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Paediatric Medulloblastoma: A Case Report

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Abstract: ***Medulloblastoma is the most common malignant brain tumour in children, comprising 12% to 25% of paediatric brain tumours. Despite its rarity in adults, it poses significant challenges in paediatric neuro-oncology due to its aggressive nature. Classified as a WHO Grade IV embryonal tumour, it originates in the cerebellum and tends to grow rapidly, with a high risk of spreading through cerebrospinal fluid. This case report outlines the diagnosis and management of a child with cerebellar medulloblastoma presenting with extensive nodularity and obstructive hydrocephalus. Initial symptoms were vague, leading to delays in recognition until rapid clinical deterioration necessitated immediate neuroimaging and surgical intervention. A multidisciplinary team performed a resection to relieve intracranial pressure and remove as much tumour as safely possible, followed by a personalized chemotherapy regimen based on the tumour's characteristics. A distinguishing feature of this case was the inclusion of a Public Health Nurse in the post-discharge care plan. The nurse provided ongoing neurological monitoring, supported treatment adherence, and guided the family through the recovery phase. This partnership between clinical and community-based care teams ensured continuity, reinforced family support, and contributed to positive health outcomes. The report highlights the impact of coordinated care that bridges hospital treatment with community follow-up. Early intervention and collaborative support systems were essential in achieving clinical stability and optimizing the child's recovery and neurological function.***

Keywords: Medulloblastoma, Multidisciplinary care

1. Introduction

Medulloblastoma is the most common malignant brain tumour in children, comprising approximately 12% to 25% of all paediatric brain tumours while being exceedingly rare in adults[1]. Classified as a World Health Organisation (WHO) Grade IV embryonal tumour[2][3], it primarily arises in the cerebellum from undifferentiated neuroectodermal cells—primitive cells with high proliferative potential that fuel the tumour's rapid growth and aggressive nature[4]. Medulloblastoma accounts for nearly 20% of all paediatric brain tumours[3]and its management is complex and requires a multidisciplinary approach to ensure optimal outcomes.
This case report details the journey of a young child diagnosed with cerebellar medulloblastoma, WHO Grade IV, with extensive nodularity and hydrocephalus. The report highlights the challenges of early diagnosis, surgical intervention, and post-operative care, emphasizing how effective treatment was achieved through surgery, chemotherapy, and diligent follow-up at home by the Public Health Nurse.

History

The term *medulloblastoma cerebelli* was first introduced by Bailey and Cushing in 1925 to describe a distinct type of fast-growing, small-cell tumour located in the centre of the cerebellum[5][6]. This classification remained largely unchanged for decades. In 1983, Rorke, along with Becker and Hinton, proposed a broader approach—suggesting that all aggressive small-cell brain tumours, including medulloblastomas, be grouped under the term *primitive neuroectodermal tumours* (PNETs) and classified based on their location in the brain[7].

However, advances in clinical research and molecular biology led to a significant shift in how these tumours are understood. In 2016, the World Health Organization (WHO) revised its classification system, recognizing that medulloblastomas of the cerebellum are biologically and clinically distinct from PNETs found in other brain regions. As a result, medulloblastomas are now classified as separate entities, reflecting their unique molecular profiles and behaviours. This change has important implications for diagnosis, treatment, and prognosis, especially in paediatric neuro-oncology[7].

Incidence and Prevalence

Globally, the incidence of medulloblastoma is estimated at 0.5 to 0.7 per 100,000 children annually[7]. In India, though exact national epidemiological data are limited, several institutional studies report similar patterns, with medulloblastoma consistently ranking among the top paediatric brain tumours treated at tertiary centers[1]. Age-specific occurrence highlights a strong predilection for younger age groups, with the majority of cases diagnosed between the ages of 3 and 8 years[1]. While medulloblastoma can occur at any age, it is rare in adults[7] and infants younger than 3 years tend to present with more aggressive forms and atypical symptoms. Peak incidence is typically observed between 5 and 7 years of age[7], and it is more common in males than females[8][2] with a male-to-female ratio of approximately 1.9:1[2].

Risk Factors

The exact cause of medulloblastoma remains unclear; however, several risk factors have been identified, particularly related to genetic predisposition[2][9]. One of the most significant is the presence of hereditary cancer

syndromes involving germline mutations in specific signalling pathways[2].

WNT pathway mutations, such as *APC* gene alterations found in Turcot syndrome, are strongly associated with the development of WNT subtype medulloblastomas. Similarly, germline mutations in genes related to the Sonic Hedgehog (SHH) pathway—such as *PTCH1* (linked to Gorlin syndrome), *SUFU*, *TP53* (Li-Fraumeni syndrome), and *SMO* (Curry-Jones syndrome)—are associated with SHH subtype medulloblastomas[9][4]. While other cancer predisposition syndromes, including Fanconi anaemia, are also linked to medulloblastoma, the associated risk is generally lower compared to the WNT and SHH pathways[2][10].

Beyond genetic factors, some studies suggest environmental or prenatal influences may contribute to medulloblastoma development. Associations have been reported between maternal diet, immune system, or hematologic disorders during pregnancy, and an increased risk of brain tumours in offspring. Additionally, early exposure to certain viral infections, such as John Cunningham (JC) virus or human cytomegalovirus (CMV), has been proposed as a possible risk factor in some cases[3].

Although rare, familial clustering of medulloblastoma has also been documented, further supporting a genetic contribution to its pathogenesis. Overall, while definitive environmental causes remain elusive, current evidence emphasizes the importance of hereditary syndromes, particularly those affecting the WNT and SHH signalling pathways, in increasing susceptibility to medulloblastoma.

Pathophysiology

Medulloblastoma is thought to begin in granule cell precursors found in the external germinal layer (EGL) of the developing cerebellum. It usually starts in the area around the fourth ventricle of the brain and can grow to fill this space. As the tumour grows, it can spread to nearby areas like the cerebellar vermis and brainstem[11]. It may also travel through the cerebrospinal fluid (CSF) along the brain and spinal cord—a type of spread known as leptomeningeal or "drop" metastasis. This pattern reflects the tumour's aggressive nature, with both local invasion and the ability to spread throughout the central nervous system (CNS)[3].

Spread of medulloblastoma outside the brain and spinal cord (called extra neural metastasis) is rare in children, occurring in about 7% of cases. When it does happen, it most commonly affects the bones (78%), lymph nodes (33%), liver (15%), and lungs (11%). These distant metastases usually appear around 20 months after the initial surgery and are linked to poor outcomes, with survival often less than six months after diagnosis[3].

On a genetic level, one of the most common abnormalities seen in medulloblastoma is the presence of isochromosome 17q. This involves the loss of the short arm of chromosome 17 (17p) and duplication of the long arm (17q). A related finding, known as 17p loss of heterozygosity (17pLOH), is also frequently observed. Although the *TP53* gene—a key tumour suppressor located on 17p—is rarely mutated in medulloblastoma, scientists believe other important tumour suppressor genes in this region may play a role in the disease. Ongoing research is focused on identifying these genes to better understand how medulloblastoma develops and progresses[3].

2. Signs and symptoms

Medulloblastoma often presents with neurological symptoms due to its infratentorial origin and rapid growth. Typically arising in the cerebellum and extending into the fourth ventricle, it frequently obstructs cerebrospinal fluid (CSF) pathways, resulting in obstructive hydrocephalus and intracranial hypertension. Early clinical features include morning-predominant vomiting and headaches exacerbated in the recumbent position[8][9].

Visual disturbances such as diplopia, lateral gaze palsy, and nystagmus may occur due to increased intracranial pressure and cranial nerve involvement. Papilledema observed on fundoscopy further supports a diagnosis of raised intracranial pressure[10][8]. Cerebellar dysfunction manifests as truncal ataxia, gait instability, and impaired coordination[3]. As the tumour enlarges, compression of adjacent structures may lead to facial asymmetry, reduced consciousness, and, in infants, bulging anterior fontanelles[12].

In younger patients, particularly infants, clinical signs may be subtle and nonspecific[12]. Presenting features can include lethargy, poor weight gain, or rapidly increasing head circumference. These age-related differences underscore the need for a high index of suspicion to ensure prompt diagnosis and prevent long-term neurological complications[12]

Classification of Medulloblastoma (WHO 2016)

Over the years, understanding of its biology has evolved significantly, leading to a classification system that integrates both microscopic features and molecular profiles. The 2016 World Health Organisation (WHO) classification of central nervous system tumours [2] introduced a major shift by combining histological subtypes with molecular subgroups to form an integrated diagnosis. This approach improves diagnostic accuracy, enhances risk stratification, and guides treatment decisions more effectively.

Histological Classification (WHO 2016)

Based on traditional microscopic appearance, medulloblastoma is divided into the following histological types[13]:

- Classic Medulloblastoma – Characterized by densely packed small round cells and high mitotic activity.
- Desmoplastic/Nodular (DN) – Shows nodular architecture and internodular desmoplasia, often with better outcomes in young children.
- Medulloblastoma with Extensive Nodularity (MBEN) – A variant of DN, typically seen in infants and associated with a favourable prognosis.
- Large Cell/Anaplastic (LCA) – Displays marked nuclear pleomorphism and increased mitoses, often linked with a more aggressive course.
- Medulloblastoma, Not Otherwise Specified (NOS) – Used when specific subtyping is not possible due to inadequate tissue or testing limitations.

Molecular Classification (WHO 2016)

Parallel to histological typing, molecular subgrouping plays a vital role in understanding tumour biology. The four principal molecular groups[5] identified are:

a) WNT-activated
Tumours driven by activation of the WNT signalling pathway. These typically occur in older children and have an excellent prognosis.

b) SHH-activated
Tumours involving the Sonic Hedgehog pathway. This group is further divided based on TP53 status:

- SHH-activated, TP53-wildtype
- SHH-activated,
- TP53-mutant

SHH tumours are more common in infants and adults, and TP53 mutation is associated with a poorer prognosis[4].

c) Non-WNT/Non-SHH

This category includes:

- Group 3 (G3) – Often shows MYC amplification, commonly presents in infants and young children and is associated with a poor prognosis.
- Group 4 (G4) – The most prevalent subgroup, typically affecting older children. It has an intermediate prognosis and is characterized by distinct chromosomal changes such as isochromosome 17q[14].

The WHO 2016 classification encourages an integrated diagnostic format that includes:

- Histological type
- WHO grade (medulloblastoma is WHO Grade IV)
- Molecular subgroup

This framework allows to better correlate the tumour's biology with clinical behaviour, enabling more accurate predictions of treatment response and long-term outcomes. Recent advances in methylation profiling have further refined molecular classification, supporting individualised therapy in medulloblastoma.

Diagnosis

Diagnosis of Medulloblastoma relies on clinical symptoms and advanced imaging techniques[4]. Key presenting symptoms often include morning headaches, nausea, vomiting, fatigue, and balance issues, typically arising from increased intracranial pressure and cerebellar dysfunction. Clinical Examination begins with a thorough neurological assessment to identify signs of intracranial pressure elevation and cerebellar involvement. Fundoscopy is performed early to check for papilledema, indicating raised intracranial pressure, before performing a lumbar puncture (LP)[3].

CT scan is often the first imaging modality used due to its accessibility. It can reveal contrast-enhancing lesions and hydrocephalus but lacks the detail required for accurate staging [8].MRI is considered the gold standard for Medulloblastoma diagnosis due to its superior anatomical resolution. It is essential for evaluating tumour size, location, and potential spread via the subarachnoid space. Postoperatively, MRI is crucial for assessing residual tumours[3][8].

Spinal MRI is performed to check for metastasis, ideally before surgery or at least two weeks afterward to avoid false results due to surgical manipulation. A lumbar puncture (LP) is typically conducted 2–4 weeks post-surgery to assess for leptomeningeal dissemination, although CSF cytology may not always correlate with radiologic findings. To prevent false positives, LP is delayed until at least two weeks after surgery. This timing also allows for accurate evaluation of any CSF involvement[3].

Before initiating chemotherapy, baseline assessments such as audiometry, echocardiography, and pulmonary function tests are essential, especially to evaluate potential side effects of cisplatin treatment[3]

Approximately 5% of MB patients harbour germline mutations in cancer susceptibility genes such as APC, BRCA2, PALB2, PTCH1, SUFU, and TP53. As a result, genetic testing has become an integral part of the diagnostic process, offering valuable information for both treatment planning and familial risk assessment. The increasing focus on molecular profiling allows for more personalized treatment approaches, particularly for patients with unique genetic predispositions[18].

Treatment:

When Bailey and Harvey Cushing described Medulloblastoma in 1929, surgical intervention was considered the primary method of treatment at that time[2][5]. Over the decades, treatment strategies have evolved significantly, transitioning to a multimodal approach that now includes surgery, radiation, and chemotherapy[15][16]. This progression has substantially improved survival rates, making the current standard of care highly effective.

Initially, radiation therapy was used intermittently in the treatment of MB. A breakthrough occurred in 1953 when a study demonstrated significantly improved survival rates with the use of craniospinal radiation (CSI). This approach became the standard treatment, helping to increase survival rates to around 60%[2]. The addition of chemotherapy in the 1970s further enhanced survival outcomes, with five-year survival rates rising to 70–80% for standard-risk patients and 60–65% for high-risk patients[2][17].

In recent decades, molecular genetics has provided profound insights into the pathophysiology of MB. Gene expression studies revealed that MB is not a single disease but consists of at least four molecular subgroups: WNT, SHH (Sonic Hedgehog), Group 3, and Group 4. Each subgroup is associated with distinct genetic drivers, clinical features, and outcomes, underscoring the need for personalized treatment[16]. This molecular understanding has spurred interest in tailoring treatment regimens to minimize the side effects of chemotherapy and radiation while maximizing therapeutic efficacy.

The cornerstone of MB treatment remains surgical resection, which serves both as a diagnostic tool and as a means to alleviate pressure on the brainstem and resolve obstructive hydrocephalus[15]. Following surgery, patients typically undergo craniospinal irradiation (CSI) and chemotherapy. Recent advancements in radiotherapy techniques—such as

tomotherapy, volumetric modulated arc therapy (VMAT), and proton therapy—promise to enhance the therapeutic ratio by reducing long-term toxicities, particularly in paediatric patients. Despite these innovations, CSI continues to be a fundamental component of MB treatment[15].

Post-surgical treatment is based on risk stratification, categorizing patients into standard-risk (SR-MB) and high-risk (HR-MB) groups. High-risk patients, including those with tumour remnants larger than 1.5 cm^2, anaplastic histology, metastatic disease, positive cytology, or MYC gene amplification, receive 36 Gy of CSI, with additional radiation to the tumour bed. Combined radiation and chemotherapy have resulted in a five-year survival rate ranging from 60% to 85%[17]. However, all patients experience some degree of side effects from these treatments. For children under three years of age, chemotherapy alone is often preferred to mitigate the neurocognitive consequences of radiation.

The treatment of medulloblastoma has progressed remarkably from its early surgical roots to a more sophisticated, multimodal approach. While the overall cure rate for MB stands at approximately 70%, high-risk patients still face considerable challenges. Personalized treatment, based on molecular profiling and genetic testing, is emerging as a promising strategy to improve patient outcomes and reduce the long-term side effects associated with conventional therapies.

Prognosis

Medulloblastoma (MB), the most common malignant primary brain tumour in children, has seen significant advancements in both biological understanding and treatment techniques over the past decade. However, despite substantial improvements in patient outcomes, there remain disparities in survival rates, particularly between high- and low-resource settings.

A study conducted over ten years at a medical centre involving fifty-eight patients revealed several factors that significantly influence prognosis. These include the patient's age, tumour histology, stage of the disease, the surgical method employed, the patient's performance status, and the period during which treatment was administered—reflecting the evolution of treatment protocols. Notably, in the latter half of this period, there was a shift towards more aggressive surgical procedures and chemotherapy, alongside enhancements in radiation techniques, which contributed to better survival outcomes[1].

The five-year survival rate for MB varies, ranging from 50% to 90%. This variability is influenced by factors such as age at diagnosis, tumour burden, presence of metastases, and the histological subtype of MB[19]. Treatment strategies are adapted to these variables, with the primary goal of achieving maximal safe resection followed by chemotherapy and/or radiation. Despite advances in treatment, high-risk cases continue to present significant challenges, and ongoing research aims to refine therapeutic strategies to further improve outcomes[17][13].

However, the success rates seen in Western countries do not always translate to developing nations such as India. Several factors contribute to this disparity, including socio-demographic challenges, variations in healthcare access, and inconsistent treatment approaches. Delays in diagnosis and referral, coupled with limited resources and specialized care, contribute to poorer outcomes in these regions. Inadequate access to timely surgical interventions and adjuvant therapies, such as radiation and chemotherapy, further exacerbate this issue. These delays, along with increased morbidity and mortality due to advanced disease at presentation, emergency surgeries, and infections, result in significant challenges in managing MB effectively[16].

Despite advances in multimodal treatment, including surgery, radiation, and chemotherapy, approximately 30% of patients still succumb to the disease[2][2], and survivors often face long-term side effects. These side effects can severely impact quality of life, with secondary malignancies and chronic medical conditions contributing to a 15-year mortality rate of over 20%[20].

Children under the age of three are considered high-risk due to the potential neurocognitive effects of treatment. However, certain histological variants, such as desmoplastic MB and MB with extensive nodularity (MBEN), have a more favourable prognosis, even in younger children. Tumour histology plays a critical role, as the large cell/anaplastic variant of MB is associated with aggressive disease and poorer outcomes due to its rapid progression.

The extent of disease at diagnosis is another critical factor. High-risk features include the presence of residual tumour greater than 1.5 cm^2 after surgery, along with evidence of metastatic spread to the brain and spinal cord. These features are linked to a higher likelihood of relapse and poorer survival rates. Additionally, the biological and molecular characteristics of the tumour are vital for risk stratification. Subgroups such as Group 3, Group 4, and Sonic Hedgehog (SHH) medulloblastomas with TP53 mutations are considered high-risk and associated with worse outcomes[18]. In contrast, SHH medulloblastomas with wild-type TP53 and the WNT subgroup are linked to lower risk and better long-term prognosis[4][15].

In developing countries, survival rates for MB patients, particularly in high-risk groups, tend to be lower than those observed in high-income nations. However, some centres in India have reported 5-year survival rates of 62% in high-risk cases, comparable to outcomes in Western countries, demonstrating the potential for improved outcomes with advancements in treatment and healthcare access[20].

While considerable progress has been made in the treatment of medulloblastoma, challenges remain, particularly in resource-limited settings. Effective management requires a multidisciplinary approach, including expertise in neurosurgery, neuroradiology, molecular biology, paediatric oncology, and rehabilitative services. Understanding the role of molecular subgroups and patient-specific factors in prognosis will be crucial in further refining treatment strategies and improving survival rates worldwide[17][18].

3. Case Report

A one-year- eight-month-old girl child presented with a history of vomiting for several months. The child had no significant prior medical history or family history of neuro-oncological diseases. Her symptoms progressively worsened, prompting further investigation.

On clinical examination, the child exhibited no gross sensory-motor deficit but was irritable and crying. A non-contrast CT scan of the brain was conducted, which revealed hydrocephalus with a marked enlargement of the ventricles. Given the suspicious nature of the findings, an MRI of the brain was performed, confirming the presence of a large posterior fossa mass, consistent with a cerebellar medulloblastoma. The tumour was noted to be characterized by extensive nodularity, a feature that raised suspicion for a desmoplastic variant, further confirmed by histopathological analysis.

To alleviate the symptoms of hydrocephalus, the patient underwent an urgent ventriculoperitoneal (VP) shunt insertion. This procedure was aimed at relieving the increased intracranial pressure and stabilizing the patient's condition before definitive tumour management. The VP shunt was successful in controlling the hydrocephalus and improving the child's clinical state.

After the stabilization of his condition, the patient underwent a surgical resection of the tumour. The procedure involved a near-total removal of the cerebellar mass, which was confirmed to be a medulloblastoma through histopathological examination. The biopsy revealed the tumour to be a WHO grade 4 medulloblastoma with extensive nodularity, characteristic of the desmoplastic variant. Immunohistochemical analysis supported the diagnosis and excluded other differential diagnoses.

Following the surgery, the patient was started on chemotherapy (5 cycles), consisting of a combination of cyclophosphamide, vincristine, and cisplatin. This approach was chosen based on the patient's risk stratification and the need for adjuvant therapy to address potential micro metastasis and reduce the risk of recurrence.

However, the postoperative course was complicated by a wound infection, which was promptly identified and managed with appropriate antibiotics. The infection was effectively controlled, and the patient was monitored closely for any further complications. Following the resolution of the infection, the child continued his chemotherapy regimen without further issues.

The patient is currently under regular follow-up, with routine imaging and clinical assessments. At the 6-month follow-up, there were no signs of recurrence or significant adverse effects from the treatment. The VP shunt remains in place to manage any residual hydrocephalus, and the patient's neurological development is being closely monitored.

Nursing Care Plan

The child has completed the primary treatment for medulloblastoma and is currently under follow-up by the Public Health Nurses at home. Despite being cleared of the disease, ongoing follow-up care in the community is essential for monitoring long-term recovery, managing potential side effects, and ensuring the child's overall well-being. This nursing care plan focuses on providing continuous care and support to the child and family during the post-treatment phase, with an emphasis on community-based follow-up.

Nursing Diagnosis & Interventions

Ineffective Cerebral Tissue Perfusion related to increased intracranial pressure secondary to medulloblastoma

- Monitor neurological status hourly (GCS score, pupil size and reaction, limb strength).
- Elevate the head end 30 degrees to promote venous drainage from the brain.
- Maintain neutral head and neck alignment to avoid kinking of venous return.
- Administer prescribed medications: Osmotic diuretics, Corticosteroids
- Minimise stimuli: Quiet environment, Limit suctioning unless necessary
- Monitor vital signs: watch for Cushing's triad (bradycardia, hypertension, irregular breathing).

Fluid Volume Deficit Related to vomiting and decreased intake

- Monitor intake and output hourly.
- Assess signs of dehydration, such as dry mucous membranes, sunken fontanelles (in infants), and poor skin turgor.
- Administer IV fluids as prescribed (e.g., isotonic fluids like normal saline).
- Monitor serum electrolytes (especially sodium and potassium).
- Control vomiting using antiemetics (e.g., ondansetron) if prescribed.

Risk for Aspiration related to persistent vomiting and decreased level of consciousness

- Position the patient on the side (lateral position) to prevent aspiration during vomiting.
- Suction airway as needed using sterile technique.
- Keep emergency airway equipment at bedside (suction, oxygen, Ambu bag).
- Keep NPO status if the patient is vomiting excessively or until cleared for oral intake.
- Monitor respiratory status (rate, effort, O2 saturation).

Risk for long-term side effects related to chemotherapy, radiation, and surgery.

- Educate the family about potential long-term side effects, including neurocognitive deficits, growth delays, and possible endocrine changes (e.g., growth hormone deficiencies).
- Advise regular follow-up to monitor the late effects of treatment, such as growth and cognitive assessments.
- Collaborate with specialists such as endocrinologists, neurologists, and neuropsychologists to ensure early detection of any long-term side effects.
- Monitor developmental milestones and growth parameters during home visits, with regular documentation of changes.

- Provide emotional support to both the child and caregivers as they adjust to the child's post-treatment phase, addressing concerns about future health.

Risk for recurrence of cancer is related to the potential for residual microscopic disease or late recurrence.

- Emphasise the importance of follow-up care, including regular imaging (MRI/CT scans) and clinical evaluations to detect any signs of recurrence early.
- Educate caregivers on the early signs of recurrence, such as changes in neurological status, headaches, vomiting, or vision disturbances, and encourage them to report these promptly.
- Promote healthy lifestyle choices (diet, exercise, and emotional well-being) to support the child's long-term health and reduce recurrence risk.
- Establish a dedicated support network in the community to assist the family with coping mechanisms and psychological support during follow-up care.

Risk for developmental and cognitive delays related to the impact of treatment on neurodevelopmental functions.

- Conduct cognitive and developmental assessments during follow-up visits to monitor for any delays in speech, language, motor skills, and academic performance.
- Engage the family in home-based activities that promote cognitive stimulation, such as puzzles, reading, and age-appropriate learning games.
- Provide resources for educational support, such as referrals to special education services or tutors if needed, to address any academic delays.
- Coordinate care with therapists (speech, occupational, physical) to support any identified developmental needs and facilitate progress.
- Offer guidance to caregivers on fostering emotional and social development, including play-based activities with peers and encouragement of social interactions.

Risk for social isolation related to prolonged treatment period, cognitive effects, and potential physical limitations.

- Encourage the child's participation in social activities within the community, including school events, peer playgroups, or family outings, to foster emotional and social development.
- Monitor for signs of emotional distress, such as withdrawal, sadness, or difficulty making friends, and address these concerns promptly with appropriate interventions.
- Provide family support services, such as connecting with local support groups for cancer survivors and their families, where both the child and caregivers can share experiences.
- Ensure that the child's social and academic needs are being met and that any adjustments or accommodations are made as needed.

Risk for impaired family coping related to ongoing stressors from the child's illness, recovery, and follow-up care.

- Offer emotional and psychological support to the family, acknowledging the emotional toll of the child's illness and the ongoing anxiety about future health and recurrence.
- Provide information on community resources, including mental health services, counselling, and respite care for caregivers to reduce stress and prevent burnout.
- Encourage family participation in support groups for families of paediatric cancer survivors to share experiences and receive peer support.
- Monitor for signs of caregiver fatigue or anxiety, offering coping strategies such as relaxation techniques, time management tips, and promoting self-care routines.

Knowledge Deficit regarding the child's ongoing care and follow-up needs.

- Provide clear and concise information to caregivers about the follow-up schedule, necessary tests, and the importance of early detection of any complications.
- Educate the family about managing the late effects of treatment, such as growth issues, cognitive changes, and potential endocrine dysfunctions.
- Reinforce the importance of adherence to follow-up appointments, medications, and lifestyle recommendations that support the child's long-term health.
- Provide written instructions and resources for caregivers regarding symptom monitoring and when to seek medical attention for any potential complications.
- Offer continuous support by addressing any concerns the family has regarding the child's recovery, as well as answering any questions about future health needs.

Risk for Impaired Skin Integrity related to the side effects of chemotherapy and surgical scars.

- Monitor the child's skin condition regularly, particularly areas affected by radiation or surgical incisions, for signs of irritation, dryness, or breakdown.
- Educate caregivers on proper skincare, including moisturising affected areas and using gentle, non-irritating skin products to prevent further damage.
- Provide recommendations for wound care if the child has surgical scars or radiation burns, ensuring that the wounds remain clean and are protected from infection.
- Encourage regular assessments of skin integrity during follow-up visits, documenting any changes in skin condition.
- Advise on protective clothing to shield the skin from excessive sun exposure, particularly in areas affected by radiation therapy.

Risk for Fluid Volume Deficit related to reduced appetite and potential nausea post-treatment.

- Encourage the child to consume fluids throughout the day, offering lesser amounts of water, electrolyte drinks, and soups to maintain hydration.
- Monitor for signs of dehydration, such as dry mouth, lethargy, and dark-coloured urine, and intervene immediately if these symptoms occur.
- Promote a balanced diet with the help of a nutritionist to address potential long-term effects on appetite and digestive function post-treatment.
- Provide anti-nausea medications if prescribed, ensuring that caregivers are informed on how to administer them and when to seek further help if nausea persists.
- Educate caregivers on dietary strategies that can help manage nausea and improve appetite, including offering

small, frequent meals and limiting oily or strong-smelling foods.

Risk for Emotional Distress related to the ongoing fear of recurrence and challenges adapting to post-treatment life.

- Provide ongoing psychological support to both the child and family, offering counselling or therapy services if needed.
- Create a supportive environment that fosters the child's emotional recovery, incorporating activities that promote happiness and positive reinforcement.
- Address any fears of recurrence by normalizing feelings and providing education about the likelihood of relapse and how it can be managed if it occurs.
- Encourage mindfulness and relaxation techniques to help both the child and caregivers cope with anxiety about the future.
- Maintain open communication with the child's healthcare team, ensuring that any psychological or emotional concerns are addressed promptly.

4. Conclusion

In conclusion, this case emphasizes the pivotal role of early diagnosis, timely surgical intervention, and adjuvant therapy in the management of paediatric medulloblastoma. While the aggressive nature of the disease poses significant challenges, favourable histological variants, such as the desmoplastic type, offer better prognostic outcomes when appropriately treated. Crucially, interventions such as the ventriculoperitoneal shunt for hydrocephalus and prompt antibiotic therapy for postoperative infections played a key role in stabilizing the patient. However, the importance of continued care extends beyond the hospital setting. The ongoing involvement of nurses and healthcare professionals in the community is critical for managing the child's recovery, monitoring for potential complications, and ensuring adherence to the chemotherapy regimen. Home-based nursing care facilitates a seamless transition from hospital to home, providing essential support in pain management, infection prevention, and monitoring the child's neurological status. This holistic approach, integrating both hospital and community care, not only enhances the child's recovery but also contributes to long-term quality of life. Regular follow-up visits and continuous community care remain essential to ensure optimal outcomes and to address any long-term treatment effects. This case highlights the significance of a multidisciplinary approach and the essential role of home-based nursing care in improving the prognosis and overall well-being of paediatric medulloblastoma patients.

References

[1] Gaur S, Kumar SS, Balasubramaniam P. An analysis of medulloblastoma: 10-year experience of a referral institution in South India. Indian J Cancer 2015;52(4):575–8.
[2] Juraschka K, Taylor MD. Medulloblastoma in the age of molecular subgroups: A review: JNSPG 75th Anniversary invited review article. J Neurosurg Pediatr 2019;24(4):353–63.
[3] Mahapatra S, Amsbaugh MJ. Medulloblastoma Pathophysiology. 2025;1–9.
[4] Maria Yiallouros. Medulloblastoma – Brief Information. Kinderkrebsinfo 2024;
[5] Millard NE, De Braganca KC. Medulloblastoma. J Child Neurol 2016;31(12):1341–53.
[6] Slika H, Shahani A, Wahi R, Miller J, Groves M, Tyler B. Overcoming Treatment Resistance in Medulloblastoma: Underlying Mechanisms and Potential Strategies. Cancers (Basel) 2024;16(12).
[7] Panagopoulos D, Karydakis P, Giakoumettis D, Themistocleous M. The 100 most cited papers about medulloblastomas. Interdiscip Neurosurg Adv Tech Case Manag [Internet] 2021;23(June 2020):100855. Available from: https://doi.org/10.1016/j.inat.2020.100855
[8] Ghosh AK, Saha SK, Amin ANM Al, Das Gupta SP, Islam MJ. Medulloblastoma: A Common Paediatric Tumour: Experience of a Tertiary Care Cancer Center of Bangladesh. Bangladesh J Neurosurg 2021;10(2):154–62.
[9] Jackson K, Packer RJ. Recent Advances in Paediatric Medulloblastoma. Curr Neurol Neurosci Rep [Internet] 2023;23(12):841–8. Available from: https://doi.org/10.1007/s11910-023-01316-9
[10] Roth GA, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N, et al. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet 2018;392(10159):1736–88.
[11] Srinivasan V, Ghali M, North R, Boghani Z, Hansen D, Lam S. Modern management of medulloblastoma: Molecular classification, outcomes, and the role of surgery. Surg Neurol Int 2016;7(45):S1135–41.
[12] Fangusaro J. Clinical presentation, diagnosis, and risk stratification of medulloblastoma - UpToDate. UpToDate 2024;1:3–5.
[13] Orr BA. Pathology, diagnostics, and classification of medulloblastoma. Brain Pathol 2020;30(3):664–78.
[14] Roussel MF, Stripay JL. Modeling paediatric medulloblastoma. Brain Pathol 2020;30(3):703–12.
[15] Bailey S, Jacobs S, Kourti M, Pizer B, Cliofford S, Otth M. Clinical Recommendations Medulloblastoma. Siope Eur [Internet] 2022;1–85. Available from: https://siope.eu/media/documents/escp-medulloblastoma.pdf
[16] Gupta T, Sarkar C, Rajshekhar V, Chatterjee S, Shirsat N, Muzumdar D, et al. Indian Society of Neuro-Oncology consensus guidelines for the contemporary management of medulloblastoma. Neurol India 2017;65(2):315–32.
[17] Thompson EM, Ashley D, Landi D. Current medulloblastoma subgroup-specific clinical trials. Transl Pediatr 2020;9(2):157–62.
[18] Prados MD. Current Strategies for Management of Medulloblastoma. Diagnostics 2023;13(16).
[19] Lazow MA, Palmer JD, Fouladi M, Salloum R. Medulloblastoma in the Modern Era: Review of Contemporary Trials, Molecular Advances, and Updates in Management. Neurotherapeutics 2022;19(6):1733–51.
[20] Cooney T, Lindsay H, Leary S, Wechsler-Reya R. Current studies and future directions for medulloblastoma: A review from the Pacific Paediatric neuro-oncology Consortium (PNOC) disease working group. Neoplasia (United States) [Internet] 2023;35(November 2022):100861. Available from: https://doi.org/10.1016/j.neo.2022.100861