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Original Research Paper

Radiology

"DANDY–WALKER MALFORMATION IN A SCHOOL-AGED CHILD: DIAGNOSTIC INSIGHTS FROM COMPUTED TOMOGRAPHY"

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ABSTRACT Dandy–Walker malformation (DWM) is a congenital anomaly of the posterior fossa characterized by cystic dilatation of the fourth ventricle, hypoplasia of the cerebellar vermis, and enlargement of the posterior fossa. It may coexist with supratentorial malformations, including agenesis of the corpus callosum. We report the case of a seven-year-old girl who presented with recurrent headache and fever. Non-contrast computed tomography (NCCT) of the brain demonstrated a large posterior fossa cyst communicating with a dilated fourth ventricle, bilateral lateral ventriculomegaly, hypoplastic vermian remnant, and partial agenesis of the corpus callosum with superior displacement of the torcula, consistent with Dandy–Walker malformation. This case emphasizes the role of neuroimaging in diagnosing congenital midline malformations and guiding multidisciplinary management to improve neurodevelopmental outcomes.

KEYWORDS : Dandy–Walker, Malformation

INTRODUCTION

Dandy–Walker malformation represents a group of posterior fossa developmental anomalies resulting from defective embryogenesis of the rhombencephalon between the 7th and 10th weeks of gestation[1]. The hallmark triad includes cystic dilatation of the fourth ventricle, hypoplasia or agenesis of the cerebellar vermis, and enlargement of the posterior fossa[2]. It accounts for nearly 4–12% of all cases of congenital hydrocephalus[3]. The condition is frequently associated with other intracranial malformations such as agenesis of the corpus callosum, polymicrogyria, and heterotopia[4].

The clinical spectrum is highly variable, ranging from incidental findings in older children to severe developmental delay and hydrocephalus in infancy[5]. Here, we present a detailed imaging-based case description of Dandy–Walker malformation with partial corpus callosal agenesis in a seven-year-old female, correlating radiological findings with clinical presentation.

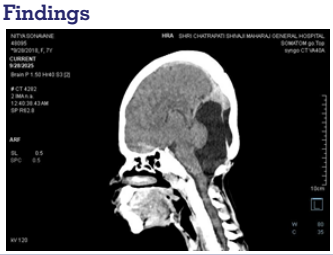
Case Presentation

A 7-year-old female, presented to the outpatient department of government medical college, with complaints of intermittent headache and fever for two weeks. There was no history of seizures, head trauma, visual blurring, vomiting, or developmental regression. Her antenatal and perinatal history was uneventful, and developmental milestones had been achieved within normal age limits. There was no significant family history of neurological disorders.

Clinical Examination

The child was alert, afebrile, and hemodynamically stable at presentation. Neurological examination revealed no cranial nerve deficits, normal tone and power, and no cerebellar signs such as ataxia or dysmetria. Fundoscopy showed no papilledema, ruling out significant raised intracranial pressure.

Radiological Findings



Non-contrast CT Scan Of The Brain Revealed The Following:

A large posterior fossa cystic lesion measuring approximately 2.5 × 6.2 × 7.2 cm (AP × TR × CC) occupying the posterior fossa and communicating with a mildly dilated fourth ventricle, extending cranially to the bilateral lateral ventricles and the third ventricle, suggesting communication through the aqueduct.Vermian hypoplasia was clearly seen, with superior displacement of the residual vermian tissue (vermis remnant visible as a thin superior band on the sagittal CT image).Superior displacement of the torcula (torcular-lambdoid inversion) was noted, consistent with posterior fossa expansion typical of Dandy–Walker malformation. Partial agenesis of the corpus callosum was identified as absence of the posterior body and splenium, with preserved anterior genu and rostrum. The lateral ventricles appeared mildly dilated and parallel in orientation, supporting the diagnosis.

Mild mucosal thickening of the maxillary and sphenoidal sinuses was observed, likely secondary to upper-airway inflammation. Soft tissue density was seen in bilateral mastoid air cells consistent with otomastoiditis. The cerebral hemispheres and brain parenchyma appeared normal in attenuation pattern, with no midline shift, intracranial haemorrhage, or fractures.

Diagnosis- Based on imaging and clinical findings, the features were consistent with Dandy–Walker malformation associated with partial corpus callosal agenesis.

DISCUSSION

The presented case demonstrates the classic neuroimaging triad of Dandy–Walker malformation: vermian hypoplasia, cystic dilatation of the fourth ventricle, and torcular elevation[6]. The sagittal CT image provided clearly shows a large posterior fossa cyst communicating with the fourth ventricle, displacing the tentorium and occipital lobes superiorly — a distinguishing feature from posterior fossa arachnoid cysts[7]. The partial agenesis of the corpus callosum, identified as absence of posterior callosal components, is a well-recognized association, occurring in up to one-third of DWM cases[8]. These midline anomalies result from abnormal mesenchymal differentiation and failure of normal CSF pathway formation during early embryogenesis[9].

Although this child was neurologically intact, long-term monitoring is advised, as subtle cognitive and coordination deficits may emerge later. Management is primarily symptomatic and multidisciplinary, involving pediatric

neurologists, neurosurgeons, and developmental specialists[10]. Hydrocephalus, if present, may require ventriculoperitoneal shunting. The absence of significant hydrocephalus in this case suggests a relatively milder variant of the Dandy–Walker spectrum, likely the Dandy–Walker variant rather than the classic form.

CONCLUSION

This case highlights the importance of neuroimaging in children presenting with non-specific neurological symptoms, as congenital anomalies such as Dandy–Walker malformation can remain clinically silent until later childhood. Identification of associated anomalies, such as corpus callosal agenesis, aids in prognostication and management planning.Early recognition and periodic neuro-developmental assessment are crucial to ensure timely intervention and to improve long-term cognitive and motor outcomes.

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