerve dysfunction include muscular weakness,  
38 paralysis, and secondary skeletal changes, highlight the importance of early and effective  
39 diagnosis and treatment.

40 At birth, it is impossible to determine, through examining the child, the type of injury involved.  
41 Only the progression of paralysis over the first few months of life can guide towards a  
42 favorable or unfavorable outcome.

43 The severity of paralysis varies with the type of injury and the number of affected roots,  
44 ranging from a stretch injury to a complete avulsion of the nerves from the spinal cord (4) .  
45 The most common involvement affects the upper plexus (C5-C6), where the hand is spared,  
46 but there is a deficiency in shoulder active abduction, external rotation, and elbow flexion.  
47 This is the mildest form, with complete recovery occurring in only one-third of cases. (3)  
48 Involvement of C5-C6-C7 causes additional deficits in elbow and finger extension, adding to  
49 the previous deficits, and is known as Erb's palsy. Finally, involvement of all roots results in a  
50 "complete plexus" condition, where the limb is completely flaccid with no mobility, particularly  
51 at the hand level. This is the most severe form and inevitably leads to sequelae. Nearby  
52 roots to the brachial plexus may also be affected. Involvement of C4 (phrenic nerve) leads to  
53 paralysis of a diaphragmatic dome, potentially causing respiratory distress. Involvement of  
54 C8 and T1 can result in Claude Bernard-Horner syndrome, characterized by miosis, ptosis,  
55 and enophthalmos.

56 In order to achieve a correct diagnosis, it is necessary to perform adequate neurological and  
57 electrophysiological exploration plus radiological imaging. The MR is the gold standard  
58 imaging technique used to evaluate and differentiate between preganglionic and  
59 postganglionic traumatic injury. (5)

60 MR protocol includes axial , coronal and sagittal images including the entire brachial plexus in  
61 the same plane. Sequences include T1-weighted images to have a fine anatomical  
62 resolution; STIR or T2-weighted images with fat suppression help to identify brachial plexus  
63 edema. T1 with injection of GADOLINIUM is not necessary in traumatic palsy . (5)

64 Traumatic plexopathies are generally classified according to the degree, location, and  
65 mechanism of injury. The goal is to estimate the likelihood of spontaneous recovery and help  
66 identify cases that would benefit from surgical management.

67 The location of an injury also is important for injury classification. Pre- versus postganglionic  
68 injury is a major distinction .

69 Preganglionic injuries refer to an avulsion of the nerve rootlets from the spinal cord. In a  
70 recent systematic review (6) of MRI for presurgical assessment of preganglionic injury, it is  
71 estimated that mean sensitivity and specificity are around 93% and 72%. Discontinuity of the  
72 ventral or dorsal nerve roots from the spinal cord MRI is the main direct sign of root avulsion  
73 Pseudomeningocele, is one of the most frequent indirect sign of preganglionic injury. It refers  
74 to meningeal tear and consequently leakage of cerebrospinal fluid through it . On MRI , it  
75 appears as a cystic collection that can track through the neural foramen and communicate  
76 with the subarachnoid space .Also up to 23% of root avulsions do not have an associated  
77 pseudomeningocele, and pseudomeningocele-like lesions are identified in the absence of  
78 root avulsion in up to 24% of cases . Non traumatic extradural meningeal cyst the most  
79 frequent differential diagnosis , which is uncommon at the cervicothoracic junction. ( 8).  
80 Spinal cord edema near the level of a root avulsion associated to denervation change of the  
81 ipsilateral paraspinal muscles, supplied by the dorsal branch of the spinal nerve, are also  
82 other indirect sign of preganglionic injury. The lower C8–T1 roots more commonly manifest  
83 with preganglionic injury (7). Although most cases resolve spontaneously, permanent injury

84 is not uncommon. Injuries involving the lower trunk are more likely to result in permanent  
85 deficits (9).

86 In a study on fifteen patients (mean age: 14.5 months) where radiologists reviewed MR  
87 imaging in infants with Erb's palsy, before surgical exploration of the brachial plexus . They  
88 found at least one pseudomeningocele in 8 of the 15 patients (53.3%) while 3 of the 15  
89 patients (20%) had multiple pseudomeningoceles. Posterior shoulder subluxation was seen  
90 in 11 patients (73.3%). Fourteen children (93.3%) had imaging abnormalities consistent with  
91 either a reparative neuroma or scar tissue investing plexus elements. (12)

92 Postganglionic injuries have better prognosis, the more distal location is better . On MRI it  
93 manifests as stretching, rupture, oedema, hematoma, and neuronam as nodular thickening .  
94 The upper C5–C7 roots are more susceptible to postganglionic injury (rupture at an  
95 extraforaminal location). (7)

96 Lesion severity is also graded on the basis of the estimated damage to the various layers of  
97 connective tissue surrounding the nerve (10). Seddon initially described three types of nerve  
98 injuries: neuropraxia, axonotmesis, and neurotmesis (in order of severity). Neuropraxia  
99 occurs after disruption of the myelin sheath, without distal Wallerian degeneration, and  
100 typically results in transient conduction abnormality and sensory dysfunction. Axontmesis  
101 refers to axonal injury resulting in distal Wallerian degeneration with intact endoneurium or  
102 perineurium. This may result in motor and/or sensory nerve dysfunction. Neurotmesis, the  
103 most severe injury, refers to disruption of multiple layers of the nerve with or without  
104 complete transection.(11)

#### 105 **Conclusion :**

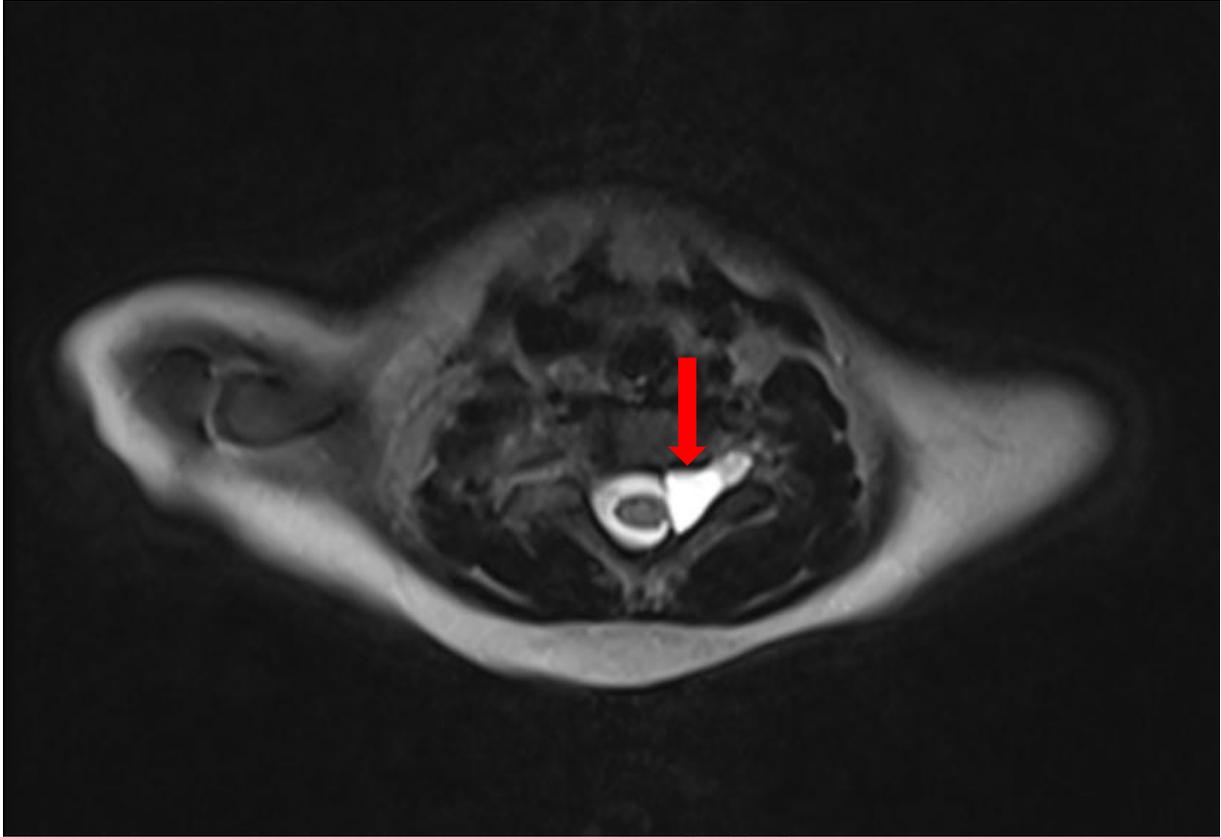
106 To conclude, paralysis of brachial plexus in newborn is a rare and severe complication . MRI  
107 is the gold standard to explore this affection. The goal is to estimate the likelihood of  
108 spontaneous recovery and help identify cases that would benefit from surgical management

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157 Figure 1: MRI image in axial section of T2 SPACE showing foraminal and  
158 extraforaminal cystic formation spoiled in hypersignal T2 without  
159 individualization of the nerve roots ( red arrow)



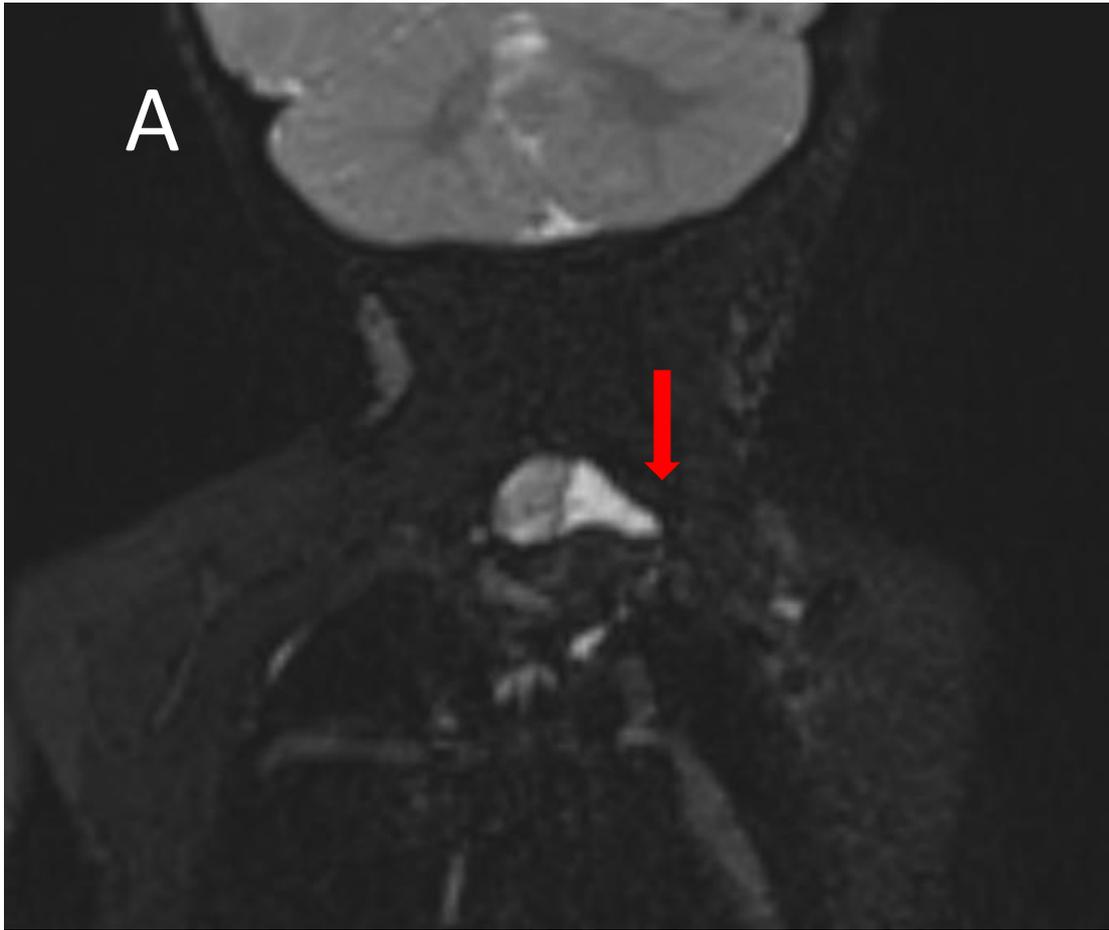
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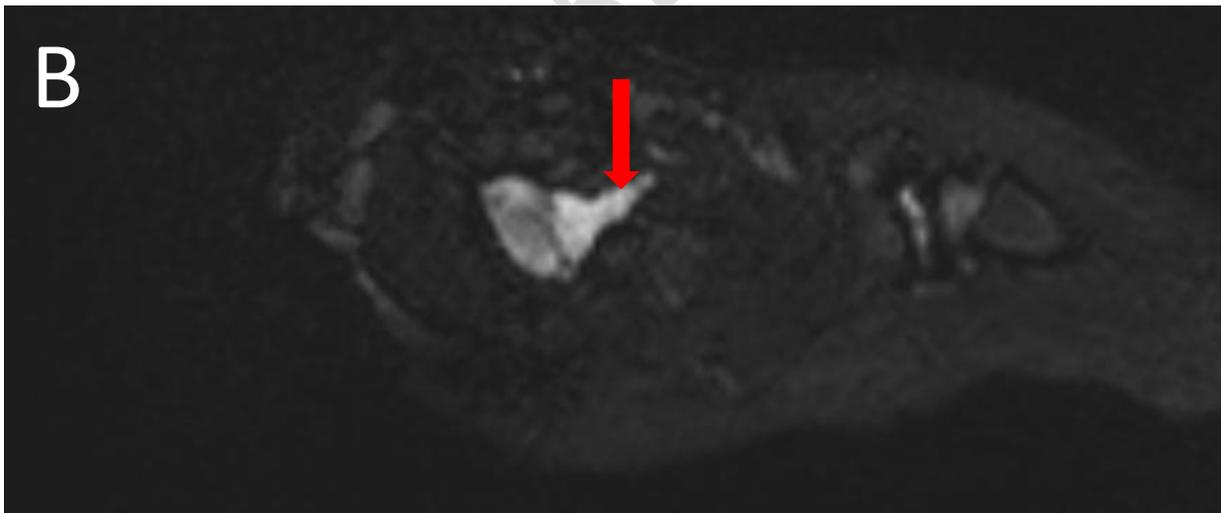
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163 Figure 2: MRI images in coronal (A) and axial ( B) sections of T2 3D SPACE  
164 showing foraminal and extraforminal cystic formation spoiled in hypersignal  
165 without individualization of the nerve roots ( red arrow)

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