Genuine Brief Overview: Brain Tumours and their Related Evaluation and Curative Possibilities

ABSTRACT

A brain tumour may start somewhere else and go to the brain or originate in the brain cells (as demonstrated). Signs and symptoms including headaches, nausea, and balance issues are brought on by the tumour's growth, which puts pressure on adjacent brain tissue and alters its function.They may originate in the brain itself or adjacent tissue. Meninges, the brain's membranes, may be among the nearby tissue. Additionally, brain tumours can develop in the pituitary, pineal, and nerves. Brain tumours develop when DNA alterations occur in the brain or nearby brain cells. Tumours often develop when cells in the body divide and expand uncontrollably. The body normally regulates the division and development of cells. Older cells are replaced by new ones or given new roles by the creation of new cells. Damaged or unnecessary cells die to create room for healthy ones.In India, the frequency of central nervous system (CNS) tumours is between 5 and 10 per 100,000 people, and it is on the rise, making up 2% of all cancers.2019 data on the incidence and death of brain cancer. 2019 saw 347,992 new instances of brain cancer reported globally; 187,491 (54%) males and 160,501 (46%) females received the diagnosis.We review the aetiology, prevalence, possible therapies, and present state of brain tumours in this study.

Keywords:Brain Tumours, Epidemiology, Etiology, Pathophysiology, Diagnosis, Therapies

INTRODUCTION

Meningiomas and lymphomas, for example, do not originate from brain tissue; instead, they are better known as "intracranial neoplasms." The phrase "brain tumour" refers to a group of neoplasms, each with its biology, prognosis, and treatment.A cell growth inside or close to the brain is called a brain tumour. Brain tissue can develop brain tumours. Brain tumours may also occur near brain tissue. Neural pathways, the pituitary, pineal, and brain surface membranes are all near one another.Both localized and widespread neurological symptoms can be brought on by brain tumours. Severe symptoms include headache, nausea, vomiting, and sixth-nerve palsy, which are indicative of elevated intracranial pressure. Hemiparesis and aphasia are examples of focal symptoms and indicators that indicate the tumour'sintracranial placement(1,56,57,58,59).Glioneuronal tumours, also known as neuronal brain tumours, are a

broad category of primary central nervous system cancers that include lesions with a predominantly neuronal or mixed (glial and neuronal) origin. Although other symptoms including headaches, focal neurological impairments, and acute hydrocephalus may also occur, epilepsy is the most prevalent symptom of these uncommon tumours. The assessment and management of neuronal brain tumours are reviewed in this exercise, which also emphasizes the role of the interprofessional team in diagnosing and treating individuals who are found to have these tumours.A rare class of tumours affecting the central nervous system that develops from cells with neuronal differentiation are known as neural brain tumours. These tumours, which make up a subset of glioneuronal tumours, might contain mixed neuronal and glial components or be solely neuronal in origin. There are fourteen different cancers classified as central nervous system (CNS) tumours by the World Health Organization (WHO) in 2021.Gangliocytoma, dysplastic cerebellar gangliocytoma (Lhermitte-Duclos disease), central neurocytoma, extraventricular neurocytoma, cerebellar lipo neurocytoma, and multinodular and vacuolating neuronal tumour (MVNT) are all included in the category of solely neuronal tumours. Ganglioglioma, myxoid glioneuronal tumour (MGT), diffuse leptomeningeal glioneuronal tumour (DLGNT), rosette-forming glioneuronal tumour (RGNT), papillary glioneuronal tumour (PGT), desmoplastic infantile ganglioglioma/desmoplastic infantile astrocytoma (DIG/DIA), and dysembryoplastic neuroepithelial tumour (DNET) are among the glioneuronal tumours that fall under this category. In the WHO CNS tumour categorization system, MVNT, DGONC, and MGT are newly categorized entities.With few exceptions, the majority of these tumours are low-grade and have little chance of developing into an aggressive clinical course. Although other symptoms of space-occupying cerebral lesions, such as headache, vomiting, papilledema, cerebellar dysfunction, and localized neurological impairment, have also been reported, seizures are the most prevalent presentation(2–4).Leukaemia is the leading cause of juvenile cancers, with pediatric brain tumours being the most common form of solid childhood cancer. Lesions are divided into two categories: supratentorial and infratentorial. Additionally, they might be divided into three categories based on the age at which they manifested: tumours of infancy (less than a year old), tumours of older children, and congenital brain tumours (CBT), which are detected during pregnancy and within the first sixty days of life. The age of presentation, histological type, and degree of resectability are the primary factors influencing the prognosis of pediatric brain tumours. The pathophysiology, presentation, and genesis of childhood brain tumours are reviewed in this paper, which also emphasizes the importance of the interprofessional team in managing these cases(5–8).

EPIDEMIOLOGY AND HISTOPATHOLOGY

About 0.5 to 2% of all primary CNS tumours are in this uncommon category of brain tumours. The majority of the lesions in the group are ganglioglioma and DNET. Still, case reports and small case series provide descriptions of several of the newly categorised lesions, including MVNT, DGONC, MGT, and others. These lesions presumably constitute a tiny fraction of all primary CNS cancers. These are mostly tumours that affect children and young adults. A diagnosis made at an age older than thirty years old is an uncommon clinical situation.The

prevalence of brain tumours in children varies by nation. The rates of 1.15 to 5.14 instances per 100,000 children have been reported, with the United States having the highest rates. In different parts of the world, the incidence of CBT varies from between 0.3 to 2.9 instances per 100,000 live births. The location and histological subtype are two of the many variables that affect their prognosis and survival rates.Within the congenital group, teratomas account for 26.6% to 48% of cases. Next in order of prevalence are ependymoma (4.4%), craniopharyngioma (5.6% to 6.8%), embryonal tumour (3% to 13%), choroid plexus papilloma (3.7% to 13.2%), and astrocytoma (7.4% to 28.8%)(5,9–12).Both neuronal and glioneuronal cancers have diverse histological characteristics. When neuronal cell markers like synaptophysin or neuron-specific enolase (NSE) are stained with neurohistochemically, all neuronal and glioneuronal malignancies should show positive results. However, only the glioneuronal subgroup of tumours—which is typically associated with glial fibrillary acidic protein (GFAP) positivity—should show positivity for glial differentiation markers in addition to neuronal marker positivity. The Ki-67 indicator of cell proliferation is generally low, while higher-grade malignancies such as anaplastic astrocytoma may exhibit higher rates of proliferation(13,14).

ETIOLOGY

This varied group of tumours does not have a particular aetiology or risk factor associated with their development. On the other hand, the present focus on the molecular characterisation of brain tumours offers fresh information about the genetic abnormalities that cause them to grow. Particularly, two primary molecular subgroups involving mutations in several cellular regulatory pathways may exist. A mutation in the mitogen-activated protein kinase (MAPK) pathway, which is involved in cell proliferation, is seen in the first subgroup. Particularly, these tumours frequently exhibit modifications to the proto-oncogene BRAF in the form of BRAF fusion and single nucleotide missense mutations. Gangliocytoma and ganglioglioma have the highest incidence of these mutations, with 25% showing fusion mutations and 13-56% showing single nucleotide missense mutations.Additionally, they have been reported in isolated MVNT cases and DLGNT cases.These mutations result in uncontrolled cell growth because they cause a gain of function. Fibroblast growth factor (FGFR) genes, primarily FGFR-1, which is upstream of the MAPK pathway, are mutated in the second group. Although it has also been seen in neurocytoma and RGNT, this seems to be particularly prevalent in DNETs. It's interesting to note that these molecular subgroups may aid in differentiating between different types of tumours, especially for the glioneuronal group. The glial component of the BRAF mutant group often resembles astrocytes, whereas the glial component of the FGFR group more closely resembles oligodendroglia(15–22).Within the categories of neuronal and glioneuronal tumours, MGTs are recently identified lesions. They are comparable to DNET, except they prefer to be found in the septum pellucidum and are linked to mutations in the platelet-derived growth factor receptor (PDGFR) gene rather than the FGFR gene. The terms "intraventricular DNET" and "DNET-like neoplasm of the septum pellucidum" were frequently used to describe this tumour in the past.PGNTs have distinct genetic abnormalities concerning the neuronal and glioneuronal tumour categories. These frequently have a fusion aberration between PRKCA (protein kinase C alpha) and SLC44A1 (choline transporter 1), resulting in SLC44A1-PRKCA. Interestingly, PRKCA participates in the MAPK signaling cascade. Nevertheless, the BRAF mutations

observed in other neuronal and glioneuronal malignancies with alterations in the MAPK pathway are not identified in these tumours.Within this category of tumours, dysplastic cerebellar gangliocytoma, also known as Lhermitte-Duclos disease, is unique since it is linked to Cowden syndrome, also known as phosphatase and tensin homolog (PTEN) hamartoma tumour syndrome. PTEN is involved in the regulation of the cell cycle through the mammalian target of the rapamycin (mTOR) pathway. Proliferation is decreased by the pathway's downregulation by wild-type PTEN. Tumorigenesis can arise from dysregulated mTOR signalling caused by mutations in PTEN(23–27).

PATHOPHYSIOLOGY

The pathogenesis of most of these diseases is greatly influenced by the tumour site, as these lesions are usually WHO 1. WHO grade 2 lesions include cerebellar lipo neurocytoma, extraventricular neurocytoma, and central neurocytoma. The only WHO grade 3 lesion in the group is the anaplastic type of ganglioglioma. Due to their rarity, several recently described tumours, like DLGNT and MGT, do not yet have a classification. As mentioned, although some cancers are more likely to occur in a particular area, these tumours can occur anywhere in the supratentorial or infratentorial space. For instance, in the context of Cowden syndrome, dysplastic cerebellar gangliocytoma manifests as Lhermitte-Duclos illness in the cerebellum.Nonetheless, it appears that the temporal lobe is the most frequently found tumour location. This may be due in part to the fact that DNET and ganglioglioma are the most prevalent tumour types in this group and have a preference for this area. Additionally, these tumours might be discovered in extra-temporal areas. The most frequent presenting symptom of this class of tumours is seizures, which can arise from location in the temporal lobe and other epileptogenic regions. Because of this, they are frequently referred to as long-term epilepsy-associated tumours (LEATs), and maximal medical therapy is frequently ineffective in treating their seizures.The placement also affects other symptoms. There have also been reports of cerebellar dysfunction, hydrocephalus, papilledema, headache, nausea, vomiting, and localized neurological deficiency. With space-occupying lesions, higher intracranial pressure is expected, as evidenced by symptoms such as headache, vomiting, and papilledema. Given that many of these lesions grow slowly, they can enlarge before they are clinically recognized, with these symptoms and indicators serving as the first clue that points to the presence of a tumour.Tumours in the intraventricular or posterior fossa might interfere with the natural pathways for the outflow of cerebrospinal fluid (CSF), which is the primary cause of hydrocephalus. Typically, this is obstructive-type hydrocephalus, which can become acute in rare instances of central neurocytoma if normal CSF outflow channels are abruptly blocked. Because of its location, dysplastic cerebellar gangliocytoma frequently causes cerebellar dysfunction; also, specific tumour locations in the cerebrum are linked to specific localized neurological abnormalities(28–30).

DIAGNOSIS

As previously mentioned, a thorough history and physical examination are necessary for the initial assessment of a patient suspected of having a neuronal or glioneuronal brain tumour. Importantly, albeit infrequent, patients presenting with abrupt changes in their neurological

status require an urgent assessment of their breathing/oxygenation, circulatory system, and airway status (ABCs). This situation can happen to individuals who arrive with subclinical or widespread tonic-clonic seizure activity, as well as people who have acute hydrocephalus. When compromise on the first assessment of the ABCs is recognized, there should be a low threshold for resuscitation and intubation. On the other hand, the majority of patients arrive with headaches and focal partial seizures, which are symptoms of elevated intracranial pressure.*MRI*: When assessing this kind of brain tumour, MRI is the preferred imaging test. T1- and T2-weighted pictures with and without gadolinium contrast should be included in the sequences. When a primary CNS tumour is suspected, a standard brain tumour imaging protocol should include fluid-attenuated inversion recovery (FLAIR) imaging for tumour characterization and oedema, susceptibility-weighted imaging (SWI) for haemorrhage and calcification, diffusion-weighted imaging (DWI) for an assessment of cellularity, and perfusion-weighted imaging (PWI) for an assessment of angiogenesis.Functional MRI (fMRI), diffusion tensor imaging (DTI), and MR spectroscopy (MRS) are a few more sophisticated MRI methods that may be helpful. For every tumour, the most prevalent MRI features are displayed. Since many of these lesions are rare, it is still too early to describe their full imaging properties.*CT*: Due to its speed and capacity to show frequent causes of acute status change, such as bleeding, patients presenting with acute deficits should first get a head CT without contrast after achieving clinical stability. The capacity to demonstrate hydrocephalus, which may necessitate urgent surgical intervention, is its most valuable characteristic. Depending on where the tumour is located, CT findings that are indicative of hydrocephalus may include triventricular or tetraventricular enlargement, enlargement of the temporal horns, and hypodensities surrounding the frontal horns that may indicate transependymal CSF flow.When it comes to intraventricular lesions such as central neurocytomas, CT results show hyperdensity inside the ventricular system, and when it comes to intraparenchymal lesions, the brain parenchyma shows hypo- or iso-density. Though this is probably not present in more than half of all neuronal and glioneuronal brain tumours, the head CT scan may also reveal calcification. About 10% of DNET and 35 to 50% of gangliogliomas exhibit calcifications, but up to 90% of lesions with other primary CNS lesions, such as oligodendrogliomas, show some degree of calcification(31–36).WADA Testing: Several LEATs are situated in areas where language function may be jeopardized by surgical excision. Before resection, WADA testing is usually used on these individuals to determine language dominance and help with surgical planning. Although fMRI has been employed for this objective as well, WADA testing is still the gold standard for identifying linguistic dominance and may be more accurate.Electroencephalography (EEG): Since this type of brain tumour frequently manifests as seizures, EEG is a crucial factor to take into account. Spot EEG might be helpful in certain situations, but continuous video EEG monitoring helps identify an epileptogenic focus and provides a better understanding of seizure semiology. When noninvasive recording approaches fail to localize seizures to the tumour site, invasive EEG monitoring with either depth electrodes or subdural strip electrodes may be considered for the patient. The EEG properties may be influenced by the unique histologic features of a given tumor. The histology of tumours that are mostly neuronal frequently exhibits continual spiking(37–39).

PROBLEMS RELATED TO TUMOURS

The complications associated with brain tumours, both neuronal and glioneuronal, vary depending on the treatment approach used. Anti-epileptic drug side effects are frequent and include irritability, lightheadedness, sleepiness, blurred vision, impaired coordination, generalized exhaustion, and weight gain. If taken during pregnancy, more serious adverse effects could also occur, including severe skin responses and congenital defects in children. Additional complications associated with surgical intervention include infection, bleeding both inside and outside the brain, postoperative hydrocephalus, new neurological deficit, inability to control seizures, damage to blood vessels or cranial nerves surrounding the tumour, cerebral edema and swelling, and death(40,41).

MEDICAL SUPERVISION FOR BRAIN TUMOURS

Medical Management of Seizures: Anti-epileptic drugs for LEATs are the mainstay of medical management for this class of tumours. The particular agents that are utilized can vary, and usually, a range of compounds are tested. Nevertheless, malignant tumours usually require surgery because they are frequently resistant to the most aggressive medical treatment.Because these tumours are primarily low-grade, lesions that are asymptomatic or minimally symptomatic but were discovered on imaging for another reason can be reasonably followed up with serial examination and neurological imaging. At interprofessional conferences, changes in the tumour's radiological features or the emergence of fresh neurological symptoms should spark conversation about possible interventions.The preferred course of treatment for symptomatic neuronal and glioneuronal tumours is surgical resection. Emergent surgery is necessary for patients with acute obstructive hydrocephalus to avoid death or irreversible brain damage. That clinical setting is unique to this group, though, and most others may usually be planned as outpatient procedures once the need for surgery has been decided upon through interprofessional conferences. The location of the tumour has a significant impact on the details of the intervention. Usually, a craniotomy is required to achieve gross complete tumour excision. On the other hand, certain findings indicate that in the case of LEATs, a tiny percentage of the remaining tumour can nevertheless lead to adequate resection.For higher-grade lesions, gross complete resection seems to be more significant. Anaplastic ganglioglioma patients appear to have a better prognosis at higher extents of resection (EOR), which is consistent with results from other high-grade CNS tumours like glioblastoma. An additional improvement in survival in central neurocytoma has been shown by the increased EOR. Traditionally, traditional craniotomy and tumour excision have been used to treat intraventricular lesions in this group; however, newer, less invasive surgical methods that employ tubular retractor devices are starting to become available.Chemotherapy and immunotherapy: Similar to radiation therapy, these treatments are often limited to grade 2 and 3 malignancies. As an adjuvant treatment for anaplastic ganglioglioma, immunotherapies that target BRAF are becoming more popular. BRAF inhibitors include dabrafenib, encorafenib, and vemurafenib. Chemotherapy for central neurocytoma is sometimes used, but only in cases where the patient has recurrent tumours.Case reports and short case studies have recorded the use of agents such as vincristine, cisplatin, etoposide, carboplatin, cyclophosphamide, and lomustine in the treatment of central neurocytoma. But lately, temozolomide—an alkylating drug that may be taken orally or intravenously and is frequently used to treat cancers of the central nervous system—has gained appeal(29,42–

49,49,50).Radiation Therapy: As most of these cancers are WHO grade I, radiation therapy's application to this class of tumours is somewhat limited. Adjuvant radiation therapy may be necessary for higher-grade lesions such as lipo neurocytoma, anaplastic ganglioglioma, and certain central/extraventricular neurocytomas. Radiation therapy is most frequently used, though not always, to treat anaplastic ganglioglioma.Postoperative radiation therapy is another option for treating lipo neurocytoma. Adjuvant radiation has proven to be a beneficial treatment for patients who underwent subtotal resection or experienced recurrent lipo neurocytoma in case reports and small case series(51–55).

DISCUSSION AND CONCLUSION

An introduction to brain tumours, including their many causes, epidemiology, and alternative therapies, is given at the beginning of our review articles. Our results show that while drugs are effective in treating certain conditions, they do not replace other forms of treatment such as chemotherapy, radiation therapy, and surgery in curing or inhibiting tumour growth. Additional randomized controlled trials are required to address brain cancers. We plan to carry out an initial study on brain malignancies in the future. Future counselling-based research in our nation or state will evaluate patients' mental and physical well-being and provide more precise information on brain tumours and their treatment, thanks to the help of our colleagues.

CONFLICT OF INTEREST

The authors attest that they are free of any known financial or personal conflicts of interest that would taint the findings of this study.

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References

1. Gleason C, Devaskar S. Brain Tumors. Brain Tumors. 2012;344(2):114–23.
2. Louis DN, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, et al. The 2021 WHO Classification of Tumors of the Central Nervous System: a summary. Neuro Oncol. 2021 Aug;23(8):1231–51.
3. Shin JH, Lee HK, Khang SK, Kim DW, Jeong AK, Ahn KJ, et al. Neuronal tumors of the central nervous system: radiologic findings and pathologic correlation. Radiogr a Rev Publ Radiol Soc North Am Inc. 2002;22(5):1177–89.
4. Giulioni M, Rubboli G, Marucci G, Martinoni M, Marliani AF, Bartiromo F, et al. Focal epilepsies associated with glioneuronal tumors: review article. Panminerva Med. 2013 Jun;55(2):225–38.
5. Abdel Razek AAK, Elsebaie NA, Zamora C, Castillo M. Imaging of Neuronal and Mixed Glioneuronal Tumors. J Comput Assist Tomogr. 2020;44(3):356–69.
6. Larouche V, Huang A, Bartels U, Bouffet E. Tumors of the central nervous system in the first year of life. Pediatr Blood Cancer. 2007 Dec;49(7 Suppl):1074–82.
7. El-Ayadi M, Ansari M, Sturm D, Gielen GH, Warmuth-Metz M, Kramm CM, et al. High-grade glioma in very young children: a rare and particular patient population. Oncotarget. 2017 Sep;8(38):64564–78.
8. Ostrom QT, de Blank PM, Kruchko C, Petersen CM, Liao P, Finlay JL, et al. Alex's Lemonade Stand Foundation Infant and Childhood Primary Brain and Central Nervous System Tumors Diagnosed in the United States in 2007-2011. Neuro Oncol. 2015 Jan;16 Suppl 10(Suppl 10):x1–36.
9. Alamo L, Beck-Popovic M, Gudinchet F, Meuli R. Congenital tumors: imaging when life just begins. Insights Imaging. 2011 Jun;2(3):297–308.
10. Carstensen H, Juhler M, Bøgeskov L, Laursen H. A report of nine newborns with congenital brain tumours. Child's Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg. 2006 Nov;22(11):1427–31.

11. Oi S, Kokunai T, Matsumoto S. Congenital brain tumors in Japan (ISPN Cooperative Study): specific clinical features in neonates. Child's Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg. 1990 Mar;6(2):86–91.

12. Johnson KJ, Cullen J, Barnholtz-Sloan JS, Ostrom QT, Langer CE, Turner MC, et al. Childhood brain tumor epidemiology: a brain tumor epidemiology consortium review. Cancer Epidemiol biomarkers Prev a Publ Am Assoc Cancer Res cosponsored by Am Soc Prev Oncol. 2014 Dec;23(12):2716–36.

13. Smith TW, Nikulasson S, De Girolami U, De Gennaro LJ. Immunohistochemistry of synapsin I and synaptophysin in human nervous system and neuroendocrine tumors. Applications in diagnostic neuro-oncology. Clin Neuropathol. 1993;12(6):335–42.

14. Isgrò MA, Bottoni P, Scatena R. Neuron-Specific Enolase as a Biomarker: Biochemical and Clinical Aspects. Adv Exp Med Biol. 2015;867:125–43.

15. Sekar A, Rudrappa S, Gopal S, Ghosal N, Rai A. Rosette-Forming Glioneuronal Tumor in Opticochiasmatic Region-Novel Entity in New Location. World Neurosurg. 2019 May;125:253–6.

16. Bale TA. FGFR- gene family alterations in low-grade neuroepithelial tumors. Acta Neuropathol Commun. 2020 Feb;8(1):21.

17. Slegers RJ, Blumcke I. Low-grade developmental and epilepsy associated brain tumors: a critical update 2020. Acta Neuropathol Commun. 2020 Mar;8(1):27.

18. Métais A, Appay R, Pagès M, Gallardo C, Silva K, Siegfried A, et al. Low-grade epilepsy-associated neuroepithelial tumours with a prominent oligodendroglioma-like component: The diagnostic challenges. Neuropathol Appl Neurobiol. 2022 Feb;48(2):e12769.

19. Stone TJ, Keeley A, Virasami A, Harkness W, Tisdall M, Izquierdo Delgado E, et al. Comprehensive molecular characterisation of epilepsy-associated glioneuronal tumours. Acta Neuropathol. 2018 Jan;135(1):115–29.

20. Choi E, Kim SI, Won JK, Chung CK, Kim SK, Choi SH, et al. Clinicopathological and molecular analysis of multinodular and vacuolating neuronal tumors of the cerebrum. Hum Pathol. 2019 Apr;86:203–12.

21. Krauze AV. Glioneuronal Tumors: Insights into a Rare Tumor Entity. In: Debinski W, editor. Brisbane (AU); 2021.

22. Srinivasa K, Cross KA, Dahiya S. BRAF Alteration in Central and Peripheral Nervous System Tumors. Front Oncol. 2020;10:574974.

23. Dragoo DD, Taher A, Wong VK, Elsaiey A, Consul N, Mahmoud HS, et al. PTEN Hamartoma Tumor Syndrome/Cowden Syndrome: Genomics, Oncogenesis, and Imaging Review for Associated Lesions and Malignancy. Cancers (Basel). 2021 Jun;13(13).

24. Pages M, Lacroix L, Tauziede-Espariat A, Castel D, Daudigeos-Dubus E, Ridola V, et al. Papillary glioneuronal tumors: histological and molecular characteristics and diagnostic value of SLC44A1-PRKCA fusion. Acta Neuropathol Commun. 2015 Dec;3:85.

25. Nagaishi M, Nobusawa S, Matsumura N, Kono F, Ishiuchi S, Abe T, et al. SLC44A1-PRKCA fusion in papillary and rosette-forming glioneuronal tumors. J Clin Neurosci Off J Neurosurg Soc Australas. 2016 Jan;23:73–5.

26. Lucas CHG, Villanueva-Meyer JE, Whipple N, Oberheim Bush NA, Cooney T, Chang S, et al. Myxoid glioneuronal tumor, PDGFRA p.K385-mutant: clinical, radiologic, and histopathologic features. Brain Pathol. 2020 May;30(3):479–94.

27. Zamora C, Castillo M. From Dysembryoplastic Neuroepithelial Tumors to Myxoid Glioneuronal Tumors, a New Entity. Vol. 42, AJNR. American journal of neuroradiology. United States; 2021. p. E77–8.

28. Cheng CH, Liu CL, Chen CC, Lin SZ, Cho DY. Single-port endoscopic removal of intraventricular central neurocytoma. J Clin Neurosci Off J Neurosurg Soc Australas. 2010 Nov;17(11):1417–20.

29. Giulioni M, Marucci G, Martinoni M, Marliani AF, Toni F, Bartiromo F, et al. Epilepsy associated tumors: Review article. World J Clin cases. 2014 Nov;2(11):623–41.

30. Poyuran R, Mahadevan A, Mhatre R, Arimappamagan A, Sinha S, Bharath RD, et al. Neuropathological spectrum of drug resistant epilepsy: 15-years-experience from a tertiary care centre. J Clin Neurosci Off J Neurosurg Soc Australas. 2021 Sep;91:226–36.

31. Verbančič T, Ravnik J, Kavalar R. Rosette-forming glioneuronal tumor of the fourth ventricle; A case report and review of the literature. Vol. 9, Clinical case reports. England; 2021. p. e04355.

32. Sáez-Alegre M, Saceda Gutiérrez JM, Utrilla Contreras C, Aracil Santos FJ, García-Feijoo P, Carceller Benito F. Diffuse leptomeningeal glioneuronal tumour: where to biopsy? Case report and literature review. Child's Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg. 2021 Jul;37(7):2405–8.

33. Zhang D, Wen L, Henning TD, Feng XY, Zhang YL, Zou LG, et al. Central neurocytoma: clinical, pathological and neuroradiological findings. Clin Radiol. 2006 Apr;61(4):348–57.

34. Ramsahye H, He H, Feng X, Li S, Xiong J. Central neurocytoma: radiological and clinico-pathological findings in 18 patients and one additional MRS case. J Neuroradiol = J Neuroradiol. 2013 May;40(2):101–11.

35. Luzzi S, Elia A, Del Maestro M, Elbabaa SK, Carnevale S, Guerrini F, et al. Dysembryoplastic Neuroepithelial Tumors: What You Need to Know. World Neurosurg. 2019 Jul;127:255–65.

36. Demir MK, Yapıcıer O, Yılmaz B, Kılıç T. Magnetic resonance imaging findings of mixed neuronal-glial tumors with pathologic correlation: a review. Acta Neurol Belg. 2018 Sep;118(3):379–86.

37. Ishikawa T, Muragaki Y, Maruyama T, Abe K, Kawamata T. Roles of the Wada Test and Functional Magnetic Resonance Imaging in Identifying the Language-dominant Hemisphere among Patients with Gliomas Located near Speech Areas. Neurol Med Chir (Tokyo). 2017 Jan;57(1):28–34.

38. Vivas AC, Reintjes S, Shimony N, Vale FL. Surgery of the amygdala and uncus: a case series of glioneuronal tumors. Acta Neurochir (Wien). 2020 Apr;162(4):795–801.

39. Schramm J, Aliashkevich AF. Surgery for temporal mediobasal tumors: experience based on a series of 235 patients. Neurosurgery. 2008 Jun;62(6 Suppl 3):1272–82.

40. Mezzacappa FM, Thorell W. Neuronal Brain Tumors. [Updated 2022 Nov 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan- . Available from: https://www.ncbi.nlm.nih.gov/books/NBK576406/. 2024;576406.

41. Perucca P, Gilliam FG. Adverse effects of antiepileptic drugs. Lancet Neurol. 2012;11(9):792–802.

42. Kowalewski A, Durślewicz J, Zdrenka M, Grzanka D, Szylberg Ł. Clinical Relevance of BRAF V600E Mutation Status in Brain Tumors with a Focus on a Novel Management Algorithm. Target Oncol. 2020 Aug;15(4):531–40.

43. Klimko A, Dandes M, Paslaru F, Giovani A. Primary Anaplastic Ganglioglioma of the Temporal Lobe With Brainstem Involvement: A Case Report and Literature Review. Vol. 12, Cureus. United States; 2020. p. e12060.

44. Marks AM, Bindra RS, DiLuna ML, Huttner A, Jairam V, Kahle KT, et al. Response to the BRAF/MEK inhibitors dabrafenib/trametinib in an adolescent with a BRAF V600E mutated anaplastic ganglioglioma intolerant to vemurafenib. Pediatr Blood Cancer. 2018 May;65(5):e26969.

45. Salem U, Kumar VA, Madewell JE, Schomer DF, de Almeida Bastos DC, Zinn PO, et al. Neurosurgical applications of MRI guided laser interstitial thermal therapy (LITT). Cancer imaging Off Publ Int Cancer Imaging Soc. 2019 Oct;19(1):65.

46. Okasha M, Ineson G, Pesic-Smith J, Surash S. Transcortical Approach to Deep-Seated Intraventricular and Intra-axial Tumors Using a Tubular Retractor System: A Technical Note and Review of the Literature. J Neurol Surg A Cent Eur Neurosurg. 2021 May;82(3):270–7.

47. Vasiljevic A, François P, Loundou A, Fèvre-Montange M, Jouvet A, Roche PH, et al. Prognostic factors in central neurocytomas: a multicenter study of 71 cases. Am J Surg Pathol. 2012 Feb;36(2):220–7.

48. Haydon DH, Dahiya S, Smyth MD, Limbrick DD, Leonard JR. Greater extent of resection improves ganglioglioma recurrence-free survival in children: a volumetric analysis. Neurosurgery. 2014 Jul;75(1):37–42.

49. Zaghloul KA, Schramm J. Surgical management of glioneuronal tumors with drug-resistant epilepsy. Acta Neurochir (Wien). 2011 Aug;153(8):1551–9.

50. Goel A, Vutha R, Shah A, Singh K, Goel N, Shenoy A, et al. Large lateral intraventricular tumors - Outcome of radical surgery. J Clin Neurosci Off J Neurosurg Soc Australas. 2021 Jun;88:205–12.

51. Al-Umran MR, Al-Umran SR, Arab AF, Dababo MA, Alotaibi FA. Lateral ventricular liponeurocytoma: Review of literature and case illustration. Neurochirurgie. 2021

Nov;67(6):579–86.

52. Wang KE, Ni M, Wang L, Jia G, Wu Z, Zhang L, et al. Cerebellar liponeurocytoma: A case report and review of the literature. Oncol Lett. 2016 Feb;11(2):1061–4.

53. Gembruch O, Junker A, Mönninghoff C, Ahmadipour Y, Darkwah Oppong M, Sure U, et al. Liponeurocytoma: Systematic Review of a Rare Entity. World Neurosurg. 2018 Dec;120:214–33.

54. Terrier LM, Bauchet L, Rigau V, Amelot A, Zouaoui S, Filipiak I, et al. Natural course and prognosis of anaplastic gangliogliomas: a multicenter retrospective study of 43 cases from the French Brain Tumor Database. Neuro Oncol. 2017 May;19(5):678–88.

55. Selvanathan SK, Hammouche S, Salminen HJ, Jenkinson MD. Outcome and prognostic features in anaplastic ganglioglioma: analysis of cases from the SEER database. J Neurooncol. 2011 Dec;105(3):539–45.

56 Glory E, Daniel O, Favour U, Oyewole A, Browne K, Okikiade A, Afolayan-Oloye O. Pineal Body Tumor: An Overview of the Pathophysiology. Asian J. Med. Health. [Internet]. 2022 Jul. 6 [cited 2024 Jun. 4];20(10):75-89. Available from: https://journalajmah.com/index.php/AJMAH/article/view/709

57 Sarhan AM. Detection and Classification of Brain Tumor in MRI Images Using Wavelet Transform and Convolutional Neural Network. J. Adv. Med. Med. Res. [Internet]. 2020 Jul. 30 [cited 2024 Jun. 4];32(12):15-26. Available from: https://journaljammr.com/index.php/JAMMR/article/view/3731

58 Kienzler JC, Becher B. Immunity in malignant brain tumors: Tumor entities, role of immunotherapy, and specific contribution of myeloid cells to the brain tumor microenvironment. European Journal of Immunology. 2024 Feb;54(2):2250257.

59 Sailunaz K, Alhajj S, Özyer T, Rokne J, Alhajj R. A survey on brain tumor image analysis. Medical & Biological Engineering & Computing. 2024 Jan;62(1):1-45.