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Medulloblastoma

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Continuing Education Activity

Medulloblastoma is the most common malignant brain tumor in children, accounting for approximately 25% of pediatric central nervous system tumors. It typically arises in the cerebellum in children younger than 10 years and is classified as a WHO grade 4 embryonal tumor. Despite advances in therapy, it carries significant morbidity and mortality, with 5-year survival rates of 65% to 70%. The 2021 WHO classification defines 4 molecular subgroups: WNT-activated, SHH-activated (TP53-wildtype and TP53-mutant), and non-WNT/non-SHH (groups 3 and 4), each with distinct prognostic and clinical features. This activity reviews the clinical presentation, diagnostic evaluation (including neuroimaging and cerebrospinal fluid analysis), and risk-adapted management with surgery, radiation, and chemotherapy. Emphasis is placed on early recognition, molecular classification, interprofessional care, and strategies to reduce treatment-related complications and support long-term outcomes.

Objectives:

- Differentiate the molecular subgroups of medulloblastoma according to the WHO Classification.
- Identify the typical clinical presentation of medulloblastoma.
- Apply risk stratification criteria to determine the appropriate treatment approach for patients with medulloblastoma diagnostic features.
- Collaborate effectively within the interprofessional care team by coordinating treatment planning for patients with medulloblastoma to improve outcomes.

Access free multiple choice questions on this topic.

Introduction

Medulloblastoma represents the most common malignant brain tumor in children, accounting for approximately 25% of all pediatric central nervous system (CNS) tumors.[1][2][3] Classified as a World Health Organization (WHO) grade 4 embryonal tumor, medulloblastoma arises primarily in the posterior fossa, most often from the cerebellum. Pediatric populations account for the majority of cases, with 70% diagnosed in children younger than 10 years. Although rare in adults, medulloblastoma constitutes less than 1% of adult CNS tumors.[4] Annual incidence in children younger than 19 years old is 0.39 per 100,000, peaking between ages 3 and 7.[2][3][5] Despite advances in multimodal therapy, including surgical resection, craniospinal irradiation, and chemotherapy, medulloblastoma remains a leading cause of cancer-related morbidity and mortality in children. Overall, 5-year survival ranges from 65% to 70%, with survivors frequently experiencing long-term neurocognitive and neuroendocrine complications.

The 2021 World Health Organization (WHO) Classification categorizes medulloblastoma by molecular subgroup: WNT-activated, SHH-activated/*TP53*-wildtype, SHH-activated/*TP53*-mutant, and non-WNT/non-SHH

(encompassing groups 3 and 4), incorporating integrated molecular-histologic diagnostics. Each subgroup carries distinct histologic, genetic, clinical, and prognostic features.[6]

Etiology

The etiology of medulloblastoma remains incompletely understood. Approximately 5% of cases arise in the setting of an underlying germline mutation in a cancer predisposition gene, most commonly within the WNT-activated and SHH-activated molecular subtypes.[7][8] Syndromic associations include Turcot syndrome (*APC* mutation), Rubinstein-Taybi syndrome (C*REBBP* mutation), Gorlin syndrome (*PTCH1* or *SUFU* mutations), Li-Fraumeni syndrome (*TP53* mutation*),* and Fanconi anemia *(BRCA2* mutations*)*.[9][10][11][12][13] Germline *APC* variants are exclusively associated with the WNT-activated subtype, whereas germline *TP53* variants are confined to the SHH–activated subtype.[7][12]

The majority of medulloblastomas are believed to arise sporadically due to somatic mutations that affect subgroup-specific signaling pathways. WNT-activated tumors harbor activating *CTNNB1* mutations in approximately 90% of cases. SHH-activated tumors frequently demonstrate alterations in *PTCH1*, *SMO*, or amplifications of *GLI1* or *GLI2*. Group 3 medulloblastomas are characterized by *MYC* amplification and marked genomic instability, whereas group 4 tumors commonly exhibit *MYCN* and *CDK6* amplifications along with isochromosome 17q.[14][15]

Epidemiology

The overall incidence of medulloblastoma is approximately 0.39 per 100,000 children younger than 19 years, based on data from 2017 to 2021.[2] Children are 10 times more likely to be diagnosed with medulloblastoma than adults, with a peak incidence between the ages of 3 and 7.[3][5] Overall, medulloblastoma demonstrates a male predominance and is more frequently diagnosed in the White population.[16][17] Epidemiologic features vary substantially across molecular subgroups, including differences in age distribution, sex predilection, and relative incidence.

The WNT-activated subtype accounts for approximately 10% of medulloblastomas and occurs primarily in children older than 4 years and adolescents. This subgroup affects males and females at similar rates.[18][19] The SHH-activated subtype comprises approximately one-third of medulloblastomas and exhibits a bimodal age distribution, with peak incidence in infancy and adulthood. The sex distribution in this subgroup is approximately equal.[19] Group 3 medulloblastomas represent roughly 25% of cases and occur almost exclusively in infants and young children. This subgroup demonstrates a male predominance, with an approximate male-to-female ratio of 2:1.[19] Group 4 medulloblastomas account for approximately 35% of cases and typically present in childhood and adolescence. This subgroup shows the strongest male predominance, with an estimated male-to-female ratio of 3:1.[19]

Pathophysiology

Medulloblastoma is an embryonal malignant tumor of the cerebellum arising from dysregulated proliferation and impaired differentiation of neural precursor cells within the developing hindbrain. Its pathophysiology is defined by aberrant activation of distinct molecular signaling pathways, most commonly the WNT and SHH pathways, as well as *MYC*- or *MYCN*-driven oncogenesis in groups 3 and 4. These molecular alterations lead to uncontrolled cell cycle progression, genomic instability, and resistance to apoptosis. Medulloblastoma is a highly malignant posterior fossa tumor with a propensity for local invasion, as well as metastatic spread within the brain and spinal cord.[20]

Histopathology

Using the WHO 2021 guidelines, a tumor is classified as "medulloblastoma, histologically defined" if no molecular testing has been performed.[6] Histologic subtypes include classic, desmoplastic/nodular, medulloblastoma with extensive nodularity (MBEN), and large cell/anaplastic (LC/A).

Classic medulloblastoma histology is characterized by a small, round, blue-cell tumor with poorly differentiated cells, a high nuclear-to-cytoplasmic ratio, and high mitotic activity (See **Image**. Medulloblastoma Histology). Homer-Wright rosettes are seen in less than half of the samples.[21] Classic histology is the most common pattern and is

represented among all subgroups.

Desmoplastic/nodular histology is characterized by highly proliferative, reticulin-rich areas surrounding reticulin-free nodules with reduced mitotic activity.[6][22] This histologic pattern is exclusive to the SHH subgroup.[19] MBEN histology is similar to desmoplastic/nodular but differs in having larger reticulin-free zones.[6][22] MBEN histology is a rare variant seen almost exclusively in infants with SHH-activated tumors and has a favorable prognosis.

LC/A histology demonstrates a combination of large cell and anaplastic features. These features are large tumor cells with abundant cytoplasm, pleomorphic nuclei, and prominent nucleoli. Tumors with LC/A histology are typically located in the cerebellar vermis and are highly aggressive, exhibiting marked mitotic and apoptotic activity with extensive necrosis. This pattern is most commonly seen in group 3 tumors and SHH-activated *TP53*-mutant tumors and is associated with the poorest prognosis.[23]

History and Physical

The most common presentation of medulloblastoma is signs and symptoms of increased intracranial pressure, including headache, nausea, and vomiting. As the majority of tumors arise in the fourth ventricle, many patients present with obstructive hydrocephalus. Others may present with a combination of cerebellar signs, eg, clumsiness or gait disturbance. Infants may present with fussiness, an increasing head circumference, delayed or missed milestones, or lethargy.

Physical examination should include a thorough neurological assessment with particular attention to cerebellar function (gait, coordination, Romberg test, finger-to-nose testing), a cranial nerve examination (especially extraocular movements for sixth nerve palsy due to increased intracranial pressure), and a fundoscopic examination for papilledema.

Evaluation

The evaluation of suspected medulloblastoma requires a comprehensive approach that integrates neuroimaging, cerebrospinal fluid (CSF) analysis, histopathology, and molecular profiling to establish the diagnosis, determine the extent of disease, and guide risk stratification. Because many patients present to the emergency department, computed tomography (CT) is often the first imaging modality obtained. Medulloblastomas are typically hyperdense on CT due to their extensive cellularity.[24]

Magnetic resonance imaging (MRI), with or without contrast, is the recommended imaging modality for suspected medulloblastoma.[25] Initial imaging should include MRI of both the brain and craniospinal axis to evaluate for metastatic disease (see **Image**. Primary Medulloblastoma). This evaluation should be performed preoperatively to avoid confusion with blood products or other postoperative changes. Medulloblastoma typically presents as a large, heterogeneous posterior fossa mass occupying the fourth ventricle or cerebellar hemisphere. These masses typically show restricted diffusion on diffusion-weighted imaging (DWI).

Advanced imaging with MR spectroscopy (MRS) can help differentiate medulloblastomas from other posterior fossa tumors, as high levels of taurine at 3.4 ppm are almost exclusively found in medulloblastomas.[24] Depending on the molecular subtype, imaging findings may vary. SHH-activated tumors are more likely to be found in the cerebellar hemispheres compared to the more frequent midline location of WNT, group 3, and group 4 tumors. Furthermore, SHH-activated tumors are more likely to have peritumoral edema compared to other subtypes. WNT-activated tumors often arise in the brainstem, which explains their typical midline location.[24] After neurosurgical intervention, a postoperative brain MRI should be obtained within 24 to 72 hours. Lumbar puncture for CSF cytology is a mandatory component of staging and should be performed at least 10 to 14 days postoperatively to avoid false-positive results after surgical resection.[25]

Treatment / Management

Current treatment modalities for medulloblastoma combine surgical resection with craniospinal irradiation (CSI) and chemotherapy. All patients should be treated by an interprofessional team with experience managing pediatric CNS tumors, with early referral to radiation oncology and pediatric neuro-oncology.

Intervention begins with maximal safe resection, aiming for gross total resection (GTR) or near-total resection (NTR), followed by risk-stratified adjuvant therapy for patients aged 3 years and older. See the staging section below for more details on risk stratification. Average-risk patients (localized disease, GTR/NTR, classic or desmoplastic histology, non-*MYC*-amplified) receive CSI at 23.4 Gy with an involved-field boost to 54 Gy, followed by adjuvant chemotherapy.[NCCN. Pediatric Central Nervous System Cancers. 2026][26][27]

High-risk patients (metastatic disease, subtotal resection, large-cell/anaplastic histology, *MYCN* amplification, or *TP53* mutation) receive high-dose CSI to 36 Gy, with a boost to 54 to 55.8 Gy, plus adjuvant chemotherapy. [NCCN. Pediatric Central Nervous System Cancers. 2026][28] Very high-risk Group 3 tumors with *MYC* amplification may receive concurrent carboplatin with radiation, as it has been shown to improve event-free survival in this group.[28] Chemotherapy regimens for both average and high-risk groups include cisplatin, cyclophosphamide, lomustine, and vincristine administered during and after radiation therapy.

CSI is not recommended for children younger than 3 years due to severe neurodevelopmental toxicity; radiation-sparing chemotherapy strategies are preferred.[29] No standard chemotherapy approach has been established for these patients. Those younger than 3 years of age often fall into the SHH and group 3 subgroups. Various regimens include Head Start protocols, which use intensive, radiation-sparing chemotherapy with high-dose consolidation and autologous stem cell rescue.[30][31][Dhall G, et al. MDB-83. Outcomes of Infants and Young Children With Newly Diagnosed SHH Medulloblastoma Treated on the Next Consortium "Head Start" 4 Protocol. 2024] St. Jude infant protocols also emphasize radiation avoidance with multi-agent chemotherapy, incorporating molecular risk stratification and selective radiation for high-risk or progressive disease.[32] The Children's Oncology Group's ACNS0334 trial evaluated intensive chemotherapy with high-dose consolidation, with or without isotretinoin, in young children and demonstrated benefit in select nonmetastatic patients, particularly those with SHH-activated disease.[33] Outcomes remain poor for infant group 3 tumors, highlighting the need for novel therapeutic approaches in this population.[34]

For recurrent or progressive medulloblastoma, treatment options include repeated operation followed by adjuvant temozolomide/irinotecan with bevacizumab, temozolomide/topotecan (TOTEM), metronomic antiangiogenic therapy (MEMMAT regimen), or carboplatin/etoposide.[25]

Differential Diagnosis

The differential diagnosis for a posterior fossa mass in a child includes pilocytic astrocytoma, ependymoma, atypical teratoid/rhabdoid tumor (AT/RT), and diffuse midline glioma. Pilocytic astrocytoma is typically a cystic cerebellar hemispheric mass with a nodular component, in contrast to medulloblastoma's generally solid, heterogeneous appearance. Ependymomas arise from the floor of the fourth ventricle and classically extend through the foramina of Luschka and Magendie. AT/RT occurs predominantly in children younger than 3 years and is associated with greater invasion of the brainstem and middle cerebellar peduncle. Diffuse midline glioma typically involves the brainstem or thalamus and exhibits infiltrative growth patterns.[35] Ultimately, tumor sampling is required to distinguish these entities.

Surgical Oncology

Maximal safe resection is the first-line intervention for medulloblastoma to relieve presenting symptoms, establish histopathologic and molecular diagnosis, and maximize local control.[36][NCCN. Pediatric Central Nervous System Cancers. 2026] The goal of initial surgical intervention is gross total resection, or at least near total resection with less than 1.5 cm^2 of tumor remaining.[36][37] For those with residual tumors greater than 1.5 cm^2 on postoperative MRI, a second-look surgery is recommended, as the extent of resection is important in overall staging and prognosis. In some cases, surgical management of obstructive hydrocephalus may be necessary via an endoscopic third ventriculostomy or ventriculoperitoneal shunt.[38][39][40]

Posterior fossa syndrome, also known as cerebellar mutism syndrome, is a significant complication occurring in up to 30% of children following medulloblastoma resection.[41] The syndrome typically develops 1 to 2 days postoperatively and consists of diminished speech progressing to complete mutism, severe ataxia, emotional lability, and hypotonia. Recovery is often spontaneous, but many patients have long-term speech and neurocognitive

difficulties.[42][43] The reasons some patients develop posterior fossa syndrome remain unclear, but vermis incision, superior cerebellar peduncle involvement, and gross total resection are notable intraoperative risk factors.[44]

Radiation Oncology

CSI is the backbone of medulloblastoma treatment for children older than 3 years, targeting both the primary tumor site and potential microscopic leptomeningeal disease. CSI is generally not recommended for children younger than 3 years due to severe neurodevelopmental toxicity; radiation-sparing chemotherapy strategies are preferred in this population.

Radiation dosing is risk-stratified. Average-risk patients receive CSI at 23.4 Gy with a tumor bed boost to 54 Gy, while high-risk patients receive CSI at 36 Gy with additional boosts to metastatic sites.[25] The Children's Oncology Group ACNS0331 trial demonstrated that reducing the boost volume from whole posterior fossa to involved-field radiation therapy is safe, whereas reducing the CSI dose from 23.4 Gy to 18 Gy resulted in inferior outcomes.[26] Proton therapy is recommended when available to reduce toxicity to the heart, lungs, cochlea, and developing brain.[45]

Late effects of radiation therapy are substantial and include neurocognitive decline, endocrinopathies (growth hormone deficiency in nearly all patients receiving 36 Gy CSI), hearing loss, and secondary malignancies.[46]

Treatment Planning

Craniospinal irradiation is commonly delivered using proton beam therapy or intensity-modulated radiotherapy to minimize exposure of normal tissues to radiation and spare organs at risk. Treatment is commonly delivered in fractions of 180 cGy. Target volumes consist of the craniospinal axis, with subsequent cone-down boosts to areas of resected or gross disease.

Staging

Staging of medulloblastoma depends on neuroimaging, CSF analysis, the extent of surgical resection, histopathology, and molecular profiling. The findings from brain and spine MRI, CSF analysis, and histopathology guide staging and risk stratification. Patients were traditionally categorized as standard- or high-risk, with high-risk defined by age younger than 3, residual tumor greater than 1.5 cm^2, or metastatic disease at presentation.[47][48]

Current NCCN guidelines stratify patients older than age 3 by molecular subgroup, though metastatic disease, subtotal resection, or *MYC* amplification still indicate high-risk status.[25] Low-risk tumors are defined as gross or near-totally resected WNT-activated medulloblastomas with classic histology and without metastasis or *MYC* amplification. Standard-risk tumors include select patients from the SHH-activated and non-WNT/non-SHH subgroups. In the SHH-activated subgroup, standard-risk disease is defined by *TP53*–wild-type status, absence of metastatic dissemination, gross or near-total resection, and absence of *MYCN* amplification or large-cell/anaplastic histology.

Similarly, patients with non-WNT/non-SHH medulloblastoma are considered standard risk when the disease is nonmetastatic, any residual tumor is minimal following surgery, and neither *MYC* amplification (group 3) nor *MYCN* amplification (group 4) is observed. No large-cell/anaplastic histology is observed. High-risk features include *TP53* mutation in SHH-activated medulloblastoma, *MYC* amplification in group 3 tumors, and *MYCN* amplification in group 4 tumors. Group 3 medulloblastomas with *MYC* amplification are among the highest-risk phenotypes and are associated with particularly poor outcomes.[25][49]

Children younger than 3 are classified as infantile medulloblastoma and do not use the above staging—they are a biologically diverse group prone to recurrence and vulnerable to neurotoxicity from craniospinal irradiation.[29]

Prognosis

The overall 5-year survival rate for pediatric medulloblastoma is around 70%, though outcomes vary dramatically by molecular subgroup and risk stratification. Key prognostic factors include age at presentation (<3 years associated with worse outcomes), presence of metastatic disease, extent of resection, histologic subtype, molecular subgroup,

and specific molecular alterations, including *MYC/MYCN* amplification and *TP53* mutation status.

Molecular subgroup is the most important prognostic determinant. WNT-activated tumors have the best prognosis, with overall survival approaching 100% in children; these patients are candidates for treatment de-escalation trials.[50][51] SHH-activated, *TP53*-wild-type tumors have an intermediate prognosis, with more than 80% survival in the absence of high-risk features, whereas SHH-activated, *TP53*-mutant tumors have poor outcomes, with approximately 40% 5-year overall survival.[14]

In breaking down the non-WNT/non-SHH group, group 3 tumors carry the worst prognosis with 5-year survival of 20% to 50%, particularly when associated with *MYC* amplification or metastatic disease. Group 4 tumors have an intermediate prognosis with a 5-year overall survival of 75% to 90%.[14][6][22][25]

Complications

Complications of medulloblastoma arise from both the disease itself and its multimodal treatment. Acute treatment-related complications include posterior fossa syndrome (cerebellar mutism) occurring in up to 30% of patients following surgical resection. Radiation-related acute toxicities include headache, nausea/vomiting, and skin erythema. Acute toxicities from chemotherapy include cytopenias, nausea, vomiting, constipation, ototoxicity, nephrotoxicity, and increased risk of infection.

Long-term complications are substantial and affect nearly all survivors. Neurocognitive impairment is one of the most pervasive late effects; younger age at treatment is associated with more severe deficits. Endocrinopathies are common, with growth hormone deficiency occurring in nearly all patients receiving 36 Gy craniospinal irradiation and 50% of those receiving 23.4 Gy; hypothyroidism affects up to 75% of survivors.[52][53]

Secondary malignancies represent a significant long-term risk when compared to the general population. The 10-year cumulative incidence of secondary neoplasms is approximately 5% to 7%, with the central nervous system being the most common site (high-grade gliomas and meningiomas), followed by thyroid carcinoma and bone/soft tissue tumors.[54] Risk is higher in patients treated at younger ages and those receiving craniospinal irradiation.
These significant late effects have driven efforts toward molecularly informed treatment de-escalation for favorable-risk subgroups.

Deterrence and Patient Education

Patients and families should be counseled that medulloblastoma requires intensive multimodal treatment, including surgery, radiation therapy, and chemotherapy, with treatment duration typically spanning 12 to 18 months. Education regarding the importance of adherence to surveillance schedules is essential, as recurrence surveillance includes serial brain and spine MRI, CSF analysis, and clinical examinations at regular intervals for at least 5 years. Families should be informed about the substantial risk of long-term complications, including neurocognitive decline, endocrinopathies, hearing loss, and secondary malignancies, which necessitate lifelong interprofessional follow-up. Referral for fertility preservation counseling should be offered before initiating treatment.

Enhancing Healthcare Team Outcomes

Medulloblastoma represents the most common malignant brain tumor in children, arising predominantly in the cerebellum and accounting for roughly 25% of pediatric central nervous system tumors. Most cases occur in children younger than 10 years old, with peak incidence between ages 3 and 7. Diagnosis relies on neuroimaging and histopathologic evaluation, while molecular subgrouping, WNT-activated, SHH-activated/*TP53*-wildtype, SHH-activated/*TP53*-mutant, and non-WNT/non-SHH, guides prognosis and treatment planning. Standard therapy combines maximal safe surgical resection with craniospinal irradiation and chemotherapy. Despite advances, medulloblastoma continues to cause significant morbidity, and survivors frequently experience long-term neurocognitive, neuroendocrine, and psychosocial challenges.

Optimal management requires an interprofessional approach integrating pediatric oncologists, neurosurgeons, radiation oncologists, pathologists, radiologists, and nurses, with pharmacists, dietitians, genetic counselors, fertility specialists, and psychosocial professionals involved as needed. Physicians and advanced practitioners coordinate

diagnosis, treatment planning, and ongoing monitoring, while nurses assess and manage acute toxicities during therapy. Pharmacists assist with safe, individualized chemotherapy dosing. Interprofessional communication through tumor boards, shared records, and family liaisons enhances care coordination. Long-term survivorship planning ensures monitoring for late effects and supports a smooth transition to adult care, improving patient-centered outcomes and safety.

Review Questions

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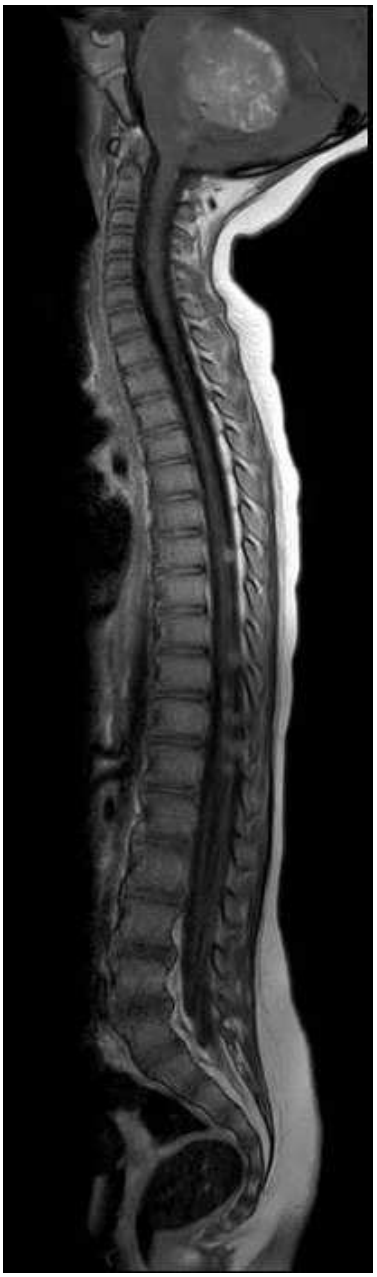
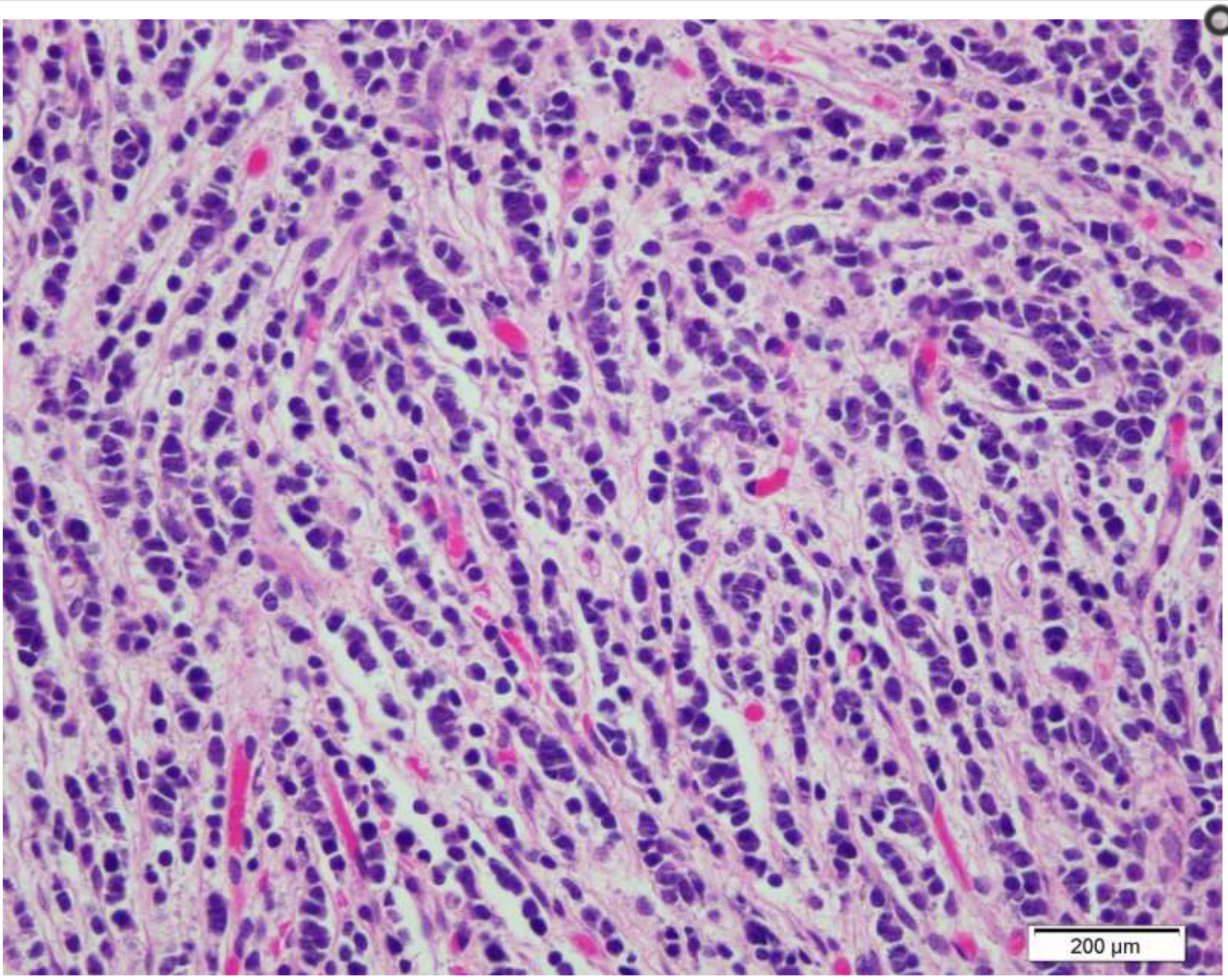
Figures



Primary Medulloblastoma. MRI spine with contrast showing primary medulloblastoma in the posterior fossa and multiple drop metastases along the spinal cord in the sagittal plane. Contributed by A Guild, DO



Medulloblastoma Histology. Classic medulloblastoma histology is characterized by a small, round, blue cell tumor with poorly differentiated cells, a high nuclear-to-cytoplasmic ratio, and high mitotic activity. Contributed by S Mahapatra, MD, PhD

Tables

Pause and Reflect	A 6-year-old child undergoes maximal safe resection of a posterior fossa medulloblastoma. Postoperative MRI demonstrates minimal residual disease, and cerebrospinal fluid cytology is negative for metastatic spread. Molecular testing identifies a WNT-activated tumor without MY amplification. How should the clinician and interprofessional oncology team use these findings to guide risk stratification, treatment planning, and family counseling? What additional specialists should be involved early in care to reduce long-term treatment-related complications and optimize survivo outcomes?

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