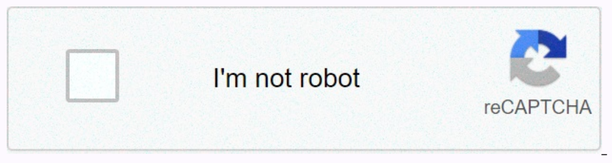

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Glioblastoma multiforme grade 4 icd 10

2016 2017 2018 2019 2020 2021 Billable/Specific Code C71.4 is a billable/specific ICD-10-CM code that can be used to indicate a diagnosis for refund purposes. The 2021 edition of ICD-10-CM C71.4 will take off on 1 October 2020. This is the American ICD-10-CM version of C71.4 - other international versions of ICD-10 C71.4 may differ. The following code(s) above C71.4 contains annotation referencesAnnotation Background referencesIn this context, annotation references refer to codes that contain:Applicable to annotations, orCode Also annotations, orCode First annotations, orExcludes1 annotations, orExcludes2 annotations, orBecludes annotations, orNote annotations, or Use Additional annotations that may apply to C71.4: C00-D49 2021 ICD-10-CM Range C00-D49NeomsplasplasnoteFunctional activityAll neoplasms are classified in this chapter, whether functionally active or not. An additional code from Chapter 4 can be used to identify functional activity associated with a neoplasm. Morphology [Histology]Chapter 2 classifies neoplasms primarily by site (topography), with broad groupings for behavior, malignant, in situ, benign, etc. The table of neoplasms should be used to identify the correct topography code. In a few cases, such as for malignant melanoma and certain neuroendocrine tumors, morphology (histological type) is included in the category and codes. Primary malicious neoplasms overlapping site boundariesA primary malicious neoplasm that overlaps two or more contiguous (side-by-side) sites should be classified in the subcategory/code .8 (overlapping lesion), unless the combination is specifically indexed elsewhere. For multiple neoplasms from the same site that are not contiguous, such as tumors in different quadrants of the same breast, codes should be assigned for each site. Malignant neoplasm of ectopic tissueMalignant neoplasms of ectopic tissue should be encoded to the said place, for example, ectopic pancreatic malignant neoplasms are coded for pancreas, not specified (C25.9). NeoplasmsC71 ICD-10-CM Diagnosis Code C712016 2017 2018 2019 2020 2021 Non-Billable/Non-Specific Code Type 1 Excludesmalignant neoplasm of cranial nerves (C72.2-C72.5)retrobulbar malignant neoplasm (C69.6-) Malignant neoplasm of brain Approximate Synonyms Anaplastic astrocytoma, occipital lobe Astrocytoma, occipital lobe Cancer of the brain, occipital lobe Ependymoma, occipital lobe Glioblastoma multiforme, occipital lobe Malignant glioma, occipital lobe Oligodendroglioma, occipital lobe Primary anaplastic astrocytoma of occipital lobe Primary astrocytoma of occipital lobe Primary ependymoma of occipital lobe Primary glioblastoma multiforme of occipital lobe Primary malignant glioma of occipital lobe Primary malignant neoplasm of lobe Primary oligodendroglioma of occipital lobe ICD-10-CM C71.4 is grouped within Diagnostic Related Group(s) (MS-DRG v38.0): 054 Nervous system neoplasms with mcc 055 Nervous system neoplasms without mcc mcc C71.4 to ICD-9-CM Code History 2016 (effective 10/1/2015): New code (first year of non-draft ICD-10-CM) 2017 (effective 10/1/2016): No change 2018 (effective 10/1/2017): No change 2019 (effective 10/1/2018): No change 2020 (effective 10/1/2019): No change 2021 (effective 10/1/2020): No change ICD-10-CM Codes Adjacent To C71.4 C69.92 Malignant neoplasm of unspecified site of left eye C70 Malignant neoplasm of meninges C70.0 Malignant neoplasm of cerebral meninges C70.1 Malignant neoplasm of spinal meninges C70.9 Malignant neoplasm of meninges, unspecified C71 Malignant neoplasm of brain C71.0 Malignant neoplasm of cerebrum, except lobes and ventricles C71.1 Malignant neoplasm of frontal lobe C71.2 Malignant neoplasm of temporal lobe C71.3 Malignant neoplasm of parietal lobe C71.4 Malignant neoplasm of occipital lobe C71.5 Malignant neoplasm of cerebral ventricle C71.6 Malignant neoplasm of cerebellum C71.7 Malignant neoplasm of brain stem C71.8 Malignant neoplasm of overlapping sites of brain C71.9 Malignant neoplasm of brain , niet nader gespecificeerd C72 Malignant neoplasma van het ruggenmerg, cranial zenuwen en andere delen van het centrale zenuwstelsel C72.0 Kwaadaardig neoplasma van ruggenmerg C72.1 Kwaadaardig neoplasma van cauda equine C72.2 Malignant neoplasma van reukzenuw C72.20 Maligne neoplasma van ongespecificeerde olfactorische zenuw Terugbetaling claims met een datum van dienst op of na 1 oktober, 2015 vereisen het gebruik van ICD-10-CM codes. The information reported is not medical advice and may not be accurate. Ik contents have only illustrative fijne and do not replace medical advice: read the warnings. Glioblastoma In the RM image note the enhancement ring (the most noticeable part of the tumor) around the central area of necrosis. Type Malignant Risk factors ionizing radiation Average age at diagnosis 53 years Ratio M:F 1.5:1 Acronyms GBM Synonyms Glioblastoma multiform,Grade IV astrocytoma Classification and external resources Neurooncology Neuroepit tissue tumors -astrocyte eli ICD-O 9440/3 Grade WHO IV glioblastoma (also known as glioblastoma multiforme or GBM -less commonly as glioblastoma polymorphic-) is the most common and most malignant cancer among glia neoplasms. Its name was established by standaard WHO-2000[1] and confirmed by standaard WHO-2007. Composed of a heterogeneous set of poorly differentiated astrocytic cancer cells, glioblastoma mainly affects adults, and usually occurs in the cerebral hemispheres; less frequently to the brain stem or spinal cord. Kom all brain tumors, except in very rare cases, do not expand beyond the structures of the central nervous system. [2] Glioblastoma can develop from a diffuse astrocytoma (grade II) or an anaplastic astrocytoma (grade III) (in this case it is called secondary, see below), but more frequently it manifests itself de novo, without any di precedent neoplasia (èè (è primary name). Treatment of glioblastoma includes surgery, radiotherapy and chemotherapy. It is difficult to treat and there are few cases of survival after three years. [3] Classification tumors of the central nervous system Tumor families according to the WHO classification of 2007. Tumors of the neuroepithelal tissue Astrocial tumors Oligodendroglial tumors Oligo-astrocytes tumors Ependimal tumors Tumors of choronic complexes Other neuropithelial tumors Neuronale and mixed neuronal-gliatumors Tumors of the pine region Embryonic tumors Tumors Tumors of the skull and spinal nerve seches Meninge tumors Meninge tumors tumorsEndotelial Tumors Mesenchymal tumors Primary melanocytic lesions Other neoplasms related to meninges Hematopoietic tumors of germ cells Tumors of the sella region Metastatic tumors In the box right are the tumor families of the Central Nervous System, according to the penultimate (2007) WHO classification. According to this classification, glioblastoma is mainly part of astrocytic tumors, along with six other types of neoplasms, in accordance with the following scheme. Tumori astrocitari (Astrocytic Tumours) Astrocitoma pilocitico (Pilocytic Astrocytoma, IDC-O 9421/1, WHO grade I) Astrocitoma pilomixoide (Pilomyxoid Astrocytoma, IDC-O 9425/3, WHO grade II) Astrocitoma subependimale a cellule giganti [Astrocitoma gigantocellulare subependimale] (Subependymal Giant Cell Astrocytoma, IDC-O 9384/1, WHO grade I) Xantoastrocitoma pleomorfo (Pleomorphic Xanthoastrocytoma, ICD-O 9424/3,WHO grade II) Astrocitoma diffuso [Astrocitoma] (Diffuse Astrocytoma, IDC-O 9400/3, WHO grade II) Astrocitoma anaplastico (Anaplastic Astrocytoma, IDC-O 9401/3, WHO grade III) Glioblastoma [Glioblastoma Multiforme, Astrocitoma di grado IV] (Glioblastoma, IDC-O 9440/3, WHO grade IV) Glioblastoma a cellule giganti (Giant Cell Glioblastoma, IDC-O 9441/3, WHO grade IV) Gliosarcoma (Gliosarcoma, IDC-O 9442/3, WHO grade IV) Gliomatosis cerebri (Gliomatosis Cerebri, IDC-O 9381/3, WHO grade III) In parentesi tonda viene riportata la denominazione ufficiale del WHO-2007, insieme al codice ICD-O (International Classification of Diseases for Oncology , International classification of diseases for oncology). [4] The WHO Grading is then added (see Gradation of tumors of the central nervous system), with the acronym WHO numeral followed by Roman numbering. The Italian definition is the most widely used version in literature. Others, if any, are in parentheses. The variants are indicated in italics. Historical notes Rudolf Virchow, who recognized the glial origin of the tumor in the first half of the 19th century glioblastoma was considered of Mesenchymale origin and therefore determined by the term sarcoma. [5] In 1863 Rudolf Virchow[6] established his glial origin. F.B. Mallory, in a 1914 memoir, states the term glioblastoma multiforme. [7] However, it is to wait until 1925 for a neoplasm, by J.H. Globus and I. Strass. [8] At this time, the most common name of the tumor is multiform spongioblastoma. [8] In 1926, a publication by P. Bailey and H. Cushing successfully reproduced Mallory's diction. [9] The 2000 WHO classification of nervous system tumors eventually puts the name to glioblastoma. [1] For a more detailed historical account, see K.J. Zülch[10] and D.S. Russell and L.J. Rubinstein. In the development of the concept that glioblastoma sometimes emerges through progression and malignancy of a lower degree lesion, the studies of H.J. Scherer (1940)[12] and J.W. Kernohan et al. played a decisive role. (1949). [13] This view has received strong support from molecular genetics studies, which have shown that there is a characteristic successive accumulation of gene changes from Grade II diffuse astrocytomes to glioblastoma (see furtherfoblastoma (see furtherfoblasticte(see furtherfoblastie, Tabb. 1 and 2). Epidemiology Glioblastoma is the most common brain tumor,[14] affecting about 12-15% of all intracranial neoplasms and 50-60% of all astrocytary tumors (see classification). [10] In most European and North American countries, the incidence is 2-3 new cases per year per 100,000 inhabitants. [15] Glioblastoma can manifest itself at any age, but is preferred in adults, peaking between 45 and 70 years. [16] Approximately two thirds of patients (70%) is of the above range. The average age was about 53 years, with a male/female ratio of 1.5:1. The latest data comes from work on 1003 biopsies for glioblastoma, by the University Hospital in Zurich. They are mentioned in P. Kleihues et al. (2000). [16] Similar data are reported by other authors. [10] In work on 488 cases, G.J. Dohrman and others[17] found that 8.8% of glioblastomes are pediatric. Cases of congenital glioblastoma are rare,[18] although the diagnosis of malignant glioma by ultrasound in utero[19][20][21] has shown that prenatal glioblastoma can occur even at 29 weeks of pregnancy. [21] Glioblastomes are most common in subcortical white matter in the hemispheres of the brain. The most affected locations are the temporal lobe (31%), the parietal lobe (24%), the frontal (23%) and occipitaal (16%). The fronto-temporal combination is typical. Neoplasm often extends through infiltration to the adjacent cortex, the basal ganglia, and then the counterlateral hemisphere. This data comes from a report of 987 glioblastomi, by the University Hospital Zurich. They are mentioned in P. Kleihues et al. (2000). [16] Intraventricular glioblastoma is exceptional,[22] while the glioblastomas of the brain are rare, although they often affect children. [17] The cerebellum and spine are rarely affected this neoplasm. [16] Etiology Traditional vision Tumors are formed as a result of abnormal and unregulated cell growth. Once the human brain completes its development, immediately after birth, the vast majority of the cells enter a state of rest, in which they no longer divide. The only exception to this rule is when a tumor develops. Tumor brain cells resume the cell cycle due to changes in some of the many genes that control cell division and growth. Although much is known about the changes of these genes in brain tumors, the main reason for the changes is currently unknown. [23] Inheritance Note that when it comes to genes, it doesn't mean that brain tumors are hereditary. Although there are syndromes in which such tumors are known, these situations (neurofibromatosis, Turcot syndrome, Li-Fraumeni syndrome, etc.) are very rare and normally known in the family before a tumor develops in a family member. [23] Risk Factors Ionizing radiation is the only unambiguous risk factor identified for glial and mening-shaped neoplasms. Radiation therapy of the skull, even at low doses, can increase the incidence of glial tumors by a factor of 3 to 7 and meningiomas by a factor of 10, with a latency period of 10 to more than 20 years after exposure. [24] [25] No other environmental or patient behaviour has been clearly identified as a risk factor. It has been reported from many angles that the use of cell phones, the proximity of high voltage cables, the use of hair dyes, head trauma, nutrition with N-nitrosamines, or other dietary factors, all increase the risk of brain tumors; However, [26] [27] [28] [29] these data are considered contradictory and inconclusive. [30] The association between the type of professional occupation and the appearance of glioblastomi has been the subject of numerous studies. Workers chronically exposed to vinyl chloride, phenol compounds and aromatic hydrocarbons were found to be at greatest risk. [31] [32] [33] [34] [35] Neoplastic brain stem cells Neoplastic stem cell hypothesis. The cell in yellow is a tumor stem. To beat the disease, you need targeted therapy for such a type of cell. Since the 1990s, studies first of animals and then of humans have shown that in the brain there is a continuous production of new cells. In particular, multipotent neuronal stem cells have been identified in the serrated gyro of the hippocampus and in the subventricular region of the lateral ventricles multipotent neuronal stem cells, i.e., capable of producing new undifferentiated cells (stem cells) and adult cells, such as neurons, astrocytes and oligodendrocytes. [36] [37] [38] [39] [40] [41] They are also suitable for self-renewal, the total number of cells remains constant. [42] On the other hand, a strand of research has found, starting in 2002, that in brain tumors, especially glioblastours, there is a hierarchy of cancer cells. In the sense that a (small) part of the tumor is made of cells that have the same characteristics as neuronal stem cells, so the authors have given the name of neoplastic stem cells of the brain (brain tumor stem cells). [43] [44] [45] These are the engine of the tumor: they continuously reproduce cancer stem cells and cancer cells (non-stem cells). And they are just the last to be subjected to treatment attacks. Neoplastic stem cells are in fact refractory radiotherapy and chemotherapy, because they are able to repair themselves over time the damage performed by traditional therapies, before the damage becomes irreversible and such that the cell becomes inactive. [46] [47] [48] [49] [50] It is therefore sufficient for a single neoplastic brain stem cell to escape surgery, restart the mechanism and undergo a resumption of the disease. It is believed that the existence of these neoplastic stem cells of the brain emerges from a flaw in the self-regulation of neuronal stem cells (self-renewal), previously mentioned. [42] Conceptual arrangement reported here in very concise form, in literature is called the Neoplastic Stem Cell Hypothesis. This pattern is followed by the vast majority of researchers. Nevertheless, there is a small but fierce minority who tend to give a different explanation of the phenomena described or to place them in a different conceptual framework. [51] [52] [53][54] Pathogenea In the following, schematically the sequence of genetic changes leading to glioblastoma is reported, as described in the last two editions of who's ranking of CNS tumors. [16] [56] We distinguish two types of changes: the activation of oncogenic factors: EFG/R (Epidermal Growth Factor/Receptor, epidermis growth factor) MDM2 (Mouse Double Minute 2 oncoprotein promotes cell survival and cell cycle progression by inhibiting the oncosuppressor TP53[57]) PDGF/R (Platelet-Derived Growth Factor/Receptor) deactivation of oncosuppressor factors: 10p, 10q, 19q (chromosomes) DCC (Deleted in Colorectal Cancer tumor suppressor gene, Gene with deletion in colorectal cancer) p16 (Tumor suppressor gene/protein) TP53 (Tumor suppressor gene/protein) PTEN (Phosphatase and TENsin homolog) is an oncosuppressor that controls cell growth, proliferation and survival. From its mutation or inhibition it can result in the onset of tumors, e.g., prostate, udder, colon and brain. [58] [59] [60]) RB (RetinoBlastoma tumor suppressor gene, protein of the Table 1 (P. Kleihues and H. Ohgaki, 1999,[61] as seen in P. Kleihues et al., 2000,[16] with graphic changes) is taken from the WHO classification of 2000 and shows mutations occurring from healthy cells to glioblastoma. In the left part you can see the activation of intermediate lesions (diffuse astrocytoma and anaplastic astrocytoma) before you reach the so-called secondary glioblastoma (this adjective here usually means not metastatic but derived from previous lesions). In the right part, the table shows the mutations that lead from healthy cells directly (de novo) to glioblastoma, then primarily called. (By the way, the percentage of the individual's presence is the change.) Tab. 1 Alterazioni genetici del Glioblastoma (2000)[16] Cellule astrocitiche differenziate ovvero precursori neuroepiteliali Mutazione di TP53 (>65%)Sovraespressione di PDGF-A, PDGFR α (~60%)√ EGFR:Amplificazione (~40%)Sovraespressione (~60%)MDM2:Amplificazione (<10%)Sovraespressione (~50%)Delezione di p16 (30-40%)Perdita di etzigeroosi su 10p e 10qMutazione di PTEN (~30%)Alterazione di RB, Astrocitoma diffuso Perdita di eterozigosi su 19q (~50%)Alterazione di RB (~25%); Astrocitoma anaplastico Perdita di eterozigosi su 10qMutazione di PTEN (5%)Perdita di espressione di DCC (~ 50%)Amplificazione di PDGFR α (<10%)√ Glioblastoma secondario Glioblastoma de novo La Tabella 2 (da P. Kleihues et al., 2007,[56] con modifiche grafiche) è tratta dalla Classificazione WHO del 2007. Further years of study and in-depth study are summarized. [62] It should be noted that the asterisk (*) reports genetic changes that differ significantly in frequency between primary and secondary glioblastomas. Table I 2 Genetic Changes in Glioblastoma (2007)[56] Differentiated astrocytic cells or precursors or stem cells TP53 mutation (59%)- TP53 mutation (59%), IIII-<3 months (68%)<6 months (59%)- TP53 mutation (59%), IIII-<3 months (68%)<6 months (59%)- TP53 mutation (59%), IIII-<3 months (68%)<6 months (68%)8%)-IIII: Diffuse Astrocytoma Diffuse Astrocytoma Mutation of TP53 (53%)- II-5.1 yearsIi : Anaplastic astrocytoma (1.9 years) Loss of heterozygous at 10q (63%)Strengthening EGFR (1.9 years) Loss of heterozygous at 10q (63%)Strengthening EGFR (1.9 years) Loss of heterozygous at 10q (63%)Strengthening EGFR (1.9 years) Loss of heterozygous at 10q (63%)Strengthening EGFR (1.9 years) Loss of heterozygous at 10q (63%)Strengthening EGFR (1.9 years) Loss of heterozygous at 10q (63 Strengthening%)of EGFR (1.9 years) Loss of heterozy8%)Deletion of p 16 I N K 4 to {\displaystyle p16^{INK4a}} (19%)TP53 mutation (65%)*PTEN mutation (4%)* Loss of heterozygosis at 10q (70%)EGFR strengthening (3 6%)%*Removal of p 16 I N K 4 to {\displaystyle p16^{INK4a}} (31%)TP53 mutation (28%)PTEN mutation (25%)** Secondary glioblastoma Primary glioblastoma of the case average age : 45 years M/F report: 0.65 95% of casesAn average age: 62 years M/F report: 1.33 The links between the two tables can be derived by examining the indicated bibliographic references. However, one thing must be emphasised. A study, even superficial of these tables, leads to the conclusion that primary and secondary glioblastomas are two distinct (although histologically indistinguishable) diseases, affecting groups of patients who are different in age and gender and develop through genetic pathways with different protein expression profiles and mRNA. These differences are important, especially as they can affect the response of radio and chemotherapy cancer and may be the target of future therapeutic approaches. [63] Complications The following is a review list of complications related to glioblastoma, which differentiates to the disease from those most closely related to therapies. [64] Many of these complications are not common and a significant number of them can be effectively kept under therapeutic control. Tumor-related complications: Cerebral edema Neurological disorders Visual disorders Hydrocephalus Gliomatosis leptomeningea Deterioration of cognitive functions Worsening psychological state (distress, etc.) Complications related to therapies: Pathologies related to surgery Infections Neurological disorders Visual disorders Pathologies related to radiotherapy Neurological disorders Visual disorders Visual disturbances Worsening cognitive functions Pathologies related to chemotherapy Blood dysfunctions Disorders of the respiratory system Diarrhea-depletion Neurological disorders Pathologies related to anticonvulsant medicines Pathologies related to inflammatory drugs Pathologies related to cytostatic drugs Pathological examination Macroscopic examination Glioblastoma untreated. Macroscopic examination Glioblastoma treated. Macroscopic examination Despite the short duration of symptoms, glioblastomas are often large at the time of presentation and can occupy more than one lobe. The lesion is usually monolateral, but those of the brain stem and the eelte body can be bilaterally symmetrical. The tumor takes the same position in the two hemispheres and comes with a butterfly appearance. Bilateral supertentorial expansion is due to rapid growth along myelinized structures, especially through the callous body and along the furnaces to the temporal lobes. The boundaries of the neoplastic mass, which is never capsuled, are blurred everywhere. The color is grayish, but abundant variegatory can be found, caused by necrosis or more or less recent bleeding, so that yellowish areas appear on the gray background, for fatty degeneration or for necrosis and reddish or blackish areas due to bleeding. The peripheral area of hypercellular tumor tissue appears as a soft, gray rhyme. Necrotic tissue can also border adjacent brain structures without a macroscopically detectable tumor intermediate zone. Central necrosis can occupy more than 80% of the total mass of the tumor. Glioblastomas are usually riddled with red and brown spots for bleeding, which are sometimes large enough to cause symptoms similar to an apoplectic stroke, which is the first clinical sign of the tumour Are. Macroscopic cysts, when present, contain a cloudy fluid from liquid necrotic tumor tissue, tumor tissue, in contrast to the well-defined retention cysts of grade II diffuse astrocytoms. Most glioblastomas in the hemispheres of the brain are clearly intraparenchymal, with epicenter in the white substance. Sometimes neoplasm presents itself as largely superficial and in contact with leptomeningi and hard mother and can be confused with metastatic carcinoma or for an additional axial lesion such as meningioma. [5] [56] Microscopic examination Glioblastoma. Hyperchromatism[65] and nuclear pleomorphism,[66] the fibrillar background, help distinguish glioblastoma from metastatic tumors and Glioblastoma lymphoma. Glandular or ribboneptropic structures reminiscent of metastatic carcinoma Glioblastoma is an anaplastic glia neoplasm that consists of poorly differentiated astrocytic cancer cells, polymorphic, with marked nuclear atypies and intense mitotic activity. Characteristics specific to diagnostic purposes include the striking microvascular proliferation[67] and the presence of necrosis. As the adjective multiform of the most common synonym suggests, the histological morphology of glioblastoma is extremely variable, with round, spindle-shaped cells of relatively small or very large size. While some glioblastomas show a high degree of cellular and nuclear polymorphism, with numerous multinucleated giant cells, others have a conformation characterized by intense cellular but rather repetitive. The astrocytic nature of neoplasm can be fairly easy to identify, at least locally, in some tumors, but difficult to spot in others, due to the high degree of anaplasia. The heterogeneity from region to region of glioblastoma is relevant and makes it difficult to diagnose it on limited samples, such as those obtained by stereotactic biopsy[68] (see illustration in surgery section.). Although the presence of poorly differentiated cells prevails, more differentiated neoplastic astrocytes can be distinguished in some places. This is particularly true for glioblastoma due to progression of a diffuse astrocytoma (grade II of the WHO scale). The transition between areas that are still recognizable astrocytic differentiation and areas with high cellular anaplasia can be continuous or sudden. A sharp variation in morphology usually reflects the appearance of another tumor, born by acquiring one or more additional genetic changes. [69] In the context of neoplasm, large areas of necrosis are observed, surrounded by nuclei arranged parallel to each other, consoping typical palisades. There is a clear proliferation of endothelial cells with the formation of numerous vessels, sometimes with a cluster or ball-like appearance. Some have ialine walls[70] and others are trumpeting. However, endotelial proliferation is not widespread, but is in some places. Around neoplasm areas of missedocytic astrocytes (grade II diffuse astrocytomes) can be found. [5] [56] Clinical signs and symptoms The clinical history of the disease is normally short (less than 3 months, in more than 50% of cases), unless the tumor develops through progression of a low-grade astrocytoma (secondary glioblastoma; see above the pathogenic section). The symptoms of glioblastoma are non-specific symptoms[71] of a expanding mass in the skull, thus of increasing endocranial pressure. Often are headaches, nausea, vomiting, dilation of brain vessels with changes from the retina to papilledema, hemiaresis, hemianesthesia, hemianopsia, diplopia, aphasia and seizures. The percentage of patients subjected to seizures reaches up to a third. Finally, non-specific neurological symptoms such as obnubilization of consciousness and personality changes should be noted. [56] Characteristic imaging and brain tumors Gadolinium is the chemical element of atomic number 64. The symbol is Gd. (See Contrast Medium for MRI) PET of a glioblastoma brain tumor after 3 gross total removal operations The presence of cerebral neoplasm can be effectively revealed through computed tomography (CT) and nuclear magnetic resonance imaging (MRI). MRI has a higher sensitivity than CT scans when identifying lesions; However, it is not always easy for the patient to access and has a number of contraindications: it cannot be performed in pacemaker carriers, prostheses that are incompatible with the magnetic field, metal clips, etc. CT remains the method of choice in the detection of internal

calcifications of injuries or butterosie of the case or skull base. The use of the contrast medium (iodato in case of CT scans, paramagnetic in case of MRI (gadolinium)), makes it possible to obtain information on vascularization and integrity of the blood-brain barrier, a better definition of the tumor nodule than the surrounding cerebral edema and makes it possible to promote hypotheses about the degree of malice. Radiological research also makes it possible to evaluate the mechanical effects (and the resulting changes in the proportions of brain structures) due to the presence of the foreign mass: hydrocephalus and hernias, the effects of which can also be deadly. Finally, given the operation, the study specifies the location of the lesion and the proximity (or even involvement) of the tumor in absolutely vital areas of the brain (so-called eloquent areas). For this purpose, MRI is higher than CT because it is able to deliver three-dimensional images. [72] Before closing this section, it is useful to draw attention to some concepts and terms that are for the later sections will be useful. Radiological aspect of neoplastic tissue That you want to place the phenomenon of change from the radiological point of view of neoplastic tissue compared to normal cerebral parenchyma (changes in the electronic density of materials in case of CT and signal intensity for MRI). Like most pathological tissues, tumors are characterized by greater accumulation of intracellular water. Ipodensi, that is, of a lower density than cerebral parenchyma, mri appear ipointensi in the images T1-weighted and hyperintensite in that DP- and T2-weighted. (See the entries Computer Tomography and Magnetic Resonance Imaging.) [34] [73] Contrast improvement in an X-ray plate the healthy brain region should not signal certain luminescences. It is therefore natural to pay attention to the parts of the greatest contrast signal. In the tumor, in general, the greater proportion of contrast improvement is due to the particular blood-tumor barrier, which allows the passage of iodine (CT) and gadolinium (MRI) into the intratumor extravascular interstitial space: this increases the signal (density or intensity) of the tumor. However, be careful that the contrast enhancement does not clearly delineate the tumor from perilesional edema: in fact, the anatomic pathological find in malignant infiltrators (such as glioblastoma and anaplastic astrocytoma) shows neoplastic tissue even beyond vasogeneous edema (i.e. by the destruction of the blood-brain barrier by the tumor), which is not easily demonstrated by x-ray images. [34] [73] MRI image of a recurding glioblastoma. Improvement may be due to radioneculoshosis rather than disease recovery PET image solves the question in favor of the presence of cancer, because it shows the very luminous area, sign of intense metabolic activity Post-surgical control Post-surgical control by MRI (or CT) in order to determine the radicality of the removal of a tumor is considered questionable in the literature : the examination should be carried out within 24 hours of surgery, i.e. before changes in the blood-brain barrier supported by fibrotic-scar tissue phenomena are in place; in other words, the physiological scar has a contrast improvement that can be easily mistaken for a residue or a regrowth of tumor. Even after radiosurgical treatment, a radioneculus (see below the homonym section) can have imaging and contrast enhancement characteristics with the appearance placed almost on top of that of a malignant glioma. Only through functional methods such as positron emission tomography (PET) with fluorodesoxyglucose (FDG-PET), which demonstrates a higher set of glucose by the tumor than healthy tissue, is it possible to evaluate the absence of metabolism in necrosis compared to relapse (although necrosis and relapse may coexist). As an alternative to PET, spectroscopy analysis can be used by MRI (see nuclear magnetic resonance spectroscopy and functional magnetic resonance imaging): in the map of the metaboloids of this method there is the peak of choline (Cho) which is associated with the synthesis of cell membranes: a high peak is indicative of high cell turnover, as happens in tumors. [34] [73] Imaging diagnostics and glioblastoma Axial (horizontal) CT scan or glioblastoma-based encephalopod (25-year-old patient) Coronal CT scan (frontal) of the same tumor (25-based and Tacephalopod year-old patient) Sagittal MRI image of 15-year-old patient suffering from glioblastoma Coronal MRI image of the same 15-year-old patient CT scan CT scan shows an irregular morphology lesion, mainly hypodensa, highly uneven due to the presence of large necrotic areas of sharper hypodensity and fixed hyperdense areas. The latter are an expression of rapid growth and therefore of high malice. Frequent hemorrhagic areas ranging from small mouths to large blood areas that can involve the whole lesion. Characteristic is the butterfly morphology if present the interest of both hemispheres by the callous body. After contrast appearing coarse impregnation wax around the necrotic areas. In MRI, the solid part ipointensa appears in T1 and hyperintensa in T2 with higher signal zones in the parts with the strongest cellulality. Necrotic regions, always hyperintense in T2, can occur hypo-, iso- or hyperintense in T1, depending on protein content or hemoglobin breakdown products. Improvement after contrast medium is generally intense and irregular on the periphery of the tumor and mainly identifies the proliferive cellular component of neoplasm. Common pointy and meandering areas of absence of current signals are associated with the presence of rich neovascularization. These newly formed pathological blood vessels are free of blood-brain barrier: this explains both abundant and gross impregnation and perilesional vasogenic edema (see the previous section) due to the passage of fluid in extracellular location. [72] [73] Differential diagnosis Differential diagnosis includes: metastases, spontaneous brain hemorrhages, abscesses, atypical forms of multiple sclerosis, secondary barrier damage to radiotherapy. [72] Imaging diagnostics. Conclusions The first step to consider when evaluating a patient suspected of having cerebral neoplasm is magnetic resonance imaging. Such an examination would also be advisable for any patient suffering from seizures, for whom there is no immediate and plausible justification. Normally, the resonance reveals without particular difficulty the presence of glioblastoma as the cause of symptoms and no further investigations are required. [74] Treatment In the treatment of glioblastoma, as with any other brain tumor, we distinguish supportive therapies from curative therapies. [75] [76] [77] Supportive therapies Support treatment is intended to relieve symptoms and improve the neurological functions of the patient. Primary supporting drugs are anti-epileptic drugs and corticosteroids. Anti-epileptic drugs Valproic acid. Chemical structure Anti-epileptics are given to about 25% of patients who have had seizures when presenting the disease. Phenytoin (300-400 mg/d) is the most commonly used drug, but carbamazepine (600-1 000 mg/d), phenobarbital (90-150 mg/d) and valproic acid (750-1 500 mg/d) are equally effective. The doses of all these anti-epileptic drugs should be adjusted to the levels that are then found in the patient's blood, to provide maximum protection. Equally effective are newly developed anti-epileptic drugs, such as levetiracetam, gabapentin, lamotrigine and topiramate. Most of these new active ingredients have the benefit of causing poor cognitive side effects and, because they do not cause the microsomal liver system, do not change the metabolism of chemotherapy. These new anti-epileptic drugs quickly replace classic drugs in front-line anti-epileptic therapy. [74] Prophylaxis Future clinical trials have yielded negative results in an effort to demonstrate the effectiveness of prophylactic use of anti-epileptic drugs in the case of patients with brain cancer who had never complained of seizures. As a result, the medical literature advises against its use for this purpose, except for the period associated with surgery, when its use may reduce the incidence of postoperative seizures. In the case of patients who have never had seizures, it is advisable that anti-epileptic drugs are no longer administered within 2 weeks of surgery. [74] [75] [78] Corticosteroids Desametasone. Chemical structure Corticosteroids-based drugs are able to reduce peritumoral edema, which decreases the mass effect of neoplasm and reduces endocranial pressure. As an immediate effect, headache relief and an improvement in lateralistic signs is obtained (see symptomatology described in the epilepsy voice). The corticosteroid of choice is deametasone, due to minimal mineral corticotic activity. The starting dose is about 16 mg/d. This amount may be increased or reduced to the minimum dose needed to control neurological symptomatology. Long-term use of corticosteroids is associated with hypertension, diabetes mellitus, hyperosmolar non-chetosic hyperglycemic state (life-threatening disease), myopathy, insomnia and So that in the brain tumor patient the steroid dose should be gradually reduced as soon as possible, once treatment has begun. For most patients, corticosteroids are discontinued when they have completed radiotherapy. Patients on steroids for more than 6 weeks are advised antibiotic prophylaxis for pneumonia of cute pneumocystis, a remedy that should remain for 1 month from the cessation of corticosteroid administration. [74] Healing therapies Healing therapies of brain tumors mainly include surgery, radiotherapy, and chemotherapy. The first step, if possible, is to draw up a general therapeutic plan that makes it possible to outline the order and the individual elements of multidisciplinary treatment. Craniotomy surgery. The arachnoid is the thin bluish layer under which the brain can be seen. The hard mother is the white layer under the yellow tongs. The next layer is the skull and, finally, the skin. The surgical approach should be carefully chosen to obtain the maximum possible removal of the tumor, preserving the vital structures of the brain and minimizing the risk of postoperative neurological deficiency. The objectives of the operation are: to obtain an accurate histological diagnosis; reduce the mass effect on the brain caused by the tumor and/or peritumoral edema; where appropriate, maintain or restore the flow of cephalorachidian fluid; achieve a (potential) cure by the total removal of neoplasm (in the case of glioblastoma, surgery very rarely achieves healing, but it reduces the size of the tumor in such a way that it becomes more manageable by radio and chemotherapy). Removal greater than 98% of tumor volume (total resection) increases survival compared to subtotal or partial resection. Extended subtotal resection does not appear to confer any survival advantage over biopsy or partial resection. [74] [79] Stereotactic biopsy. You suck a small part of the tumor through a needle into a vacuum system. The structure around the patient's head ensures the right axis to the lens. (See above, Microscopic examination) In case of recurrence of the disease (and this happens in almost all glioblastomas), or enlargement of the part of the tumor that remains from the operation, or radionecrosis (both the resumption of the disease and the radionecrosis cause mass effect and edema and, as mentioned in previous sections, are indistinguishable with classical resonance) a second intervention is used, to reduce the effects of the newly formed mass on the cerebral parenchyma. In a repeat situation, it is difficult to achieve recovery, but it usually results in an improvement in the quality of life and a modest extension of the In general, a second intervention is excluded in patients with a Karnofsky index (KPS) of less than or equal to 60 or in patients who are not eligible for adjuvant therapies after surgery. [74] [79] Before closing this section, it is worth mentioning clinical studies involving intrathematic administration of chemotherapy or immunotherapy or radiant fluids during the surgical operation. These studies are in the first phase of experiments. [80] [81] [82] Placement on the working bed of carmustine-impregnated wafers is the only case of intracaviary chemotherapy currently (September 2008) approved by the FDA (Food and Drug Administration) for the case of glioblastoma. [83] [84] In the 2010s, surgery evolved to remove awake patient cancer. Thanks to this operation, it is possible to investigate whether a certain part of the brain can be removed without the patient being affected after surgery. Specific areas of the brain are electrified to understand whether the patient responds when a psychologist communicates with surgeons to understand how long the tumor can be removed. [85] [86] Radiotherapy Radiotherapy, normally performed after surgery, concerns the part of the brain affected by the surgery as well as a slight outside margin, and aims to damage the DNA of any cancer cells left after the operation and the surgeon escaped because they are not visible under a microscope (as they infiltrated more or less far from the area of the operation). If radiotherapy succeeds in damaging these cells before they have the ability to repair DNA and resume cell multiplication, the patient gains in survival. Clinical studies on high-grade gliomas (anaplastic astrocytoma, oligodenthroglioma anaplastic, anaplastic oligostrocytoma, glioblastoma) conducted by the U.S. Brain Tumor Study Group (BTSG) showed that postoperative radiotherapy at doses above 50 Gy provides an improvement in survival compared to no postoperative treatment and that 60 results in significantly longer than 50 Gy. [87] [88] This amount of radiation corresponds to a dose just above that needed for the formation of radioneculerosis, so the radiotherapy treatment of 60 gys administered in total in 30-33 fractions, one per day, was chosen as the standard. [74] [88] [89] Glioblastoma patients over 60 years of age with a shortened therapy of 40 Gy in 15 fractions show survival identical to those obtained with the standard regimen. It is therefore reasonable that such patients should use such reduced treatment. [90] Approximately half of patients with anaplastic astrocytoma respond to radiotherapy with 60 Gy (data verifiable by radiographic evidence); The 25% for patients with glioblastoma. For both neoplasms, cases of complete healing by radiotherapy are very rare. [74] In an attempt to improve results, a number of new approaches have been developed, such as hyperfractionated radiotherapy (HFRT), brachytherapy (use of directly deposited radioactive needles), radiosurgery. The latter has had a certain interest in the recent past, as it is a non-invasive procedure, which in some cases can even be carried out in a day hospital situation. It requires a very careful selection of patients, because among other things it requires that the neoplasm is not extended, but very concentrated. Apart from special cases, these new techniques have been shown to significantly improve the overall survival of the patient. [74] [88] Radionecrosis Is already mentioned in previous sections to radio-induced necrosis. This complication is primarily produced by brachytherapy and radiosurgery and determines the symptom atology of mass effect, described above, in about 50% of malignant glioma patients. With corticosteroid treatment it is often possible to control the edema around the radio necrotic area. This, in the long run, in turn produces dependence on steroids, with all the complications of long-term use listed (in the section of Corticosteroids). In severe cases, surgery should be used to remove necrotic mass. [74] Chemotherapy Chemotherapy is also intended to damage the organization of DNA of cancer cells, possibly lasting after surgery and escaping radiotherapy. If chemotherapy succeeds in releasing such DNA, the tumor cell passes into the phase of programmed death (apoptosis). Temozolomide. Chemical Structure Chemotherapy Brings Limited Benefits for Glioblastoma Patients. In clinical trials, the use of nitrosures has not significantly prolonged average survival in all patients, but a subgroup of them appears to benefit from long-term survival with the addition of chemotherapy to radiotherapy. Prognostic factors such as age, Karnofsy index, etc. [74] (See, however, later, the case of temozolomide). In a large Phase III study, patients (diagnosed with glioblastoma and without any previous radio chemotherapy treatment) were randomized to receive radiotherapy (group A) or radiotherapy at the same time as daily administration of the drug temozolomide, followed by monthly administration of temozolomide (group B). Out of a total of 573 patients, the average survival increased from 12.1 months (group A) to 14.6 months (group B). But more importantly, the two-year survival time has more than doubled, from 10.4% of group A to 26.5% for group B.[91] Combined treatment with radiotherapy temozolomide was well tolerated on average and with minimal additional toxicity, making this protocol the therapeutic standard of choice for all new glioblastoma patients. [74] As a byproduct of the above study, a tumor protein (MGMT) has been identified that can predict, with a useful approach in practice, which patients will benefit from the combined protocol. This method is still being tested by the scientific community and is only mentioned here for information. [92] [93] [94] Cannabinoids A separate speech deserves cannabinoids. Cannabis derivatives are known to be effective in oncology (via tetrahydrocannabinol (THC) capsules or the synthetic nabilone), on the one hand to combat chemotherapy-induced nausea and vomiting, on the other hand to stimulate appetite and alleviate the feeling of anxiety or actual pain. [95] [96] Their ability to inhibit growth and angiogenesis in malignant gliomas has been demonstrated. [97] [98] The results of a pilot study on the use of THC in patients (in the terminal phase) with recurt glioblastoma were found to be worthy of further study. [99] But extremely interesting is the discovery (confirmed so far on animals) that cannabinoids can attack the neoplastic stem cells of glioblastoma, with the result on the one hand to cause their differentiation in mature (and therefore more treatable) cells and on the one hand to inhibit tumorese. [100] Repetition Despite the (limited) initial successes of the therapies, virtually all glioblastomas return. In this situation, the patient can undergo a second operation (if he is under the conditions provided) or he can benefit from targeted radiotherapy techniques (radiosurgery, if neoplasm meets the requirements previously seen. Note that it is usually not possible to perform a second round of standard radiotherapy at 60 Gy.), or he may receive chemotherapy other than temozolomide (to which the patient no longer responds). [74] Mitoxantrone. Chemical structure Typical recurrence chemotherapy are procarbazine, nitrosures, melphalan, carboplatin and others. In recent clinical studies, the use of mitoxantrone[101] and the combination of hydroxyurea with imatinib mesilate have shown interesting anticancer activity. [102] Other clinical studies indicate the use of epidermis growth factor receptor inhibitors[103] or the use of anti-angiogenesis agents,[104][105][106] or combined therapies of locally injected radiopharmaceuticals along with locally injected pure chemotherapy. [107] All these studies and protocols are examined by the scientific community. However, a goal that is being pursued is to a practical method to characterize the classes of patients for whom a protocol gives the best results, so that by assigning the specific patient to the most appropriate class, it gets the protocol of greater effectiveness, utility and minimal impact. Prognosis randomized clinical trials from 1978 showed that the average survival after surgery, for patients treated only with corticosteroids, was 14 weeks, rising to 38 weeks after radiotherapy. [108] Chemotherapy prolongs survival. Patients treated with surgery, radiotherapy and chemotherapy had an average survival of approximately 1 year,[74] rising to 15-18 months in 2015. [109]. A 1998 study of 279 patients who had undergone fully aggressive treatment reported that only 5 of them (1.8%) have survived more than 3 years. [3] In fact, each patient responds differently to therapies, so that for the individual the chances of survival (in the case of full treatment, including recurrence management) in 2008 were equal to 57% to one year, 16% to two years and 7% to three years. [110] Data from 2014 confirm poor survival over 2.5 years, with only 5% of treated patients surviving 5 years after diagnosis,[111] while for untreated patients the average survival is three months after diagnosis. [112] After 3 years in literature we talk about long survival. Survival after 3 years is rare, more common in secondary glioblastoma. A long-term limit case (11 years) is the famous psychiatrist Frans David Servan-Schreiber, who survived the first 8 years of stage IV astrocytoma and then 11 years in Phase I secondary glioblastoma derived from it, for a total of 19 years of life in good quality, after the diagnosis of cancer that in 1992 had him a few months. Thanks to experimental treatment, he survived 1 year stage IV glioblastoma in the frontal lobes, in reasonable condition (2010-2011). [113] [114][115][116][117][118] An important fact comes from a 2003 study: the likelihood of surviving for another year, after one, two, three, four or five years after the craniotomy already one, two, three, four or five years after surviving the craniotomy, it is 64.8%, 58.7%, 85.7%, 80.0%, respectively. [119] Animation of a case of a glioblastoma of the patient before transverse SurgerySagittalChoonal Animation of a grosstotal removal case of a glioblastoma of the same patient, After two years TransversalSagittalCoonal Animation of a case of new grosstotal removal of a glioblastoma of the same patient, after 5 years and 6 months of the first transversal OperationSagittalChoonal Notes ^ a b (EN) Kleihues P , Cavenee World Cup, eds. (2000), Pathology and genetics of tumors of the nervous system, Classification of tumors by the World Health Organization. Lyon, France: IARC Press, ISBN 92-832-2409-4. On the WHO 2000 rating can be found on Copy Copy on brainlife.org. URL accessed December 31, 2010 (archived from the original url on January 11, 2014).. URL accessed December 31, 2010. ^ A. Templeton, S. Hofer, M. Topfer, A. Sommacal, C. Fretz, T. Cerny, S. 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