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Worldwide Risk Analysis of Brain Tumours in Young Adults and Adolescents

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ABSTRACT

In those under 20, the most common solid malignancies are brain tumours, which account for most of cancer-related deaths. By gathering information from institutional series, population-based studies, and case-control investigations done all throughout the world, this review examines the weight, incidence, and risk elements related with brain tumours in children and adolescents. Recent epidemiological data from countries like Finland, Canada, the United States, China, and Korea points to an annual incidence rate among young people ranging from 3.5 to 5. 1 case per 100,000, with the highest rates observed among youngsters aged 0 to 4. Among the most frequent tumour types are pilocytic astrocytoma, medulloblastoma, ependymoma, aberrant teratoid/rhabdoid tumours, and medulloblastoma. Studies show small local variations in the incidence of tumour types; germ cell tumours are somewhat more common in Asian populations and show a slight male bias. This review focuses on brain tumour in the young population it's a subject of growing medical and public health concern, this evaluation emphasizes the essential need of continuous monitoring using national cancer registries, molecular tumour profiling and improved early detection and genetic testing initiatives. Understanding regional variances and risk factors for paediatric brain tumour patients can inform future studies, focused prevention campaigns, and improved survival and treatment outcomes. This information was gathered from reputable websites such as DOAJ (directory of open access journals), ERIC (education resources information centre), ScienceDirect, PubMed and google scholar. The diagram has taken from biorander while the graph, pie chart and flow chart are made in MS word.

INTRODUCTION

Largest part of the nervous system is the brain and it is divided in three sections: the cerebrum responsible for memory, learning and voluntary movement; the cerebellum playing a role in balance and coordination; and the brain-stem with vital life functions (breathing, heartbeat, and awake). A brain tumour is an abnormal growth of cells in the brain that multiplies at an abnormal rate, also referred as a tumour, may affect cells around the brain or on brain and the tumour can grow large (sometimes five centimetres) since these brain regions are able to do various functions and the areal volume that this brain areas take will be much larger too. There are numerous causes for brain tumors: genetic mutations, radiation exposure, family history, environmental factors and the status of the immune system.

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Young people are vulnerable to further taunts inherited genetic disorders, fast growth of cells in the development of the brain or birth defects that leave them more susceptible. The Primary and Secondary Brain tumors may either develop in the primary or secondary parts of brain and they can metastasise to the brain from other parts of the body. The most affected cells are Glial cells- Astrocytes, Oligodendrocytes and Ependymal cells. The increasing risk of brain tumors, while remaining quite low in absolute terms at the population level, likely reflects a confluence of exposure factors that affect both older and younger age groups. Although the incidence of brain tumors is higher in adults, with a mean age at diagnosis of about 61 years, an appreciable burden occurs among younger age groups such as children and eat imaging (MRI) for better diagnosis as well adults, while for decades, no substantive advance has been made in neurooncologist over decades. and other CNS tumors are the most frequent types of cancer in children 0-14 years of age. Part of this is due to improved diagnostic imaging, such as MRI or CT scans, allowing for the detection of smaller or asymptomatic tumors at an earlier, more easily treatable stage. In younger persons, there are more frequent types, including medulloblastoma and pilocytic astrocytoma, as well as germ cell tumors. Genetic predispositions also are a factor, with rare inherited disorders — neurofibromatosis 1 and 2, tuberous sclerosis, and Li-Fraumeni syndrome — elevating the risk for the young. Early exposure to environmental agents such as pesticides and specific chemicals is also being studied but further data is required in order to establish whether these are significant factors.

In addition, subjects who had received radiotherapy for other indications, particularly to the head or neck Kids who get radiation might have a higher chance of getting brain tumors later on. Younger kids are more at risk when it comes to thinking problems linked to radiation. Brain tumors can mess with how brain cells work, and it depends on where the tumour is. When a tumour grows, it can push against the healthy parts of the brain. It gets in the way of signals and brain activity. To illustrate, the emergence of a tumour in the frontal lobe can result in altered behaviour, compromised decision-making skills, or deficient reasoning abilities. The impacts of a brain tumour are mostly determined by where it is situated inside the brain. Tumours found in the motor cortex, which is in charge of regulating movement, could potentially bring about feebleness or problems when moving the limbs. same as a tumour affecting the cerebellum, the part of the brain that governs equilibrium and synchronization, has the potential to make ambulation or detailed manual tasks demanding.

Tumours that are even in close proximity to the eyes or pathways associated with vision might also have an impact on eyesight, which could lead to unclear vision or even the loss of sight. the tumour cells make the blood flow back, which means brain cells do not get enough oxygen. Plus, tumors can stop fluid from flowing correctly in the brain. This can cause pressure and things like hydrocephalus. In addition to pushing on things, bad tumors can destroy healthy brain tissue. This messes up how brain cells talk to each other. All this pressure harms brain cells and makes symptoms worse. Where a tumour is located is important to know, since it changes a person's symptoms. It also means a person will need a plan for how to treat it. Scientists think that some bad brain tumors, like gliomas, can connect to healthy brain cells and help make the tumour grow. It could also cause cancer to spread.

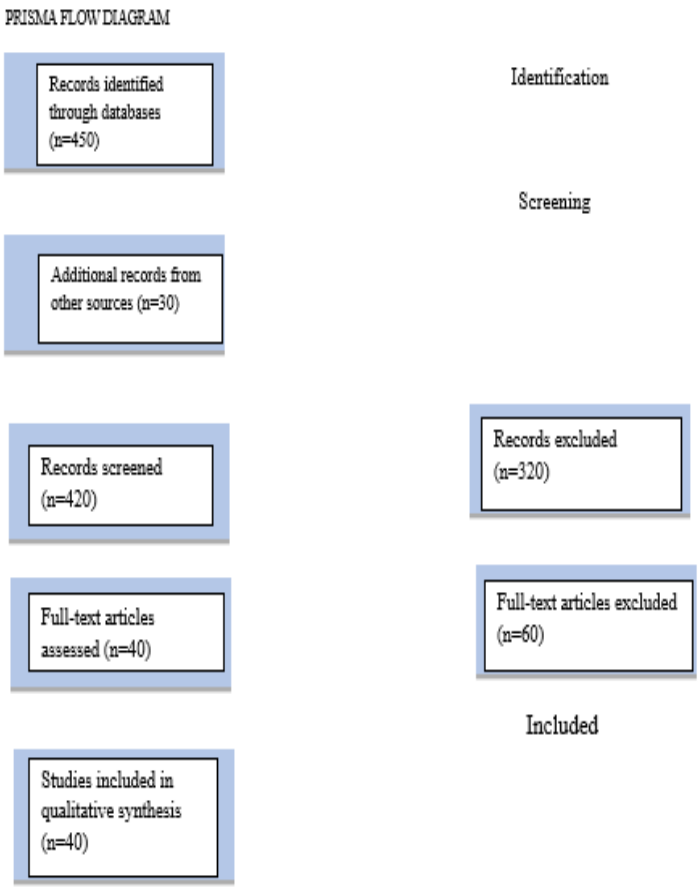


Fig 1: 2020 Prisma flow diagram

BRAIN TUMOUR PREVALENCE

Brain tumours, unlike other malignancies, are not as common; they are more complex to diagnose, and treatment options often have a poor prognosis. The global incidence of primary brain tumours is approximately 10.8 per 100,000 person years, glioblastomas have higher rates at 17.7 per 100,000 as estimated in a meta-analysis by Paula de Robles et al (2014). More specific subtype data is available including the variations of oligodendroglia tumours which sit around 3.9 and neuroepithelial tumours which are about 34.7 per 100,000. A tertiary care study from South India found meningiomas and glial tumours to be most prevalent,

showing an incidence rate of 3.9 cases per 100,000 people in males and 3.0 cases in females. Data from a Bangalore hospital (N=4,295) also noted the prevalence of meningiomas (23.2%) and glioblastomas (15.5%) as the most common malignancies in adults. In contrast, India-centric research on the paediatric population presents a different trend, where astrocytic tumours (34.7%) and medulloblastomas (22.4%) are more common. Even though these tumours are classified as under. The graph shows a noticeable difference in brain tumour reporting across India. Urban areas like Bangalore have the highest number of patients, while remote or rural regions report very few cases. Interestingly, there's a consistent trend of more males being affected in the data. The high rate of paediatric brain tumors underscores the urgent need for better diagnostic facilities and greater awareness, especially in underrepresented regions like the North-East, the data shown in the graph given in figure 2 (graphical representation of patient's male and female in India by case study) (das, 2014)

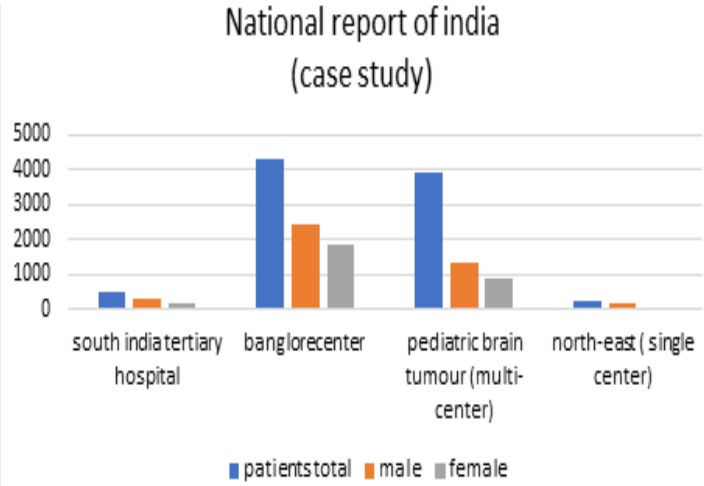
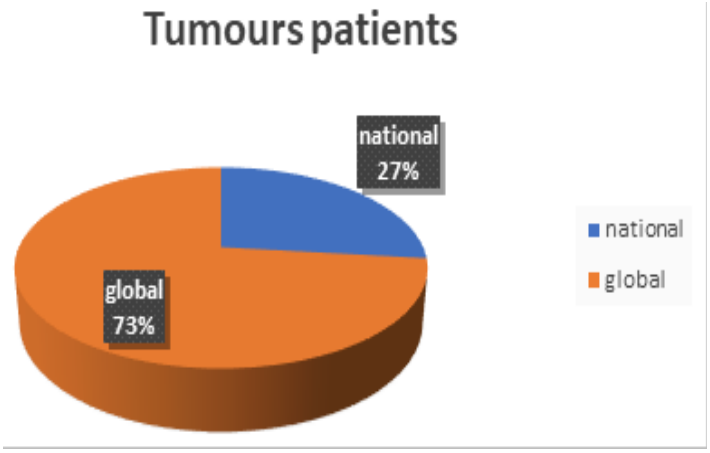


Fig 2: Graphical representation of patient's male and female in India by case study

have infratentorial tumours (Source: Arora RS et al., 2011; PubMed ID: 21483119). With an annual incidence of 5.26 per 100,000 children in the USA, according to the SEER database, paediatric CNS tumours account for approximately 20–25% of all childhood malignancies on average, highlighting a lower range of detection in comparison to other tumour kinds. Much like India, various other developing nations face issues with underreporting due to not having imaging and registry systems in place, which skews the actual numbers. Thankfully, the regions are still reliable; prompt diagnosis is vital, and astrocytoma's and medulloblastomas are most prevalent. In figure 3 (ratios of global and national tumour patients from 2002-2024) (das, 2014) the pie chart shows that India represents a notable 27% of all tumour cases worldwide, highlighting the serious nature of cancer as a public health issue. With the global share at 73%, India's figure reflects not only its large population but also the increasing awareness of tumors, thanks to advancements in medical infrastructure, reporting, and public knowledge over the last two decades. This distribution underscores the urgent need for improved cancer control strategies in India, while also placing the situation in a broader global context.



PAEDIATRIC

After leukaemia, intrusive paediatric brain tumours are the second-most frequent type of childhood cancer. Additionally, brain tumours are the most common solid tumour found in children's bodies. A multicentre centre Indian trial that looked at 3,936 children in seven significant locations is quite helpful in this regard. The study revealed that these tumours were the most frequently identified. 34.7% of astrocytic tumours Medulloblastoma/primitive neuroectodermal tumour (PNET): 22.4% 10.2% of cases involve craniopharyngiomas. 9.8% of cases are ependymomas. In addition to demographic data, this research also revealed that males are more prevalent. Most incidents in their sample occurred between the ages of 5 and 15. Younger children often had higher concentrations of supratentorial tumours, whereas older children tended to

INHERITED RISK FACTORS AND THE GENETIC SYNDROME

In children and teenagers, a considerable number of brain tumours are linked to genetic syndromes. Genetic syndromes are the cause of a significant proportion of brain tumours, especially in children and adolescents. Changes to the germline associated with tumour suppressor or DNA repair genes are typically passed down in a dominant fashion. Mutations in TP53 lead to the development of Li-Fraumeni syndrome (LFS) which presents with gliomas and medulloblastomas in some affected individuals. These tiny genetic changes can really throw a wrench in how your cells grow and divide, making it more likely for you to develop brain tumors earlier or more often than most people. If brain tumors run in your family or if you have one of those well-

known genetic disorders, your risk goes up. Doctors usually recommend that people in these situations think about getting genetic testing or chatting with a knowledgeable counsellor. It's definitely wise to be cautious. Now to clarify brain tumors are basically cells in the brain that are acting out and multiplying itself or we can say it's a neoplasia. Most of the time, it's a stroke which has no clear cause. However, in a small percentage of cases such as 5 to 10 percent, we can trace it back to your genes. Figure 4 (types of brain tumours, Genetic of common paediatrics brain tumours) Some people inherit genetic syndromes that raise the risk, not just for brain tumors but also for various other cancers and unusual health issues of tumours but for a whole problem of other cancers. (Sati S.F., 2021)

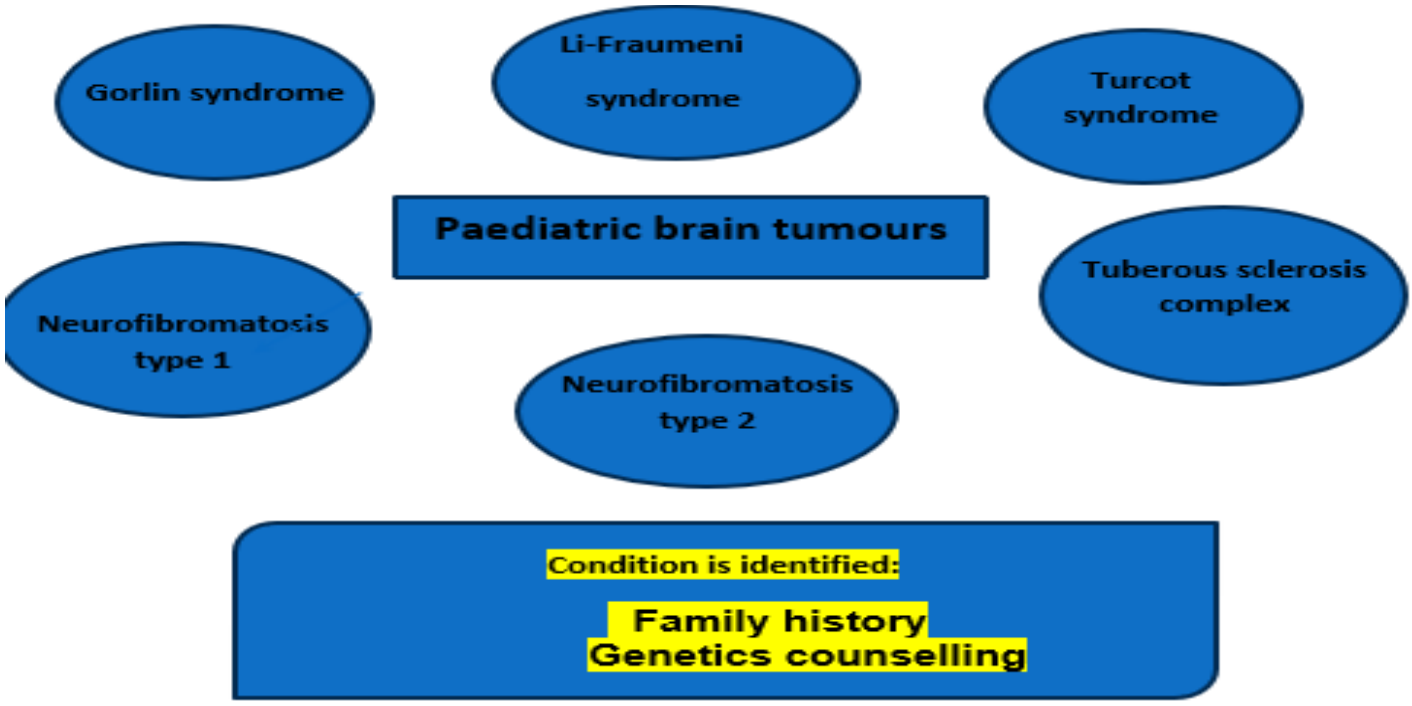


Fig 4: Types of brain tumours, Genetic of common paediatrics brain tumours

Li-Fraumeni syndrome, caused by a mutation in the TP53 gene, can increase the risk of brain tumours like Medulloblastoma, Glioblastoma and Astrocytoma's. Some young populations are more likely to develop brain tumours due to genetic conditions. This young once may also face an increase the risk of developing other types of cancer later. Quick detection and screening are crucial to manage these risks. Each child's cancer risk may differ vastly. The Neurofibromatosis type 1 (NF1) optic pathway gliomas and astrocytoma's are usually encountered along with skin markings and learning challenges due to mutations in NF1 gene.

These toddlers and teenagers might also go through a higher risk of growing other types of tumours in their life span. quick detection and monitoring it's essential to manage this risk. He brains tumour is more like to develop on young population. The Neurofibromatosis type 1 (NF1) optic pathway gliomas and astrocytoma's are usually encountered along with skin markings and learning challenges due to mutations in NF1 gene. NF2: A Genetic Condition Affecting Teenagers Neurofibromatosis type 2 (NF2) is a genetic condition that can affect teens. It's caused by mutations in the NF2 gene and can lead to the growth of tumours in the brain and spine, including: Meningiomas and Spinal ependymomas, these can cause problems like Hearing loss, Difficulty balancing and Other related issues. it improves the quality of life by helping to manage these symptoms under medical care and quick detection.

The Tuberous Sclerosis Complex (TSC): It's a Gene mutation in the TSC1 and TSC2 genes which can lead to the symptoms of (TSC) complex conditions which affect toddlers, and this tuberous sclerosis is a genetic disorder that can affect the teenager by two types epilepsy and Skin lesions. Few kids with TSC may also develop brain tumours called Subependymal giant cell astrocytoma's (SEGAs). Prompt diagnosis and treatment can help handle these symptoms and improve standard of life. Young children with Gorlin syndrome which is associated with PTCH1 mutations have an elevated risk for medulloblastomas while also developing jaw cysts and skin cancers. People with Turcot syndrome are more susceptible to glioblastomas and medulloblastomas, alongside familial colorectal cancer due to APC, MLH1, or PMS2 mutations. Medulloblastomas are linked to APC while glioblastomas are associated with mismatch repair.

In the figure 5 (the diagram shows the difference between the healthy and tumour brain) (Seano, 2020) a healthy brain maintains balance through a well-functioning network of blood vessels and a strong blood-brain barrier (BBB). On the other hand, glioblastoma tumour cells disrupt this balance

by breaking down the BBB, invading blood vessels, and throwing normal brain functions into chaos. This contrast sheds light on why glioblastoma is so aggressive, why it poses such a challenge for treatment, and why it often resists therapies, as it compromises both the integrity of blood vessels and the protective barriers in the brain.

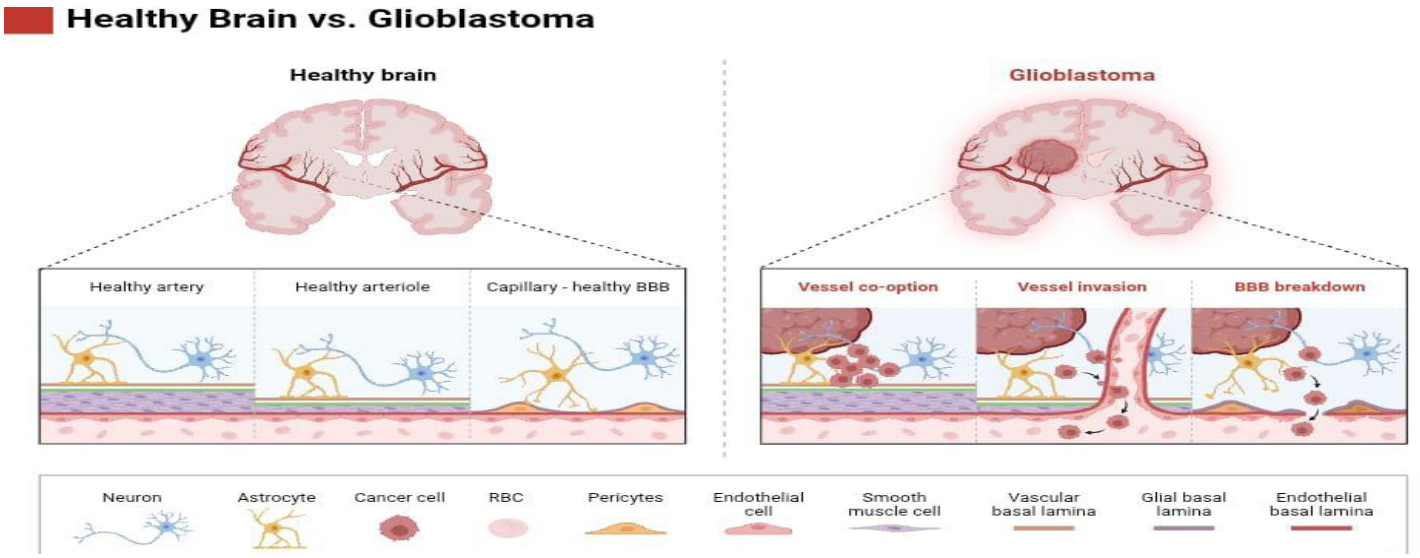


Fig 5: Diagram to show the difference between the healthy and tumour brain, bio render

THE EFFECTS OF INTERNAL AND OUTSIDE HAZARDS

The normal brain cells operation is interfered by the factors can be genetic and environmental which means internal and external sometimes mix of it which may cause the brain cancer. There are internal risks of encompassing the genetic mutations which is pass down through generation and by ancestors cause by cellular irregularities in the genes which is seen in some conditions like li-Fraumeni, tuberous sclerosis and then in some case of Neurofibromatosis. When it's difficult for the body to control the development of growth of the cancerous cells rising the risk of quick onset of brain tumours in an individual, well mutations in genes are responsible for DNA repair or tumour suppression. the significant risks factor is acquired mutation to those without any genetic predisposition are spontaneously arising from defects in cell division and aging here the external hazard are encompasses which gives an exposure to the ionizing radiation to recognise the past cancer therapies and the nuclear incidents. there are other possible environmental hazards which involves in the use of specific chemical for longer period of time examples are pesticides and industrial solvent.

The excessive use of mobile phone which have electromagnetic radiation can cause these cancerous issues. The proofs of some of these are unclear till now in this one is viral infection although these internal and external work collectively can influence that raise the chance of this unusual brain cell growth which will lead to brain tumours. Figure 6 (factors affect the genetics which cause the brain tumour) (Mazloumi2018) Internal (genetic mutations, inherited syndromes) and external (radiation, chemicals, environmental exposures) hazards act together to increase the risk of abnormal brain cell growth and tumour development.

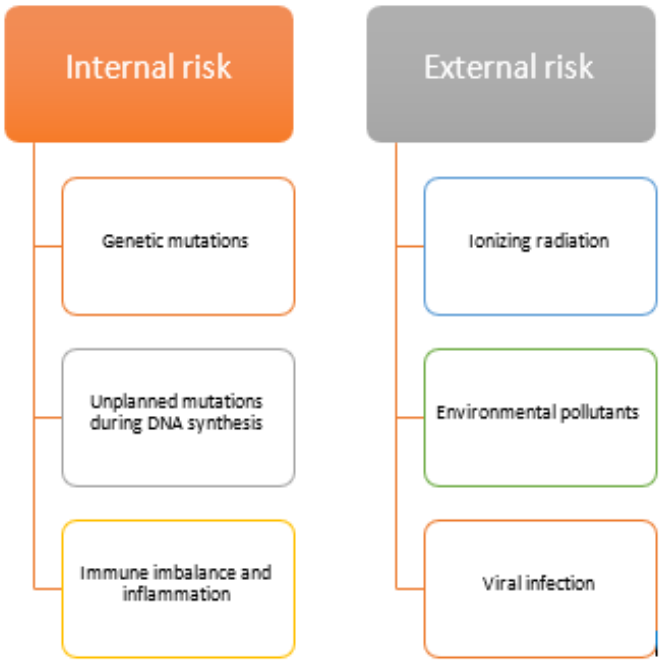


Fig 6: Factors affect the genetics which cause the brain tumour.

DISORDERS LINKED TO PREGNANCY AND NEWBORNS

Comprehensive cohort and case-control studies have identified numerous pregnancy and birth-related factors associated with childhood brain tumours: Increased fatal growth/high birth weight (weight > 4000 g or large for gestational age) has been consistently linked to a marginally

greater risk of paediatric brain tumours—especially pilocytic astrocytoma's—irrespective of gestational age. This could be affected by increased levels of growth factors like IGF-I and IGF-II that promote cell growth and prevent apoptosis. Congenital anomalies, particularly nervous system malformations, are associated with a notably higher risk of brain tumours in infancy. Children with congenital anomalies exhibit a heightened risk of central nervous system (CNS) tumours, potentially reaching 2.5 times higher, which can increase to a 5- to 6-fold rise for tumours detected in infants under 1 year old.

A maternal history of multiple pregnancy losses (≥ 2 after 20 weeks' gestation) was found to triple the risk for CNS tumours and to significantly raise the risk for high-grade gliomas in children by approximately 14 times. These findings suggest that repeated miscarriages could serve as indirect signs of underlying developmental or genetic problems. Clinical records show that maternal viral infections during pregnancy were associated with an estimated 10- to 11-fold increase in the risk of malignant brain tumours in young children (<15 years), even with the small number of cases. Immune or blood disorders in parents or foetuses during pregnancy showed weak associations with CNS tumour risk, but results were ambiguous. A study in California suggested increased risks of medulloblastoma if mothers had blood or immune problems. Studies on Neurofibromatosis Type 1 (NF1) cohorts have revealed that increased birth weight may raise the likelihood of optic pathway gliomas in affected children, possibly suggesting IGF-associated growth signalling. However, most peri-gestational characteristics (e.g., parental age, plurality) did not show a notable effect on over.

Upcoming research employing platforms like Oncaeid's DNA/RNA-centric next-generation sequencing (NGS) panel—in conjunction with chromosomal microarray analysis (CMA) for paediatric and AYA central nervous system (CNS) tumours found that 66% of instances exhibited clinically significant genomic changes, with diagnostic markers found in 62% and therapeutic targets recognized in 18% of instances. Notably, germline cancer predisposition variants were found in approximately 27% of patients, confirming inherited mutations (e.g., TP53, NF1, NF2, MSH6, PMS2) in about 9 individuals afterward.

DEVELOPMENT IN GENETIC SCREENING AND RISK PROFILING

Prevalence of Germline Vulnerability in AYA Gliomas In the SPECTA-AYA study targeting AYA high-grade gliomas, 14% of cases displayed pathogenic constitutional (germline) variants, especially in IDH-wildtype glioblastomas and paediatric-type diffuse HGGs. Molecular profiling impacted patient care by improving diagnoses and guiding targeted therapies through pathways like RAS/RAF/MAPK and PI3K/AKT/mTOR. In the 2025 case study and analysis of patients on genetic screening of kids as well as the AYA solid tumours are emphasizes the actionable alteration across multiple case studies by the NGS. Despite varying methodologies, the review concluded that standardized genetic testing could significantly improve personalized treatment strategies however, existing discrepancies demand cohesive protocols.

Retrospective and cohort studies have shown that central nervous system tumours are among the tumour types most likely to suggest an underlying cancer predisposition in AYAs. A study performed at a single centre revealed that 88% of CNS tumour cases in AYAs exhibited indications of inherited vulnerability, emphasizing the need for routine germline evaluation in this group. As we Incorporating the Germline Testing into AYA Cancer Therapy. There is notable prevalence of germline variants where the data suggest that very few if we see only 16% of AYA patients gets the formal germline testing although up to 40% patients. currently the clinical guidelines are based on their tumour background which Integrated the genetic counselling into multidisciplinary clinical care, which was crucial for improving the identification of types, then personalizing treatment for specific patient, and supporting families. The recent studies of brain tumours have understood that the complex function which involves in the genetic modification, tumours immune dynamics and the epi-genetic shifts play a role in the formation of brain tumours in AYA age groups of 15-39. the brain tumours in youngster and adolescents which diffuse the astrocytoma often show the activity of alternation in the MAPK pathways which includes the BRAF mutation, NF1 variants amplification and detection of PDGFRA, MYCN and CDKN2A. These define a distinct molecular category known as DAYA tumours, exhibiting specific epigenetic and infiltrative traits. These characterize a unique molecular group referred to as DAYA tumours with epigenetic and infiltrative characteristics. In young population of this generation are high grade gliomas which often features the mutation of histone H3 as well as alternation in ATRX, DAXX and TP53. These promote aggressive tumour characteristics and are almost non-existent in adult HGGs.

GENETIC AND CELLULAR INTERACTIONS

Cell: The recent Research found that certain genes of specific individual are crucial for the development of specific brain tumours in children, showing potential targets for new treatments. The CRISPR screens in functional genomics are shared and tumour-type are gene dependencies in adult and kids brain tumours. Paediatric isolates—such as ATRTs or DIPG—depend on genes like FGFR1, SMARCC2, and

ADAR, discovering the vulnerabilities that could pave the way for new therapeutic strategies. At the cellular level of distinct patterns are seen in immune cell infiltration which influence the tumour microenvironment (TME) in young population and AYA brain tumours. Low-grade gliomas are typically have increased the quantities of $CD8^+$ T cells and B cells, whereas high-grade embryonal tumours such as medulloblastomas and ATRTs shows that the reduced TIL levels and distinct immune subtype classifications—from immunologically "hot" to "cold" phenotypes. These patterns are associated with tumour grade, molecular subtype, and prognosis. Furthermore, the epigenetic regulation is crucial for both cancerous cells and invading immune cells. The slightest Changes in chromatin remodelling and DNA methylation in gliomas will directly influence tumour development meanwhile also modifying the immune cell activation or suppression, aiding in tumour immune symbiosis. Focusing on these epigenetic interactions appears promising for improving the effectiveness of immunotherapy in gliomas. The figure 7 (Genetical interaction) (TP, 2017) illustrates the central dogma of molecular biology: the process by which genetic information flows from DNA to RNA to protein. It shows transcription, where DNA is copied into mRNA, followed by translation, where the mRNA code is used to create a chain of amino acids, which then folds into a functional protein. In tabular-column 1 we can see all the key phrases in this review articles

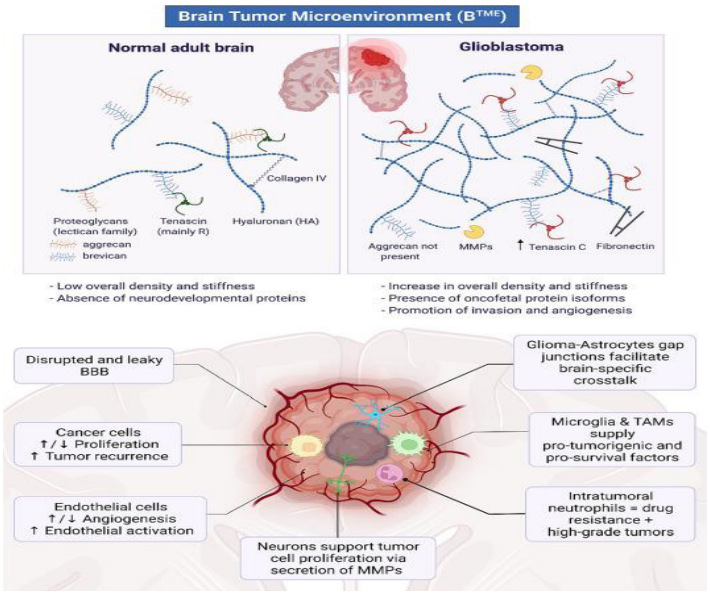


Fig 7: Genetical interaction, bio render

KEY PHRASES

Terms	meaning
· Atypical Teratoid/Rhabdoid Tumour (ATRT)	It's a Rare and aggressive cancer that typically affects young population
· Ependymoma	The tumour that causes from ependymal cells lining of the brain's ventricles.
· Medulloblastoma	the Brain tumour originates in the cerebellum, typically affecting kids.
· Astrocytoma	a kind of glial cell in the brain.
· Gliomas	which support and protect neurons. Types include. Low-grade to high-grade.
· Cancer epidemiology	Distribution and determinants of cancer in population.
· Adolescent brain tumours	Abnormal growth of cell in the brain in young adults
· Cancer registries	Track cancer in a defined population.
· AYA oncology	This field improve cancer outcomes and quality of life for AYA patients.
· Onco-genetics	Hereditary cancer syndromes
· Targeted therapy in paediatric tumours	Blocks the specific molecule involved in tumour growth.
· Immunotherapy in gliomas	To recognize and attack glioma cells by immune system
· CRISPR functional genomics	Allows precise gene editing.
· Immunological disorders	Immune system malfunctions
· Maternal infections	Infection occurs in a prevent women affects both mother and foetus
· Congenital anomalies	Infant morbidity and mortality.
· High birth weight	Refers to a newborn weighing more than 4,000 grams.

· Prenatal exposures	Environmental, chemical and lifestyle will impact on development of feats
· Pesticide exposure	Contact with any pesticide by the mother during pregnancy.
· Parental smoking	Active smoking of prevent women
· Ionizing radiation	Ionizing radiation can cause DNA damage.
· ATRX, DAXX genes	Mutations in these genes have been implicated in various cancers.
· CDKN2A, PDGFRA, MYCN alterations	Alterations in these genes have been associated with various cancers
· Histone H3 mutation	These mutations can disrupt normal chromatin function and contribute to tumorigenesis.
· BRAF mutation	These mutations can lead to uncontrolled cell growth and tumorigenesis.
· MAPK pathway	contribute to cancer development and progression, making them a target for cancer therapy.
· DNA repair defects	Understanding DNA repair mechanisms can inform cancer diagnosis and treatment
· Tumour immune microenvironment	Immunotherapies aim to modulate the TIME to enhance anti-tumour responses.
· Epigenetic regulation	involve heritable changes in gene expression without altering the DNA sequence
· Next-generation sequencing (NGS)	NGS has revolutionized genetic research and clinical diagnostics. high -throughput sequencing and Genome-wide analysis
· Molecular profiling	Identify genetic mutations and Guide personalized treatment
· Genetic screening	Enable early intervention
· Turcot Syndrome	Turcot syndrome requires multidisciplinary management, including genetic counselling, surgery, and surveillance.
· Tuberous Sclerosis Complex (TSC1, TSC2)	Treatment may involve mTOR inhibitors.
· Li-Fraumeni Syndrome (TP53 mutation)	TP53 mutations disrupt normal cell cycle regulation, DNA repair, and apoptosis.
· Neurofibromatosis type 2 (NF2)	NF2 is a tumour suppressor gene, and mutations lead to tumour development in the nervous system.
· Neurofibromatosis type 1 (NF1)	NF1 is a tumour suppressor gene, and mutations can lead to various complications, including cancer.
· Subependymal giant cell astrocytoma (SEGA)	Increased intracranial pressure
· Pilocytic astrocytoma	Is usually slow-growing and benign
· Inherited cancer predisposition	predisposition syndromes increase the risk of developing certain cancers.
· Germline mutation	which occur in non-reproductive cells.
· Gorlin Syndrome (PTCH1 mutation)	Caused by mutations in the PTCH1 gene.

Table 1: Key phrases to understand the brain tumour

CONCLUSION

Brain tumours in young population are frequently result from the inherited genetic conditions such as Li-Fraumeni syndrome, NF1, and TSC. Tumour risk is caused by internal factors like gene mutations, immune dysfunction and external hazards such as radiation, toxins. during the pregnancy and early development might also be influential. Improvements in genetic screening and profiling now assist in identifying individuals at high risk early on. At the cellular level, distinct genetic and immune interactions stimulate tumour development in younger individuals. Grasping these interconnected elements is essential for enhancing early identification and tailored therapy.

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