HORNER SYNDROME IN CHILDREN: A CLINICAL CONDITION WITH SERIOUS UNDERLYING DISEASE

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Horner syndrome corresponds to the clinical triad of miosis, ptosis and facial anhidrosis. These symptoms are related to injury of the oculosympathetic chain. In children, Horner syndrome is classified as congenital or acquired. While the diagnosis is made through clinical examination, there is some debate regarding the use of imaging modalities and the extent of anatomical coverage required. Here, we describe two cases of children with acute Horner syndrome. We then review the literature about the different aetiology and discuss the interest of some investigations.

Case 1:

An 8 months girl without personal or familial history, has presented a right acquired Horner syndrome without additional signs. Frontal chest radiography with cervical and abdominal ultrasonography was first achieved and returned normal. The cerebral and cervical MRI with angiographic sequences performed in a second time was also normal. Finally, the thoracic CT-scan demonstrated a mass at the pulmonary apex. Urinary concentrations of homovanillic acid and vanillylmandelic were normal. No ocular pharmacological testing were performed. Thoracic MRI was finally realized to clarify tumour extension before surgery. Analysis of the tumour confirmed the suggested diagnosis of neuroblastoma.



Thoracic CT-scan. with contrast demonstrate a mass of 1.67 cm of diameter at the apex of the right lung (arrow). Calcifications are presents on the periphery of this lesion

Case 2:

A 9 year boy without personal or familial history, has presented an acute headache with loss of consciousness during a basketball competition. Upon waking up, the child has right hemiplegia, aphasia and left Horner syndrome. The cerebral CT-scan realised in first line was normal. The MRI with angiographic sequences demonstrated M1 left carotid dissection with homolateral white matter infarction.



Cerebral MRI demonstrate an acute ischemic stroke in the left lenticulo-striatal region on FLAIR sequences (circle) and a decreased vascular flow in the left internal carotid artery secondary to dissection on angiographic sequences (arrow)

Conclusion

Imaging studies seem critical in delineating the nature and extent of any underlying pathology along the oculosympathetic pathway in children presenting a Horner syndrome. In these patients, a history of trauma or surgery may reduce the need for extensive systemic evaluation. Without such anamnesis, a decision to proceed with further evaluation is made with consideration of the relative incidence of tumour, especially neuroblastoma, or other treatable lesions. In this condition, the use of radiography, ultrasonography, biology and CT is limited. MIR-MRA of the head, neck and upper chest, covering at least the regions extending from the orbital apices to the T3 vertebrae, seems to be the more sensitive, simple, cost-effective and so recommended investigation.





