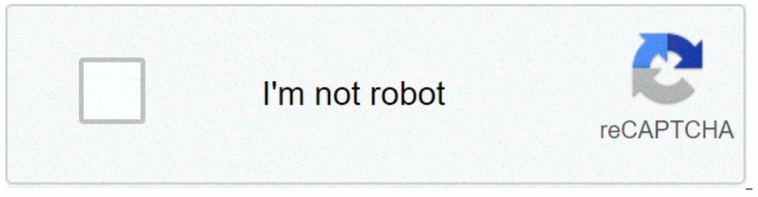

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Pilocytic astrocytoma glioblastoma multiforme

Juvenile pilocytic astrocytoma (JPA) is a slow-growing brain tumor that develops – usually in children and adolescents – from cells called astrocytes. Astrocytes are glial cells, which means they are a type of cell that supports nerve cells in the brain and spinal cord, so juvenile pilocytic astrocytoma is a type of glioma. However, unlike other gliomas, juvenile pilocytic astrocytomas are considered low degrees, which means that they are very benign tumors, and the prognosis for recovery is excellent. Juvenile pilocytic astrocytoma is a cystic (fluid-filled) tumor, not solid mass. On the World Health Organization's classification scale, JPA is a grade I tumor, highly unlikely to grow or spread to other areas. Other types of gliomas in children include: Fibrillary astrocytoma. This Grade II astrocytoma is benign, but it infiltrates into the surrounding brain tissue, making it difficult to surgically remove it. Anaplastic astrocytoma. It is a Stage III (malignant) astrocytoma. Glioblastoma multiforme (GBM). This rapidly growing Stage IV tumor is the most malignant type of astrocytoma. Read more about the types of brain tumors in children. Juvenile pilocytic astrocytoma can develop anywhere where there are astrocytes that exist throughout the brain and spine. JPAs commonly develop in the cerese, in the back of the brain; near the optic nerve; in the brain site; or in the brain. Depending on its location, JPA can cause different symptoms because it causes pressure on different areas of the brain (see Symptoms of juvenile pilocytic astrocytoma). What causes juvenile Pilocytic Astrocytoma? It is not known what causes abnormal cell division that leads to a brain tumor. But there is no way to prevent them, and there is nothing you can do to reduce a child's risk of developing one. Call our children's brain and spine centers at 212-746-2363 to make an appointment for evaluation, or use our online form to request an appointment. Meeting request | Bespoop patient: Jeffrey Greenfield, Ph.D., MD.Last reviewed/last updated: August 2018Image of Thom Graves, Grade I Astrocytoma CMI: Surgery is standard treatment. Complete surgical removal of available astrocytomas is often possible and successful. Accessible tumors are those that can be operated on without unacceptably serious damage to other parts of the brain. If an operation is performed, the surgeon will try to remove all identifiable parts of the astrocytoma, if possible. When the astrocytoma involves a key part of the brain, partial removal of growth usually reduces pressure, relieves symptoms and helps control seizures. Complete or partial removal of the astrocytoma is sometimes followed by radiation therapy, which destroys the remaining tumor cells. When using CT (computed tomography) and MRI (magnetic resonance imaging), radiation can sometimes be delayed by several months or years while the patient is scanned at regular intervals. Radiation as the primary therapy is Chemotherapy can be administered after irradiation in an attempt to destroy all cells that remain or can also be administered during radiation treatment. Chemotherapy can be used instead of radiation in very young children to prevent damage to the developing brain. The type of chemotherapy drug therapy selected is determined by a neuroo-oncologist who examines the degree of tumor, previous treatment and the current state of health of the affected individual. Grade I astrocytoma can sometimes progress to a higher degree, so follow-up scans at regular intervals are necessary to control re-growth. Stage II astrocytoma: Treatment depends on the size and location of the tumor. Surgery can be used to remove available tumors. As with all infiltrating astrocytomas (grade II-IV), it cannot be completely removed with surgery because tentacles-like projections of the tumor grow into surrounding tissue. Radiation can be used if the tumor is not accessible or in addition to surgery. Grade II astrocytoma can also progress to a higher grade, so follow-up is required to check re-growth. A recurrent tumor can be treated with surgery, radiation or chemotherapy. Stage III astrocytoma: Treatment depends on the size and location of the tumor, how it looks under the microscope and how far it has spread. Standard treatment is surgery and radiation therapy, accompanied or followed by chemotherapy. If surgery is not possible, radiation and chemotherapy may be recommended. There are several different types of radiation therapy, including conventional door radiation, concentrated radiation, stereotactic radiosurgery of implanted radiation or conformal radiation. Radiation oncologist determines the most suitable form of radiation for a particular tumor. Chemotherapy agents commonly used to treat grade III astrocytoma include carmustine (BCNU), lomustin (CCNU), procarbazine, cisplatin and temozolomide. Biodegradable plates (called Gliadel plates) containing BCNU are sometimes inserted into the cavity that remains after the tumor has been removed. Stage III astrocytoma tend to repeat, and treatment depends on the degree of tumor that is repeated. The Food and Drug Administration (FDA) has approved temozolomide (Temodar) for the treatment of adults with anaplastic astrocytoma that did not respond to other forms of treatment (refractory anaplastic astrocytoma). For more information contact:Merck Corporate Headquarters 2000 Galloping Hill Road Kenilworth, NJ 07033-0530Grade IV astrocytoma: The three main forms of GBM treatment are surgery and radiation or chemotherapy. These treatments can be used alone or in combination with each other. Initial treatment in most cases is surgical excision and removal of as much tumor as possible (resection). Often, only part of the tumor can be safely removed, since malignant cells can spread to the surrounding brain tissue. Since surgical intervention cannot completely remove radiation therapy and chemotherapy are used after surgery to continue treatment. The FDA has approved temozolomide (Temodar) for the treatment of adults with GBM. Temozolomide is used in parallel with radiation therapy, and also for a period after the completion of radiotherapy. For more information, contact: Merck Corporate Headquarters 2000 Galloping Hill Road Kenilworth, NJ 07033-0530Gliadel Wafers have been approved by the FDA for the treatment of individuals with newly-diagnosed GBM as an adjunct to surgery and radiation. It has also been approved for individuals with repetitive GBM. Several platelets are located in the cavity formed by surgical removal of GBM. Wafers release drugs into the surrounding tissue for two or three weeks. For more information, contact:MGI Pharma, Inc. 5775 West Old Shakopee Road Suite 100 Bloomington, MN 55437 Phone: (952) 346-4700 Fax: (352) 346-4800 Symptoms associated with juvenile pilocytic acystrotoma vary depending on the size and location of the tumor. JPA can develop anywhere with the central nervous system (i.e. the brain and spinal cord). Most cases occur in the lower part of the brain near the back of the neck, which controls movement and balance (cerebral structure), brain inclination, hypothalamic region or pathways of the optic nerve. The most common finding associated with JPA is increased pressure in the brain, which can be caused by the tumor itself or by blocking fluid-filled spaces in the brain called ventricles, leading to abnormal accumulation of cerebrospinal fluid (CSF) in the brain. Symptoms commonly associated with increased pressure on the brain include headaches, lethargy or drowsiness, vomiting, and changes in personality or mental state. In some cases, JPA may also be associated with seizures, vision problems such as blurred vision or double vision (diplopia), gradual changes in behavior or mood and weakness of the arms and legs, leading to coordination problems. JPA in the brain stems may be associated with nausea, vomiting, impaired ability to coordinate voluntary movements (ataxia) and wryneck (torticollis). Eye symptoms, including swelling of the optical disc (papilledema) and involuntary rapid eye movement (nystagmus) may also occur. In some cases, paralysis (palsy) of the sixth and seventh cranial nerves may develop. JPA in the pathways of the optic nerve may be associated with vision loss, degeneration (atrophy) of the optic nerve, papilledema, nystagmus, and protruding eyeball (proptosis). JPA in the hypothalamic region may be associated with weight gain or loss, premature puberty or diencephalic syndrome, which is characterized by failure to thrive, abnormal thinness, irritability, and eye abnormalities. Rare pilocytic astrocytomes (PA) have atypical histological and clinical-diardological properties that increase the differential diagnosis of glioblastoma. Whether auxiliary studies can complement histopathological when these cases are accurately placed on the WHO Grade I PA spectrum at a higher degree of glioma, it is not always clear, partly because these cases are not common. Ten PA's with atypical clinical and histological features and six paediatic glioblastoma (pGBM) multiforms for BRAF V600E, IDH1, IDH2 and TP53 mutations were analysed here. Expressions of the protein Ki-67, p53 and p16 have also been studied by imunohistochemisty. The status of the BRAF-KIAA1549 merger was assessed in the PA subgroup. The rate of BRAF-KIAA1549 fusion was high in these PA (5/7 tumours) including four extracerebellar examples. A single BRAF V600E mutation has been identified in the fusion-negative extracerebellar PA of a very young child who has succumbed to the disease. TP53 mutations were only present in malignant gliomas, including three pGBMs and one case marked as PA with anaplastic features (with a pGBM consultation opinion). IDH1 and IDH2 were wild in all cases, consistent with earlier findings that IDH mutations are not typical in high-quality gliomas of patients ≤14 years. Imunohistochemical studies have shown substantial overlap of Ki-67 marking indices, an imperfect correlation between the p53 designation and the TP53 mutation status, and a complete loss of p16 in only two pGBMs, but without PA. These results suggest that whereas (a) BRAF-KIAA1549 fusion may be common in PA with atypical clinical and histological features, including those at extracerebellary sites, (b) the BRAF V600E mutation is unusual in extracerebellary PA and (c) analysis of TP53 mutations remains a valuable tool in identifying paediatic gliomas likely to behave in malignant fashion. Fashion.

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