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Congenital Hydrocephalus with Occipital Meningoencephalocele: A Case Emphasizing the Importance of Early Diagnosis and Surgical Management

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ABSTRACT

Title: Congenital Hydrocephalus with Occipital Meningoencephalocele: A Case Emphasizing the Importance of Early Diagnosis and Surgical Management.

Background: Occipital meningoencephalocele is a significant neural tube defect, commonly seen with hydrocephalus, which is a contributory factor for worsening neurological and developmental outcomes. Early identification and surgical intervention are essential in limiting morbidity and mortality. This paper describes a case of a neonate with hydrocephalus and occipital meningoencephalocele, focusing on the importance of early diagnosis and intervention, cerebrospinal fluid diversion, and surgical repair. **Case Presentation**: A male infant was born by cesarean section with a giant occipital cystic mass noted at birth. Clinical exam showed hydrocephalus and newborn hyperbilirubinemia. Lab tests were notable for mild anemia, hypoalbuminemia, leukopenia, and hyperbilirubinemia.Imaging was consistent with an occipital defect and herniation of neural tissue consistent with meningoencephalocele. The infant underwent the placement of a ventriculoperitoneal (VP) shunt on DOL 5 for hydrocephalus and then stabilized in the NICU. The infant recovered from the procedure with stable neurological and systemic status, and gradual improvement observed. **Conclusion:** This case highlights the importance of early diagnosis and surgical management of hydrocephalus with occipital meningoencephalocele. Prompt intervention with VP shunt placement and planned excision of the encephalocele sac can improve survival and developmental outcomes. Comprehensive antenatal care, early neuroimaging, and multidisciplinary management remain essential in optimizing prognosis for affected neonates.

Keywords: *Occipital Meningoencephalocele, Hydrocephalus, Neural Tube Defect, Ventriculoperitoneal Shunt, Early Surgical Management*

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INTRODUCTION

Neural tube defects are developmental anomalies that arise due to incomplete closure of the neural tube during embryonic development; one common phenotype is meningoencephalocele, defined as herniation of brain tissue through a defect in the skull with associated meninges (Chmichi et al., 2020). Occipital encephaloceles are the most common phenotype of encephaloceles, representing about 75% of the total incidence of encephaloceles worldwide. Occipital encephalocele has a global prevalence of 0.8–4 per 10,000 live births, approximately. Hydrocephalus occurs in 40–60% of cases of occipital encephalocele and significantly increases morbidity (Chmichi et al., 2020). Clinically, occipital meningoencephalocele is presented at birth with a visible midline occipital bulge. The findings associated with the condition depends on the size of the sac and the involvement of neural tissue. Potential findings include increased head circumference, bulging fontanelle, seizures, developmental delay, visual impairment, and evidence of raised intracranial tension. Imaging, particularly MRI, is critical for defining the contents of the sac, evaluating other cranial anomalies, evaluating for hydrocephalus, and knowing how to repair surgically (Pal et al., 2021).

Management of occipital meningoencephalocele associated with hydrocephalus includes early surgical closure of the skull defect with watertight dural covering and when required with diversion of cerebrospinal fluid with a ventriculoperitoneal (VP) shunt. Outcomes are heavily influenced by timing of intervention, the presence and extent of neural tissue herniation, sac size, and associated anomalies such as microcephaly or other cranial malformations. In patients with hydrocephalus, normal developmental outcomes are less common (Chmichi et al., 2020; Punchak et al., 2025).

This case report aims to describe the clinical presentation, diagnostic evaluation, surgical management, and developmental outcome of a newborn with occipital meningoencephalocele complicated by hydrocephalus. Considering the high morbidity rates and variation in prognostic factors this case highlights the importance of early multidisciplinary evaluation, intervention, and follow-up for improving neurological and functional outcomes (Chmichi et al., 2020; Punchak et al., 2025).

CASE REPORT

A male neonate, By. M.R., born through cesarean on August 4, 2025, at 00:40 WIB to a G2P1A0 mother at 39 weeks' gestation with premature rupture of membranes, and prolonged first stage of labor. He was not crying at birth, with an Apgar score of 5/8. An obvious congenital swelling was present in the occipital region, described as a cystic mass with the overlying skin intact, and without any evidence of erythema. The neonate was admitted to Cileungsi Regional Hospital's Neonatal Intensive Care Unit (NICU) for management and further evaluation. Family history was negative for congenital anomalies and siblings had similar anomalies.

Maternal history revealed no exposure to radiation, alcohol, smoking, or teratogenic drugs during pregnancy. However, the mother admitted to inconsistent intake of folic acid supplements before and during early pregnancy, which is a known risk factor for neural tube defects such as meningoencephalocele and hydrocephalus. Antenatal care visits were performed regularly, but early trimester ultrasonographic screening was incomplete, resulting in the anomaly not being detected prenatally.

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On physical examination, the infant weighed 3700 g, with vital signs within normal limits (blood pressure 78/46 mmHg, pulse 136 bpm, respiratory rate 46/min, temperature 37°C, SpO_2 98% room air). General examination revealed an intact cystic mass in the occipital region. Cardiopulmonary and abdominal examinations were unremarkable. Cardiac auscultation revealed normal S1 and S2 with a systolic murmur, while pulmonary auscultation demonstrated vesicular breath sounds without adventitious findings. Extremities were warm with capillary refill <2 seconds.

Laboratory investigations revealed:

- Mild hyponatremia (Na 131 mmol/L; ref. 135–155 mmol/L), which may indicate dilutional effect due to fluid imbalance or associated central nervous system pathology.
- Hypoalbuminemia (3.4 g/dL; ref. 4.0–5.5 g/dL), suggesting impaired protein synthesis, malnutrition, or increased protein loss.
- Indirect hyperbilirubinemia (total bilirubin 15.2 mg/dL; direct bilirubin 0.7 mg/dL), consistent with neonatal hyperbilirubinemia, most likely due to immature hepatic conjugation mechanisms.

Hematological parameters showed:

- Leukopenia (4,800/µL; ref. 9,400–25,000/µL), which may reflect bone marrow suppression, viral infection, or systemic illness.
- Borderline low erythrocyte count (4.3 million/µL; ref. 4.76–6.95 million/µL), suggestive of mild anemia.
- Eosinophilia (5%; ref. 1–3%), which may be related to allergic response or parasitic predisposition.
- Monocytosis (10%; ref. 2–8%), often associated with chronic infection, recovery phase of infection, or inflammatory processes.

Table 1. Laboratory Examination (August 07, 2025)

Test Type/Parameter	Result	Reference Value
Liver Function		
Albumin	3.4 g/dl	4.0 - 5.5 g/dl
Elektrolytes		
Natrium (Na)	131 mmol/L	135 - 155 mmol/L

Table 2.Laborat2. Laboratoryory Examination (August 07, 2025)

Test Type/Parameter	Result	Reference Value
Hematological		
Leukocytes	4800/µL	9400 - 25000/µL
Erythrocytes	4.3 million/µL	4.76 - 6.95 million/µL
Diffcount		
Eosinophils	5 %	1 - 3 %
Monocytes	10 %	2 - 8 %

Imaging studies included chest radiographs showing right tracheal deviation, perihilar infiltrates consistent with pneumonia, and a soft-tissue opacity in the occipital region.

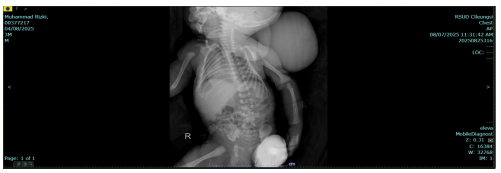

Figure 1. Chest X-ray, AP projection, supine position (August 7, 2025)

Chest X-ray in AP projection, supine position, with adequate inspiration

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and symmetry, shows radiological features of pneumonia, soft tissue density opacity in the occipital region, rightward deviation of the trachea, and a normal-appearing cardiac silhouette. No radiographic signs of ileus are observed.

The working diagnosis was *Hydrocephalus with Occipital Meningoencephalocele*. Differential diagnoses included meningocele, encephalocele, arachnoid cyst, and congenital tumor.

Surgical management consisted of ventriculoperitoneal shunt placement on August 9th, 2025, to address hydrocephalus. Operative findings included successful Kocher's point ventriculostomy with proximal catheter insertion, creation of a distal peritoneal tunnel, and secure placement of the VP shunt system. The infant tolerated the procedure well, with no intraoperative complications.

Table 3. Laboratory Examination Pre Operative (August 09, 2025)

Test Type/ Parameter	Result	Reference Value
Hematological		
Hemoglobin	**14.0 g/dL**	**15.0 - 24.6 g/dL**
Leukocytes	**7800/µL**	**9400 - 25000/µL**
Hematocrit	43%	40 - 48%
Trombosit	310.000/µL	150.000 - 450.000/µL
Liver Function		
Albumin	**3.4 g/dL**	**4.0 - 5.5 g/dL**
Diabetes		
Glucose	95 mg/dL	< 200 mg/dL
Elektrolit		
Natrium (Na)	139 mmol/L	135 - 155 mmol/L
Potassium (K)	3.5 mmol/L	3.5 - 5.5 mmol/L
Chloride (Cl)	**110 mmol/L**	**95 - 108 mmol/L**

Laboratory investigations revealed:

- **Mild anemia** (hemoglobin 14.0 g/dL; reference 15.0–24.6 g/dL), possibly due to chronic illness, nutritional deficiency, or early blood loss.
- **Leukopenia** (7,800/µL; reference 9,400–25,000/µL), suggesting decreased immune cell count, which could be related to systemic illness, bone marrow suppression, or infection.
- **Hypoalbuminemia** (3.4 g/dL; reference 4.0–5.5 g/dL), which may indicate poor nutritional status, impaired liver synthesis, or ongoing protein loss.
- **Hyperchloremia** (Cl 110 mmol/L; reference 95–108 mmol/L), which may suggest mild electrolyte imbalance, possibly related to dehydration,

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renal handling of electrolytes, or acid-base disturbance.

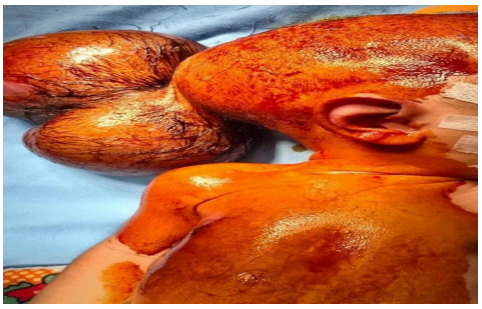

Figure 2. Patient intraoperative ventriculoperitoneal (VP) shunt placement

Postoperatively, the patient was managed in the NICU with intravenous antibiotics (cefotaxime, amikacin), supportive therapy, and close monitoring of neurological and cardiorespiratory status. Daily follow-up showed gradual improvement in respiratory function, resolution of jaundice, and stable vital signs. The occipital cyst remained intact without signs of leakage or infection. Nutritional intake was progressively advanced.

Follow-up chest radiographs post operative revealed normally expanded lungs, appropriate placement of endotracheal and gastric tubes, and the presence of a ventriculoperitoneal (VP) shunt, though proximal and distal tips were not visualized.

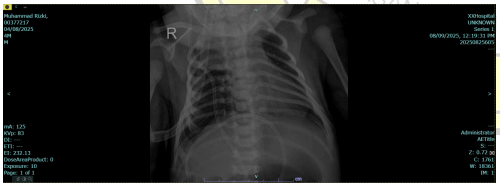

Figure 3. Chest X-ray, AP projection, supine position (August 9, 2025)

Chest X-ray in AP projection, supine position pshows clear lung fields without abnormality, normal cardiac configuration, endotracheal tube in place with distal tip located 15 mm above the carina, gastric tube in place with distal tip projected at the middle third of the esophagus, and ventriculoperitoneal (VP) shunt in situ with proximal and distal tips not visualized.

Table 4. Laboratory Examination Post Operative (August 11, 2025)

Test Type/Parameter	Result	Reference Value
Hematological		
Hemoglobin	**13.9 g.dL**	**15.0-24.6 g/dL**
Leukocytes	5500/µL	5000 - 19500/µL
Hematocrit	42%	40 - 48%
Thrombocytes	418000/µL	418000/µL
Diabetes		
Glucose	99 mg/dL	< 200 mg/dL
Liver Function		
Total Bilirubin	**15.2 mg/dL**	**0.3 mg/dL**
Direct Bilirubin	**0.7 mg/dL**	**0 - 0.2 mg/dL**

Laboratory investigations revealed:

- Mild anemia (hemoglobin 13.9 g/dL; reference 15.0–24.6 g/dL), which may reflect perioperative blood loss or dilutional changes following surgery.
- Leukopenia (5,500/µL; reference 5,000–19,500/µL), still within the lower normal range, possibly indicating transient postoperative immune suppression or bone marrow response.
- Thrombocytosis (418,000/µL; reference 150,000–450,000/µL), which may represent a reactive process secondary to surgery, inflammation, or tissue repair.
- Indirect hyperbilirubinemia (total bilirubin 15.2 mg/dL; direct bilirubin 0.7 mg/dL), consistent with neonatal hyperbilirubinemia, most likely

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due to immature hepatic conjugation mechanisms

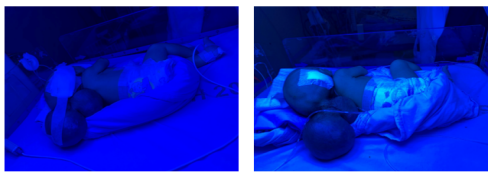

Figure 4. Postoperative condition of the patient in the NICU

The prognosis was assessed as *dubia ad bonam ad vitam*, given the successful VP shunt placement reducing intracranial pressure. However, *ad functionam* prognosis remained *dubia* because long-term neurological outcomes depend on the amount of neural tissue within the sac and the extent of prior hydrocephalus-induced brain injury. *Ad sanationam* was also *dubia*, since definitive cure is not achievable for congenital structural anomalies, although corrective surgery can improve quality of life and developmental potential.

The patient was discharged from the NICU on August 14th, 2025, in stable condition, with recommendations for nutritional optimization and outpatient follow-up for definitive repair of the occipital meningoencephalocele once the infant's body weight exceeds 5 kg.

DISCUSSION

Clinical Manifestations

Occipital meningoencephalocele frequently presents at birth with a visible occipital mass covered by skin, containing meninges, CSF, and often herniated neural tissue (Nagy & Saleh, 2021). Hydrocephalus complicates roughly 40-60% of occipital encephaloceles, being significantly more common in posterior defects than in frontal ones, and is associated with worse developmental outcomes (Nagy & Saleh, 2021; Punchak et al., 2025). In newborns, the clinical manifestation may include an enlarged head or rapidly increasing head circumference, bulging fontanelles, and tense scalp over the defect, reflecting elevated intracranial pressure (Nagy & Saleh, 2021). Other symptoms can include seizures, motor deficits such as spasticity, and delayed development or acquisition of developmental milestones and visual impairment (Punchak et al., 2025). A larger encephalocele sac size and identifiable neural tissue within the sac have been shown to correlate with an increased risk of hydrocephalus and, ultimately, neurological morbidity (Nagy & Saleh, 2021; Punchak et al., 2025).

Neonates presenting with occipital meningoencephalocele may experience feeding intolerance, respiratory compromise, and recurrent infections secondary to the mass effect from the defect and resultant neurological impairment. Developmental delay, especially in motor and cognitive domains, is frequent; moreover, the prognosis may be affected by the development of associated hydrocephalus and/or the volume of neural tissue contained in the sac (Nagy & Saleh, 2021; Punchak et al., 2025). Visual impairment may be caused by stretching or compression of the optic pathways leading to strabismus, or, in some cases, blindness (Nagy & Saleh, 2021). In the most severe situations, untreated hydrocephalus can lead to progressive macrocephaly, irritability, vomiting, and even brainstem failure, which can be life threatening (Punchak et al., 2025) The presence of hydrocephalus associated with occipital meningoencephalocele is complex because both structural and non-structural abnormalities contribute to morbidity and emphasize the need for early surgical intervention and long-term interdisciplinary care (Punchak et al., 2025).

Classification

There are a number of classifications for occipital

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meningoencephalocele. Anatomically, encephaloceles are classified by the location of the cranial defect: occipital, frontal, parietal, basal, or other rare variants, with the occipital variant being the most prevalent variant worldwide, accounting for up to 75% of occipital meningoencephalocele in Asia and Africa (Protzenko et al., 2021). On the basis of the sac contents, lesions can be classified as meningocele (meninges and CSF), meningoencephalocele (meninges and brain), or hydroencephalomeningocele (meninges, brain, and a portion of the ventricular system) (Mustafa et al., 2023). Our patient had neural tissue which protruded along with meninges, therefore is classified as a meningoencephalocele (Mustafa et al., 2023; Protzenko et al., 2021).

Prognostically, risk stratification depends on several factors: sac size (small vs. giant), presence of hydrocephalus, amount of neural tissue herniation, and associated anomalies. Patients with small sacs and no neural tissue typically have favorable outcomes, while those with hydrocephalus, large sacs, or significant neural content as in our case are considered **high-risk** with guarded prognosis (Mustafa et al., 2023; Protzenko et al., 2021; Punchak et al., 2025). Outcome-based classifications also emphasize neurodevelopmental function: mild (normal milestones with or without minor deficits), moderate (delays in motor or cognitive development requiring rehabilitation), and severe (profound neurological impairment or early mortality). Given the coexistence of hydrocephalus and herniated brain tissue, our patient would most appropriately be categorized under the **moderate-to-severe group** with increased risk of long-term deficits (Protzenko et al., 2021).

From a functional perspective, surgical outcome classifications describe survival with good neurological function, survival with deficits, or non-survival. Early surgery with watertight dural closure and correction of CSF dynamics has been shown to shift outcomes toward improved survival and reduced complications. However, associated anomalies such as microcephaly, corpus callosum agenesis, and severe hydrocephalus remain strong predictors of poor prognosis (Mustafa et al., 2023; Punchak et al., 2025).

Risk Factors

Neural tube defects such as meningoencephalocele are strongly influenced by maternal and environmental factors. One of the most important risk factors is inadequate maternal folate intake during the periconceptional period and early pregnancy, which has been consistently linked to impaired neural tube closure and the development of encephalocele and associated hydrocephalus (Mustafa et al., 2023). A further increase in risk, due to advanced maternal age and lack of antenatal surveillance, including lack of early ultrasound screening, allowing for delays in diagnosing congenital anomalies (Abebe et al., 2022). In our patient, the mother did not receive adequate folic acid supplementation and incomplete antenatal follow-up, which may have contributed to the occipital meningoencephalocele with secondary hydrocephalus.

Other maternal risk factors reported in the literature include infections during early gestation, specifically TORCH infections, which can interfere with neurodevelopment in the embryo (Isaković et al., 2022). Exposure to teratogenic drugs, specifically sodium valproate, as well as any folate-antagonist antiepileptic drug, have been associated with neural tubal defects (Isaković et al., 2022; Mustafa et al., 2023). Although the mother didn't note any drug exposure, poor nutritional

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intake in the mother was present during pregnancy, likely due to socioeconomic factors. In addition, the location of the encephalocele and the size of the defect have prognostic implications; occipital encephaloceles, particularly those of neural tissue intact, carry a higher risk of hydrocephalus and poorer neurological outcomes compared with anterior defects (Abebe et al., 2022; Isaković et al., 2022).

Diagnosis

The diagnosis of occipital meningoencephalocele with associated hydrocephalus requires an integrated approach involving perinatal history, focused physical examination, laboratory testing when infection is suspected, and multimodality neuroimaging. Antenatal ultrasound often allows prenatal detection by demonstrating a skull defect with a posterior cranial mass, but detailed assessment of intracranial contents and associated anomalies is best performed with fetal MRI, which clarifies sac contents (meninges, neural tissue, ventricular components), venous anatomy, and concurrent brain malformations (Copp & Greene, 2013).

Clinically, affected neonates typically present with a visible occipital mass at birth; when hydrocephalus is present there may be increasing head circumference, tense/bulging fontanelle, visible scalp veins, sunset eyes, poor feeding, vomiting, irritability, and seizures—signs that reflect raised intracranial pressure in infancy and warrant urgent imaging (Punchak et al., 2025).

Postnatal neuroimaging strategy prioritises MRI as the modality of choice for defining the extent of parenchymal herniation, evaluating associated intracranial malformations, assessing for intrathecal/ventricular communication, and detecting complications such as periventricular leukomalacia or brainstem involvement. CT (including bone window) is complementary when detailed assessment of the calvarial defect or preoperative planning for bone reconstruction is required; cranial ultrasound is useful at the bedside for rapid detection of ventriculomegaly in neonates and for serial monitoring of ventricular size when MRI/CT are not immediately available (Pal et al., 2021; Punchak et al., 2025).

Laboratory testing has a limited role in establishing the diagnosis but is critical when infection is suspected. Non–skin-covered encephaloceles or those with CSF leakage have an increased risk of meningitis; in such cases cerebrospinal fluid analysis typically shows pleocytosis and biochemical changes consistent with meningitis, and culture/PCR should be obtained before antimicrobial therapy when feasible (Copp & Greene, 2013). Postoperative CSF leak likewise markedly increases meningitis risk and must be actively sought and managed (Mustafa et al., 2023; Nagy & Saleh, 2021).

Differential diagnoses to be considered include meningoceles (meninges + CSF without brain tissue), cystic posterior scalp lesions (e.g., cephalocele vs. dermoid/epidermoid cyst), cystic hygroma, and other posterior fossa masses; distinction depends on imaging demonstration of a calvarial defect and intracranial continuity. The coexistence of hydrocephalus—detected by ventriculomegaly on ultrasound/CT/MRI strengthens the diagnosis of a communicative or obstructive CSF dynamic disturbance related to the encephalocele and guides need for CSF diversion planning (Abebe et al., 2022; Pal et al., 2021; Protzenko et al., 2021).

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Treatment

The management of hydrocephalus associated with occipital meningoencephalocele involves prompt surgical repair of the encephalocele sac along with careful management of cerebrospinal fluid (CSF) dynamics. In almost all reported series, excision and closure of the encephalocele sac (including watertight dural closure and skull defect repair) is the cornerstone of therapy, preferably in the neonatal period or early infancy, as early surgery is associated with fewer complications and better outcomes for neurodevelopmental function (Nagy & Saleh, 2021).

When hydrocephalus is present (either preoperatively or develops postoperatively), diversion of CSF via a ventriculoperitoneal (VP) shunt is frequently required. In one case series of 17 children with occipital encephalocele, 11 (approximately 65%) had hydrocephalus, and 10 of them (≈91%) were managed using VP shunts (Nagy & Saleh, 2021; Punchak et al., 2025). Endoscopic third ventriculostomy (ETV) has been considered in selected cases but is less favored in very young infants due to underdeveloped CSF absorption pathways and higher risk of failure (Mustafa et al., 2023; Punchak et al., 2025).

Surgical indications include: large encephalocele sac size, presence of neural tissue within the sac (which increases risk of neurological deficits), skin compromise of the sac (e.g. thin or ulcerated skin, risk of CSF leak), evidence of infection, and associated anomalies (cranial or extracranial) that may complicate outcomes (Mustafa et al., 2023; Pal et al., 2021; Punchak et al., 2025).

Complications and Prognosis

Occipital meningoencephalocele is associated with significant morbidity and mortality; complications are frequent and the prognosis is variable. Mortality rates in some case series range between 29–33%, especially in patients with large defects, postoperative infection, or delayed presentation (Mustafa et al., 2023). Frequent postoperative complications include CSF leakage, wound infection, meningitis, and development or worsening of hydrocephalus due to elevated intracranial pressure (Ngowi et al., 2024). Neurological sequelae such as developmental delay, seizure disorders, visual impairment, microcephaly, and motor deficits are common, particularly when neural tissue is contained in the sac or when hydrocephalus is present (Mustafa et al., 2023; Parmar et al., 2021). Predictors of poor outcome include large sac size, presence of neural tissue within the sac, associated intracranial anomalies (e.g. microcephaly), and delayed repair (Ngowi et al., 2024).

Prognosis is often considered **dubia ad bonam** ranging from doubtful to good for survival, neurodevelopment, and functional recovery, depending on how timely and effective the surgical intervention is, the extent of brain involvement, and postoperative care (Mustafa et al., 2023; Ngowi et al., 2024). Good outcomes defined as adequate developmental progress and minimal neurological deficits are reported in **40–50%** of survivors when risk factors are favorable (Ngowi et al., 2024; Parmar et al., 2021).

CONCLUSION

This case highlights the importance of early diagnosis and timely surgical management of hydrocephalus associated with occipital meningoencephalocele to optimize neurodevelopmental outcomes and reduce the risk of life-threatening complications. Although the coexistence

of hydrocephalus and neural tissue herniation is associated with higher morbidity and a guarded prognosis, prompt intervention with excision and repair of the encephalocele sac and cerebrospinal fluid diversion procedures, such as ventriculoperitoneal shunting, can significantly improve survival and functional recovery. The case also underscores the need for careful antenatal screening, postnatal neuroimaging, and multidisciplinary care including neurosurgery, neonatology, and rehabilitation services to provide comprehensive management and long-term follow-up for affected infants.

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