CASE SERIES

# Gliomatosis Cerebri: A Report of Three Cases with Literature Review

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Submission: 15 Apr. 2015
Accepted: 24 May 2015

**Citation**
Hendi AM. Gliomatosis cerebri: A report of three cases with literature review. JKAU Med Sci 2015; 22 (4): 35-43. DOI: 10.4197/Med. 22.4.5

## Abstract

Gliomatosis cerebri is a rare glioma of poor prognosis due to its diffuse infiltration of the central nervous system involving three or more lobes of the brain. It has non-specific symptoms such as epilepsy, cognitive disorders, headache and change in personality. Three cases of gliomatosis cerebri are reported. Extensive literature review was done. This review includes definition, type, clinical presentation, diagnosis [radiological and pathological], deferential diagnosis, treatment, and prognosis. In conclusion gliomatosis cerebri was initially a postmortem diagnosis. Following the advancement of the MRI, it became a radiological diagnosis that induces diffuse involvement of at least three contiguous lobes of the brain with relative preservation of its general anatomical configuration. It is manifested clinically as a very aggressive disease with poor prognosis. Imaging plays an important role in initial diagnosis, biopsy planning and follow up.

## Keywords

Gliomatosis cerebri; Magnetic resonance tomography; Magnetic resonance spectroscopy; Biopsy; Pathology

## Introduction

Gliomatosis cerebri (GC) is a rare, diffuse glial tumor which presents as extensive diffuse infiltration to the central nervous system (CNS). It involves at least three neighboring brain lobes, frequently in both hemispheres, and often extends to infratentorial structures and sometimes to the spinal cord[1-3]. The neuronal architecture of the brain is usually preserved[4].

According to the World Health Organization (WHO), GC is classified into one of two types[5]. Clinically, its prognosis is considered poor[6]. The most frequent pathological findings are of a Grade III astrocytoma. Nevin[7] was the first to describe GC in 1938 depending on the pathological findings in his three cases.

Histological diagnosis is limited by the focality of the biopsy probe and cannot appreciate the extent of the disease. That is why before the advancement of computed tomography (CT) and magnetic resonance imaging (MRI), GC was a postmortem diagnosis. Neuroimaging plays an essential role in antemortem diagnosis by demonstrating the extent of brain involvement. MRI especially is the method of choice for diagnosis and patient follow up.

## Case 1

A 39 year old male patient was brought to the emergency department in coma, with urinary and stool incontinence. According to his family, he suffered from headaches and vomiting and was referred by his family doctor to a neurologist who gave him antihypertensive treatment for his known hypertension. During admission he developed an epileptic status, he had

Glasgow Coma Scale (GCS) of 11 and left hemiparesis. The patient could not speak. He was referred for a brain CT, which showed central hypodensity and loss of gray-white matter differentiation in and around the corpus callosum, effacement of the ventricles and prominent leptomeningeal contrast enhancement. Subsequent MRI (Fig. 1) provided more detailed anatomical information on the extent of the lesion showing diffuse Fluid-attenuated inversion-recovery (FLAIR) signal hyperintensity with loss of white/gray matter differentiation in the corpus callosum with extension to the cingulate gyri and the frontal and temporal lobes bilaterally. There was compression of the frontal portion of the lateral ventricles. Diffusion Weighted Imaging (DWI), showed diffusion restriction with low Apparent Diffusion Coefficient (ADC) suggestive of a hypercellular infiltrative process without evidence of acute ischemic lesions. There was leptomeningeal enhancement [suggestive of meningeal irritation and venous stasis] but no intraparenchymal enhancement. Magnetic resonance spectroscopy (MRS) showed a marked increase of choline (Cho) and decrease of N-Acetyl aspartate (NAA) in the area of the signal abnormality in the corpus callosum (Fig. 1). Based on these findings the diagnosis of GC was suggested.

Biopsy was performed and histology revealed tumor tissue with moderate cell density and pleomorphic cells and three mitotic figures. Atypical rounded-oval cells were found in the glial matrix. There was no evidence of necrosis or vessel proliferation. Based on these findings the diagnosis of an anaplastic astrocytoma Grade III was established. Imaging and histological evidence confirmed the diagnosis of GC.

## Case 2

A 39 year old male patient was brought by ambulance to the emergency department with Glasgow Coma Scale 15, following sudden onset of first time epilepsy. Clinical history was unremarkable except of an episode

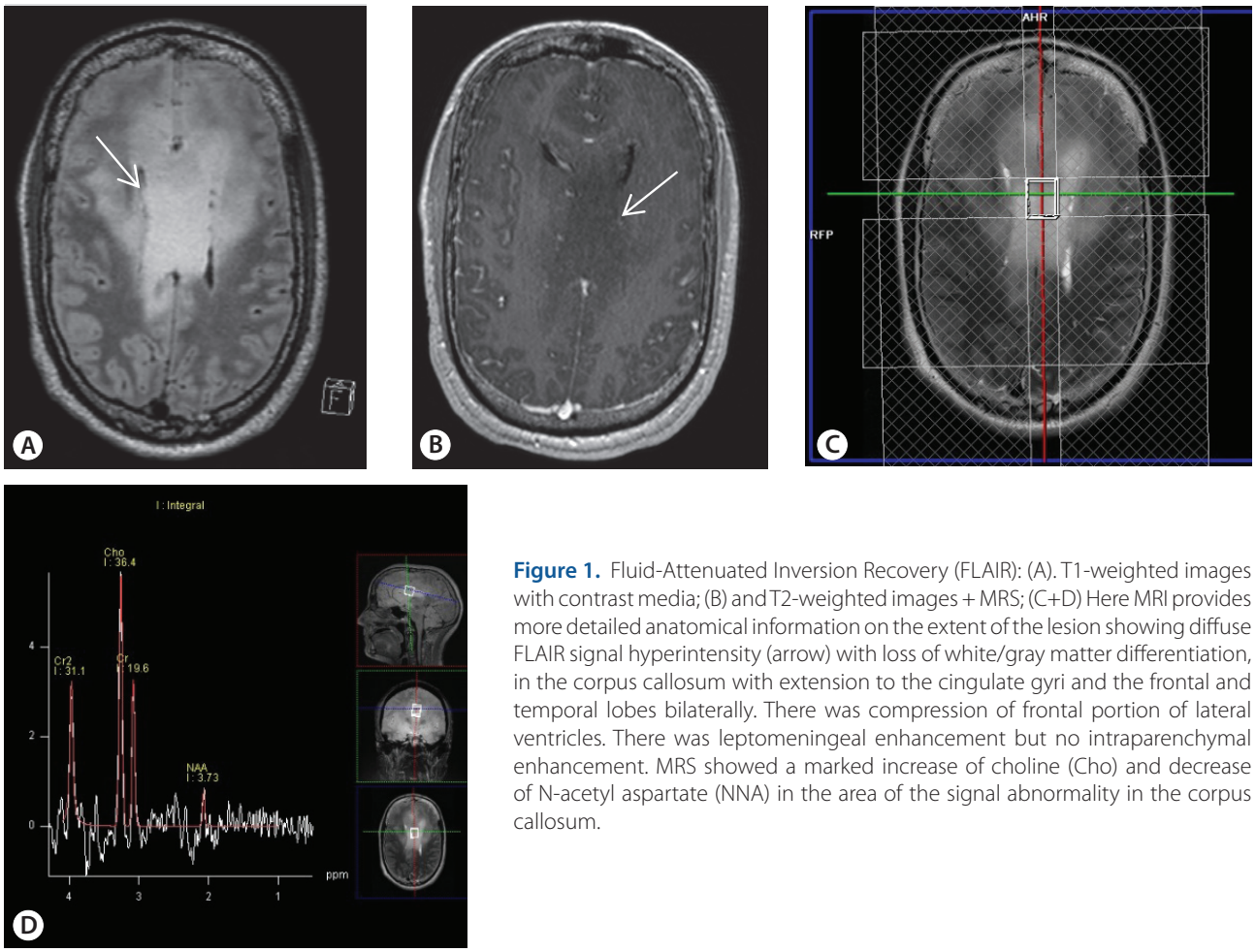


**Figure 1.** Fluid-Attenuated Inversion Recovery (FLAIR): (A). T1-weighted images with contrast media; (B) and T2-weighted images + MRS; (C+D) Here MRI provides more detailed anatomical information on the extent of the lesion showing diffuse FLAIR signal hyperintensity (arrow) with loss of white/gray matter differentiation, in the corpus callosum with extension to the cingulate gyri and the frontal and temporal lobes bilaterally. There was compression of frontal portion of lateral ventricles. There was leptomeningeal enhancement but no intraparenchymal enhancement. MRS showed a marked increase of choline (Cho) and decrease of N-acetyl aspartate (NNA) in the area of the signal abnormality in the corpus callosum.

of sudden onset headache two months before, lasting only few minutes. The clinical examination was also unremarkable.

Computed tomography examination showed a hypodense space occupying lesion in the splenium of the corpus callosum with perifocal edema without contrast enhancement. A neoplastic process was suspected and the patient was referred for further diagnostic work-up with MRI and Positron Emission Tomography–Computed Tomography (PET-CT).

MRI showed a space occupying lesion in the splenium of the corpus callosum with infiltration of the forceps major and extension, along the peritrygonal region of the left ventricle, in the thalamus and in the temporal white matter. There was no evidence of contrast enhancement at this time.

PET-CT with 18F-Fluoro-Ethyl-Tyrosine (18F-FET), revealed weak Fluoro-Ethyl-Tyrosine FET uptake in the known lesion with a hotspot in the area of the left thalamus. Computed tomography of the thorax and abdomen revealed no other lesions. Lumbar puncture and CSF cytology showed normal protein and albumen content and no evidence of malignant cells.

Biopsy was obtained from the splenium and pulvinar thalami [hotspot in PET-CT] and showed no evidence of tumor cells. Two weeks after admission the patient was discharged with 4 weeks follow up MRI and anti-epileptic treatment.

Series of follow up MRIs showed no change in the extent and the morphology of the lesion. One year later, a new MRI (Fig. 2) showed progression in the extent of the lesion and new contrast enhancement in the left temporal pole. The diagnosis of GC was raised by the neuroradiologist and a new biopsy was performed in the contrast-enhancing part of the lesion in the temporal region. Histology revealed the diagnosis

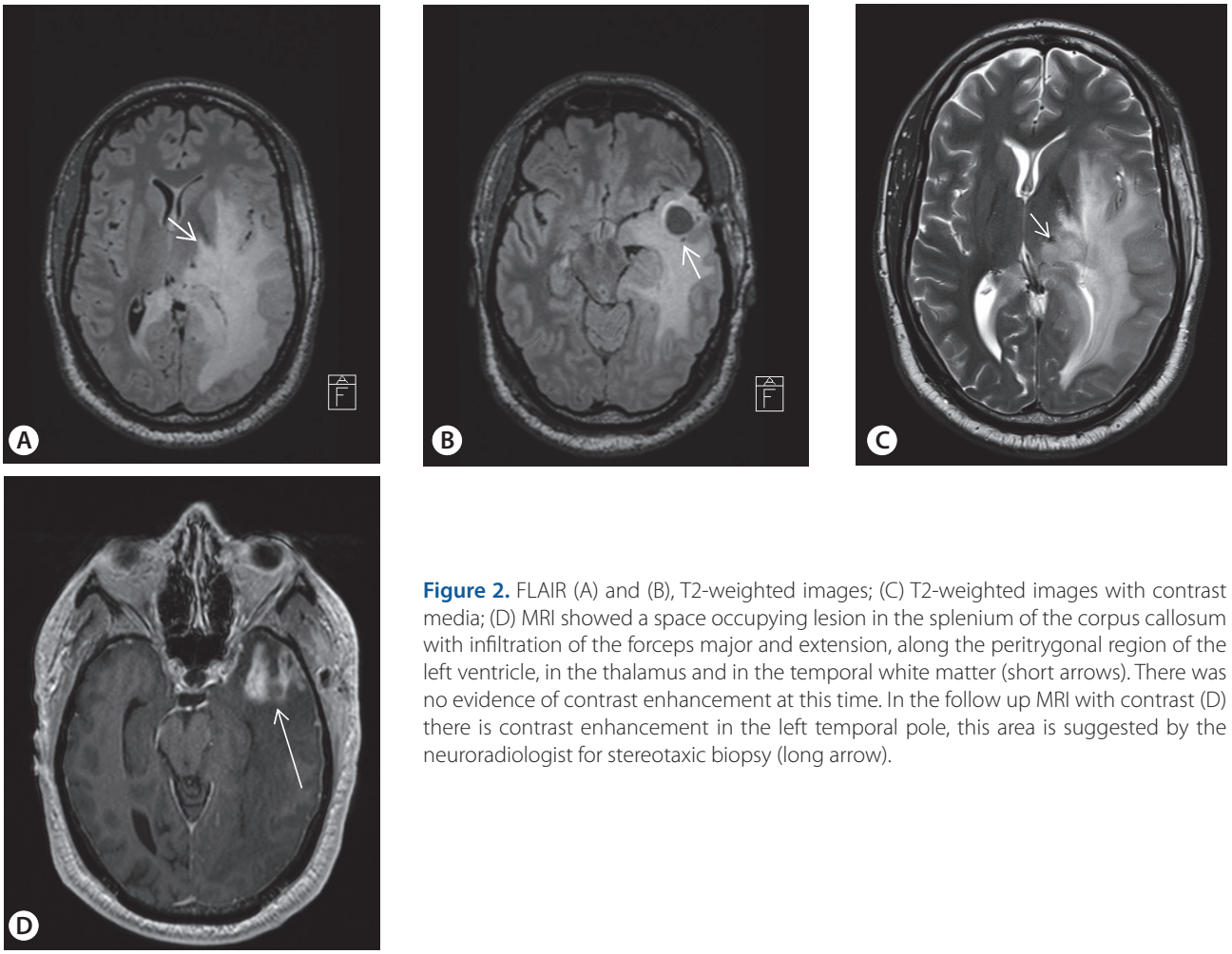


**Figure 2.** FLAIR (A) and (B), T2-weighted images; (C) T2-weighted images with contrast media; (D) MRI showed a space occupying lesion in the splenium of the corpus callosum with infiltration of the forceps major and extension, along the peritrygonal region of the left ventricle, in the thalamus and in the temporal white matter (short arrows). There was no evidence of contrast enhancement at this time. In the follow up MRI with contrast (D) there is contrast enhancement in the left temporal pole, this area is suggested by the neuroradiologist for stereotaxic biopsy (long arrow).

of anaplastic astrocytoma WHO grade III. Based on the histology results and the extensive involvement of several brain areas on MRI the diagnosis of GC was established.

## Case 3

A 55 year old male patient presented with personality change, loss of concentration and memory disturbance since four months. Since two months he complained also of recurrent vertigo and disorientation with bifrontal headache associated with vomiting. He was referred for brain MRI, which showed a diffuse, infiltrating process with FLAIR/T2-hyperintense signal, involving the posterior corpus callosum, the thalami and the parahippocampus bilaterally and extending in the temporal white matter and caudally in the mesencephalon. There was no enhancement in the T1-weighted post-contrast images. Magnetic resonance spectroscopy (Fig. 3) showed decreased N-acetyl aspartate (NAA) with minimal increase of Choline (Cho). A subsequent PET-CT revealed minimal FET-uptake in the supra- and infratentorial areas of MR signal abnormalities. All imaging finding were suggestive of GC as the most likely diagnosis. Lumber puncture (LP) was negative for malignant cells. Biopsy was obtained and histological examination revealed a high number of cells with increased amount of proliferation. Negative IDH-Mutation was not supportive for a low grade glioma. Follow up MRI (Fig. 3) showed a new

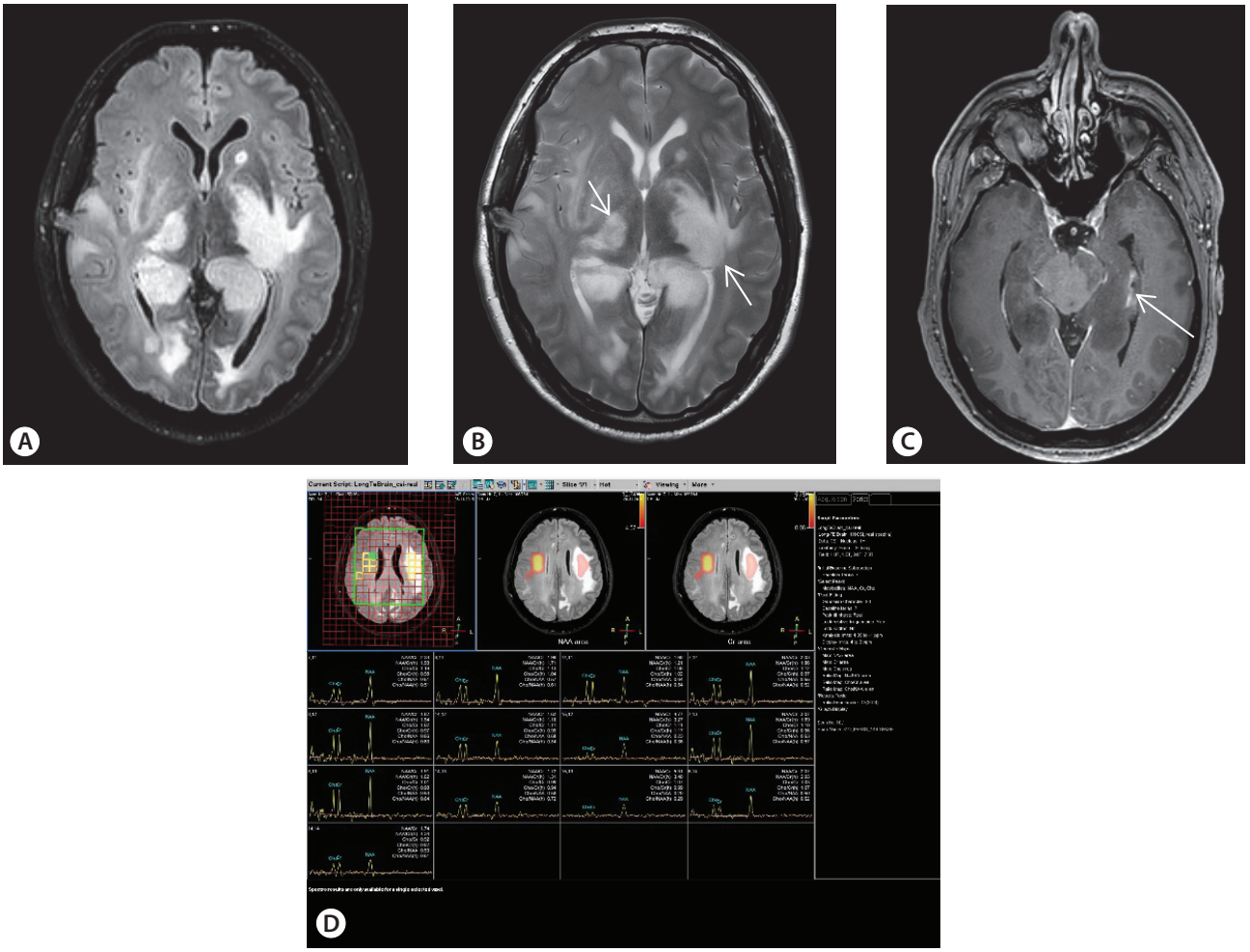


**Figure 3.** FLAIR (A), T2 (B), T1 with contrast media (C), and MRS (D) showed a diffuse, infiltrating process with FLAIR/T2-hyperintense signal, involving the posterior corpus callosum, the thalami and the parahippocampus bilaterally and extending in the temporal white matter and caudally in the mesencephalon (short arrows). MRS showed decreased NAA with minimal increase of Choline. Follow up MRI (C) showed a new nodular contrast enhancement in the occipital horn of the lateral ventricle left (long arrow).

nodular contrast enhancement in the occipital horn of the left lateral ventricle and in the crus anterior of the left internal capsule, further supporting the diagnosis of a malignant process. Finally another biopsy was done and the final histological report showed astrocytoma with WHO grade II, which supported the diagnosis of GC.

## Discussion

Gliomatosis cerebri is a diffuse cerebral infiltration of neoplastic glial cells that preserves the architecture of the brain tissue. Gliomatosis cerebri was first described by Nevin[3] in 1938 in three cases, depending on histopathological criteria and postmortem examination. In the literature, the longest GC series was published by Taillibert *et al.*[8] in 2006 and included 296 patients from the database registration and from literature. In another series, Jennings *et al.*, reviewed 160 cases of GC reported in the literature[3]. Armstrong *et al.*[9], reported 13 cases of GC in pediatric population.

In the WHO classification two types of macroscopic GC have been recognized. Type one, is the classical description of a diffuse tumor growth pattern and enlargement of the involved brain area, there is no formation of a well-defined or well-circumscribed space occupying lesion at the first presentation of the patient. Type Two, which might also be a progression of Type One, is associated with the presence of a clear tumor mass in addition to the diffuse neoplastic infiltration at the initial presentation[1,5]. Histologically type one is usually classified as a WHO grade III. Following the presence of a focal mass (type two), it has the characteristics of a WHO grade IV tumor[10].

The clinical manifestations are unspecific and can be confused with different neurological diseases entities, thus delaying the diagnosis[7]. The neurological deficits are disproportionately insignificant in comparison with the volume and extent of tumor spread on neuroimaging findings, as was the case in our patients. In the literature, the most common symptoms include seizures, intracranial hypertension, mental status change, or focal neurological deficits[8,11,12]. Headache is a common symptom and sudden onset of headaches or longstanding headaches often prompt immediate evaluation [12]. Personality change and impairment of cognitive/behavioral functions, *e.g.,* memory disturbance, and apathy, can also be seen. Hallucination is also reported, some patients complained of dysarthria, aphasia, headache, diplopia, facial palsy, sensory deficit, amenorrhea and subarachnoid hemorrhage[10,11,13]. In some patients papilledema, cranial nerves and brain stem symptoms, corticospinal tract involvement, and dementia were the main clinical symptoms and signs at presentation[14].

## Diagnosis

The diagnosis of GC depends on: 1) In the MRI, presence of a diffuse infiltration abnormality with involvement of three adjoining brain lobes and sometimes presence of extension to infratentorial areas; 2) histopathological analysis shows a glial cell tumor proliferation, either diagnostic of, or consistent with, a diffuse glioma[2]. In our patient this was the method of diagnosis confirmation.

## Radiological Diagnosis

Imaging procedures play an essential part in the diagnosis of GC. Prior to MRI, GC was a postmortem diagnosis. MRI provides accurate antemortem demonstration of the extent of brain structure abnormalities in patients with non-specific clinical presentation. MRI imaging is also the technique of choice in the diagnostic work-up. However, tissue confirmation still remains essential for the definitive diagnosis.

Diagnostic MRI protocol should include FLAIR sequences to assess the tumor infiltration with better precision than conventional T2-weighted images[15]. T1-weighted series performed before and after intravenous contrast administration to assess potential contrast enhancement are also essential. To rule out other entities in the differential diagnosis, *i.e.,* vascular pathology, diffusion-weighted imaging (DWI) should also be included. Although the disease can involve the spinal cord, spinal imaging is not routinely performed.

In the literature including our patients, MRI demonstrates a diffuse, ill defined, FLAIR/T2-weighted hyperintense signal in the area of the disease infiltration as the main morphological abnormality. It shows a diffuse and contiguous growth character in the brain structure that involves the white matter of at least three lobes of the brain[10]. A poor differentiation between the gray and white matter is also observed when there is cortical involvement. Considering the diffuse areas of signal changes, there is only minimal mass effect[16], and relatively minor distortion of the involving brain areas[14]. Although the cortical and the subcortical areas can both be effected. However, corpus callosum and the central white matter areas are

commonly involved[14]. On T1-weighted MR imaging, GC is usually seen as an ill-defined, iso- to hypointense abnormality. The majority of cases show no contrast enhancement, dependent on the type of GC[17]. That means no contrast enhancement in the classical type one GC, as in case 1, but usually there is enhancement in the type two, as in case 2 and later in case 3. The focal enhancing lesions may be related to areas of anaplastic changes and are more likely to have higher grade tumor components [13,18], which helps for biopsy planning as in cases 2 and 3. The increase in the DWI signal reflects T2-shine through effect especially if the ADC is not decreased[10]. However, in our first case there was a DWI restriction associated with low ADC value, which reflects hypercellularity pattern.

In general CT is not adequate to evaluate patients with GC. CT scan is either normal or shows areas of minimal hypodensity with little or no mass effect on the ventricle system. Nevertheless, these findings are important to recognize in the acute setting and prompt further work-up with MRI [19,20]. Presence of hemorrhage and calcifications on CT examination will be important for differential diagnosis. The study done by Freund *et al.*[21] showed that MRI is superior for the diagnosis of diffuse infiltrating CNS tumors. This is because of its high contrast differentiation between normal and pathologic tissue changes, the increase in soft tissue delineation, and its capability to make different multiplanar images. The laboratory results are only helpful to rule out infectious or inflammatory diseases.

For definitive diagnosis brain biopsy should be performed[22]. However, GC should be suspected when a patient presenting with seizure or mental status changes shows a lesion with diffuse growth pattern and involvement of at least three contiguous cerebral lobes on FLAIR MRI sequences[10,15]. MRI with biopsy and histological analysis are considered the gold standard for the final diagnosis[2,10]. Furthermore, MRI is the procedure of choice for patient follow-up.

## Magnetic Resonance Spectroscopy Finding

Magnetic resonance spectroscopy (MRS) shows a non-invasive biochemical screening of normal and pathologic brain tissue, which is based generally on measuring the concentrations of the choline (Cho), N-acetyl aspartate (NAA) and Creatine (Cr) brain metabolites and on the presence of lipid and lactate. High Cho and low NAA as well as high Cho/Cr and Cho/NAA ratios indicate malignant progression of a brain neoplasia. Low NAA, which is a major neuronal marker, reflects replacement of neuron cells by glial tumor cells. High Cho derives from the choline containing component of the cell membrane, which reflects the increase of membrane turnover in tumor cells.

Magnetic Resonance Spectroscopy (MRS) changes observed in GC reflect prominent tumor cell proliferation, permanent neuronal destruction and tumor infestation. All GC patients from the literature that had MRS examinations showed high Cho/Cr and Cho/NAA ratios as well as low NAA/Cr ratios of varying degrees in the region of hyperintensity change shown by the FLAIR sequence[16,22,23]. Anaplastic lesions show increased Cho/NAA ratio, markedly higher Cho/Cr and a lower NAA/Cr ratio. Lactate, a marker of anaerobic metabolism, as well as increased lipids were also present in these last cases. Presence of those metabolites is not only indicative of malignant lesion but also a dire prognostic factor[14,16,24].

## Pathological Diagnosis

For tumor grading and differentiation, the pathologists look for the amount of rod cells, presence of multinucleated tumor giant cells, mitosis, necrosis, endothelial proliferation, intranuclear inclusions as well as, nuclear pleomorphism, microcytic changes, perivascular lymphocytic cuffing, cellular density, calcification and some genetic markers[25].

Histologically, in the review of Bendszus *et al.*[16] and Park *et al.*[25] and depending on the density of abnormal cell, cellular pleomorphism, mitosis number, and proliferative characteristics, GC was grouped into two histologic grades. Cytological anaplasia, like multinucleated giant tumor cells, proliferation of endothelial cells, or mitotic cell changes were more common in type two. Type two shows anaplastic high grade GC[25]. Intranuclear inclusion, microcystic change, and perivascular lymphocytic cuffing was more commonly found in type one. The review of Vates GE *et al.*[13], demonstrated also that patients with space occupying lesion were more likely to possess higher grade tumor process. Although GC type one usually shows radiological and pathological morphology of a low grade tumor, the overall biologic character matches to at least a grade III of highly malignant lesion according to WHO classification[1,3].

Classically, most GC cases exhibit an astrocytic tumor type but oligodendroglioma and mixed oligoastrocytoma can also be present[3]. Our first and second cases were histologically grade III astrocytoma.

While the third case was grade II astrocytoma. Histologically and radiologically, white matter brain area is more commonly involved[14,26].

Bendszus *et al.*[16], Peretti-Viton *et al.*[18], Kararizou *et al.*[23], and Park *et al.*[25] described their histopathological findings that the cortical structure and adjacent white matter tissue showed a diffuse moderately dense infiltration of elongated tumor cell with minimal or no damage of the parenchyma. Mitosis changes were infrequent. Tumor giant cells were the most commonly observed. Park *et al.*[25] mentioned also that the most distinctive cellular components of the GC were rod cells of various morphologies. Furthermore, the normal brain tissue was invaded by glial tumor cells that frequently look like astrocytes, with oval or fusiform and often hyperchromatic nuclei [25]. There may be destruction of myelin sheath, but there is no evidence of necrosis. Similar to these findings, our case revealed a tumor tissue with moderate cell density and pleomorphic cells as well as mitotic figures. The atypical cells were rounded-oval and found in the glial matrix. No evidence of necrosis or vessel proliferation was present.

## Clinical and Radiological Differential Diagnosis

Clinical symptoms in GC are nonspecific. A large number of different diseases present with the same clinical symptomatology. On imaging, disease processes that may show extensive involvement of the brain parenchyma include vascular disease, infectious processes, pseudotumor cerebri and diffuse demyelinating diseases such as leukoencephalopahy or multiple sclerosis. Tumors like diffuse astrocytoma or multicentric glioblastoma multiform must be taken in mind as the differential diagnosis[5,21]. Consider the two most important features of GC namely, infiltration at least three cerebral lobes and the preservation of the brain tissue architecture despite the extent of the tumor involvement, should raise suspicion of the diagnosis. Histology remains essential for the definite diagnoses[13].

## Treatment and Prognosis:

Treatment options for GC include radiation and, more recently, chemotherapy. Surgery other than biopsy is usually not an option. Rare occasions may allow craniotomy and focal space occupying lesion resection at the time of initial surgical treatment[6,8,13]. The study by Taillibert *et al.*[8], conveyed that patients treated by chemotherapy, accompanied or not with radiation treatment, showed better outcomes than patients who did not take chemotherapy. The most frequently used regimen is presently Temozolomide. A more recent review by Desclée *et al.*[10] and Levin *et al.*[27] mentions that the combination of extensive tumor infiltration with minimal CNS destruction makes an initial chemotherapy approach particularly attractive. However, it should be noted that if chemotherapy fails radiotherapy is still a treatment choice for these candidates[10,27,28].

The prognosis of GC is generally poor. In the analysis of their data, Vates *et al.*[10] proposed some factors which may favorably affect the disease prognosis such as no contrast enhancement on MRI scans, lower tumor grade on histological examination and treatment versus no treatment.

## Conclusion

Gliomatosis cerebri was initially a postmortem diagnosis. Following the advancement of the MRI, it became a radiological diagnosis that induces diffuse involvement of at least three contiguous cerebral lobes with relative preservation of its general anatomical configuration. It is manifested clinically as a very aggressive disease with poor prognosis. Imaging plays an important role in initial diagnosis, biopsy planning and in the follow up.

## Acknowledgment

The author thanks Professor Spyridon Kollias for comments and critical review of the manuscript.

## Conflict of Interest

The author has no conflict of interest.

## Disclosure

The author did not received any type of commercial support either in forms of compensation or financial for this study. The author has no financial interest in any of the products or devices, or drugs mentioned in this article.

## Ethical Approval

Obtained.

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# تكثر التدبق المخي: تقرير و وصف لثلاث حالات مرضية مع مراجعات للدراسات السابقة

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***المستخلص***. تكثر الدبق المخي هو ورم نادر مع توقعات سيئة لسير هذا المرض، وذلك نظرا لتسلله وانتشاره في الجهاز العصبي المركزي للإنسان في ما لا يقل عن ثلاثة من فصوص الدماغ. لهذا المرض اعراض لا نوعية تسبب صعوبة في تشخيصه مثل الصرع، الاضطرابات الإدراكية، الصداع، واضطرابات الشخصية.

هذه الدراسة تقدم وصف تفصيلي لثلاث حالات مرضية تم تشخيصها بهذا المرض كما تمت مراجعة دراسات واسعة النطاق لهذا المرض. وهذه الدراسة تشمل تعريف هذا المرض وأنواعه، اعراضه السريرية، وطرق تشخيصيه بواسطة الاشعة التشخيصية وعلم الانسجة السريرية، بالإضافة الى مقارنته بالأمراض المشابهة له، و طرق علاجه والتنبؤ لسير هذا المرض .

ولتنويه كان هذا المرض يشخص بداية عن طريق عينة حيوية بعد الوفاة. لكن بعد التطور والتقدم في التصوير الطبي بالرنين المغناطيسي، أصبح هذا المرض يشخص بواسطة اشعة الرنين المغناطيسي للمخ ويتم تأكيد التشخيص عن طريق فحص عينة الانسجة الحيوية للمريض [الخزعة]

وختاما ان من اهم مميزات هذا المرض انتشاره وتسلله فيما لا يقل عن ثلاثة فصوص متجاورة من الدماغ مع الحفاظ النسبي على الشكل التشريحي العام له. علاوة على ذلك يتجلى هذا المرض سريريا بطريقة عدوانية جدا مع تخمين سيء لمسيرته الإكلينيكية، كما ان التصوير الطبي يلعب دورا هاما في التشخيص الأولي، والتخطيط لعمل خزعة ولمتابعة المسار والتقدم العلاجي للمريض