Pediatric Oncall Journal
Volume : 21, Issue 4:176-177
DOI: https://doi.org/10.7199/ped.oncall.2024.54

LETTER TO EDITOR (VIEWERS CHOICE)

WHEN A CHILD REFUSES TO WALK

Luzia Condessa[1], Maria Francisca Amaral[1], Carolina Guimarães[1], Dalila Forte[2], Amets Sagarribay[2], Sofia Nunes[3].

[1]Pediatric Funcional Unit, Hospital de Cascais, Cascais, Portugal,

[2]Pediatric Neurosurgery, Hospital Dona Estefânia, ULS São José, Lisbon, Portugal,

[3]Pediatric Oncology Department, Portuguese Institute of Oncology of Lisbon, Lisbon, Portugal.

KEYWORDS

Medulloblastoma, ataxia, hydrocephalus.

ARTICLE HISTORY

Received 02 July 2024

Accepted 11 September 2024

Medulloblastoma (MB), the most prevalent malignant pediatric brain tumor, is a central nervous system (CNS) that typically arises from posterior fossa.[1,2] Clinical features, which usually develop over weeks to months, result from intracranial hypertension and cerebellar disfunction.[2,3] One-third of pediatric patients present symptoms before 3-years-old.[4]

We report the challenging case of a 2-year-old female with a fulminant clinical presentation initially misinterpreted as Urinary Tract Infection (UTI). The patient, with inconspicuous family or personal health history, presented a 3-day clinical picture of persistent irritability, vomiting, anorexia and intermittent refusal to walk, without fever or other manifestations. The physical examination was unremarkable. Due to leukocyturia, urine culture was collected. Symptoms' persistence 2 days later determined her readmission. She was prostrated on her mother's lap, with no other significant findings. Gait was not evaluated. Analytically, complete blood count, inflammatory and renal function parameters were intact. Given the isolation of Escherichia coli colonies in urine and maintained oral intolerance, the toddler was hospitalized assuming a UTI and started IV antibiotic. On the second day of hospitalization, the symptoms deteriorated, with onset of ataxia, axial tremor, nystagmus and complete refusal to walk. A head-CT scan exposed an expansive lesion coincident with an embryonal tumor with IV ventricular obliteration and hydrocephalus. These findings were later confirmed by an MRI scan, which revealed intracranial and medullar leptomeningeal dissemination. The child underwent a complete lesion excision, with histological confirmation of an anaplastic medulloblastoma. On the 20th day of hospitalization, the child was transferred to Oncology Department for ongoing care and investigation, under acetazolamide and dexamethasone, with chemotherapy initiation (etoposide and carboplatin). Significant neurological improvement was observed and the child was discharged six days later. MYC gene amplification was detected and the tumor was classified as probable molecular group 3 (methylation pattern was not performed). Clinical deterioration was perceived and the girl was readmitted following a 24-day hospitalization. Triventricular hydrocephaly, seizures onset and behavior changes (bad social contact, irritability and mutism) determined respectively insertion of a ventricular shunt-peritoneal and anticonvulsants (levetiracetam and phenytoin) and fluoxetine start. Despite chemotherapy, a leptomeningeal tumoral dissemination's worsening and a clinical course's fastidious aggravation occurred. The patient died six months after MB's diagnosis.

This case illustrates the importance of considering this severe diagnosis, even when laboratory findings are coincident with another diagnosis. The acute nonspecific clinical presentation, along with leukocyturia end E.coli isolation consistent with UTI made this diagnosis challenging. The rapid progression and severity of MB are noteworthy, emphasizing the importance of maintaining a high index of suspicion. Serial neurological examinations are essential when evaluating a child with vomiting and irritability. In this case, the refusal to walk and altered neurological examination were crucial for prompt diagnosis and treatment. However, despite chemotherapy, the patient's clinical course was unfavorable. Besides anaplastic group being the most aggressive MB's histological group, the MYC gene amplification, patient's age and the presence of metastasis contributed to its categorization in a group 3 molecular group, predicting a poor prognosis (estimated 5-year survival rate of 40%).[1,5,6]

Compliance with Ethical Standards

Funding : None

Conflict of Interest : None

Address for Correspondance: Luzia Condessa, Avenida Brigadeiro Victor Novais Gonçalves, Hospital de Cascais, 2755-009, Cascais, Portugal.

Email: luziacondessa@gmail.com.

References:

1. Menyhárt O, Győrffy B. Molecular stratifications, biomarker candidates and new therapeutic options in current medulloblastoma treatment approaches. Cancer Metastasis Rev. 2020; 39(1):211-233. doi: 10.1007/s10555-020-09854-1.
2. Jackson K, Packer RJ. Recent Advances in Pediatric Medulloblastoma. Curr Neurol Neurosci Rep. 2023 ; 23(12):841-848. doi: 10.1007/s11910-023-01316-9.
3. Millard NE, De Braganca KC. Medulloblastoma. J Child Neurol. 2016 ; 31(12):1341-53. doi: 10.1177/0883073815600866.
4. Fang FY, Rosenblum JS, Ho WS, et al. New Developments in the Pathogenesis, Therapeutic Targeting and Treatment of Pediatric Medulloblastoma. Cancers (Basel). 2022;14(9):2285. doi:10.3390/cancers14092285
5. Sharma T, Schwalbe EC, Williamson D, et al. Second-generation molecular subgrouping of medulloblastoma: an international meta-analysis of Group 3 and Group 4 subtypes. Acta Neuropathol. 2019 ;138(2):309-326. doi: 10.1007/s00401-019-02020-0.
6. Cavalli FMG, Remke M, Rampasek L, et al. Intertumoral Heterogeneity within Medulloblastoma Subgroups. Cancer Cell. 2017; 31(6):737-754.e6. doi: 10.1016/j.ccell.2017.05.005.