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CASE REPORT

# Rare case of glioblastoma multiforme located in posterior corpus callosum presenting with depressive symptoms and visual memory deficits

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## SUMMARY

Most of the primary brain tumours are located in the supratentorial region, and it is uncommon to see tumour growth on deep brain structures such as posterior corpus callosum (PCC). In addition, lesions in PCC are also difficult to recognise, because construction apraxia, visuospatial perception and attentional capacity impairment may be the only presenting symptoms. Here, we represent a rare case of gliobastoma multiforme located in PCC, which solely presents with depressive symptoms and visual memory deficits. Initial manifestations of primary brain tumours with psychiatric symptoms and memory disturbances, in addition to headaches and seizures, should be kept in mind.

## BACKGROUND

Primary brain tumours mainly present with neurological symptoms like headaches, seizures, motor and sensory deficits, and their presentation with mood symptoms are relatively scarce.[1] Most of the primary brain tumours are located supratentorial, and their location in deep brain structures such as posterior corpus callosum (PCC) are uncommon, except for metastasis and stroke.[2 3] Lesions in PCC are also difficult to recognise because construction apraxia, visuospatial perception and attentional capacity impairment may be the only presenting symptoms.[2] Here, we report a rare case of gliobastoma multiforme (GBM) located in PCC and presenting with depressive symptoms and visual memory deficits.

## CASE PRESENTATION

A 46-year-old woman presented with fatigue, avolition and anhedonia for the past 6 months. She describes her premorbid personality as an outgoing, quick tempered and energetic; however, she reported that she has been more introvert and calm in the past few months despite a significant stressful trigger or an obvious cause. She stated that she was feeling depressed and has had difficulty in concentrating at her work. She did not report any suicidal thoughts or a change in her appetite. She suffered from psychomotor retardation and increased sleep. In the past month prior to her presentation, the patient and her family members noted that she was more forgetful about recent events and she was having difficulty in making decisions to the level of impairing her functionality. She did not report any delusions or hallucinations. Her psychiatric history revealed that she had depressive symptoms 2 years ago for which she received antidepressant treatment for 8 weeks and stopped her medications on her own due to dissipation of her symptoms. She reported that her depressive mood at the first episode was related to a stressor at social life and was resolved easily in 2 months. She denied history of seizures or headaches and a family history of psychiatric disorders. Her neurological examination was normal.

## INVESTIGATIONS

Her neuropsychological test evaluation revealed that her forward digit span was 5 and backward digit span was 3. Verbal memory evaluation showed she could learn 8 of the 15 words after the 10th repeat and she could remember 7 of them spontaneously and 8 of them by definition after 30 min. In Wechsler memory scale visual reproduction subtest, she scored 4 points on immediate recall, 0 points in 40 min delayed recall test, respectively. However, she scored 49 points on facial recognition test score, which was considered normal. These findings were suggestive of primary verbal and visual memory impairment.

Gadolinium-enhanced MRI revealed a peripheral enhancing well-circumscribed cystic mass with a solid enhancing component located in the splenium of corpus callosum (figure 1A–C), in addition to a large amount of associated oedema in the adjacent periventricular white matter (figure 1D). MR spectroscopy revealed decreased levels of N-acetylaspartate and elevation of the choline-to-creatine ratio indicated an aggressive neoplastic process, and an elevated lipid-lactate peak suggested necrosis. Diffusion tensor imaging showed almost total disruption of white matter tracts within the splenium of corpus callosum.

## OUTCOME AND FOLLOW-UP

Since the tumour was stiff and strictly attached to the deep venous structures, only internal decompression and biopsy were performed via interhemispheric transcallosal approach. Histopathological examination revealed GBM. The patient was discharged without any major neurological deficits on postoperative day 5.

## DISCUSSION

Even though this case experienced depressive symptoms, which impaired her functionality for a few months, an underlying cause of brain pathology was not suspected until memory disturbances

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**Figure 1** T1W and axial FLAIR images of glioblastoma multiforme located in posterior corpus callosum. (A–C) Axial, coronal and sagittal postgadolinium T1W images show peripheral enhancing well-circumscribed cystic mass with a solid enhancing component in posterior corpus callosum. (D) Axial FLAIR image represents severe associated oedema in the adjacent periventricular white matter. FLAIR, fluid-attenuated inversion recovery; TIW, T1-weighted image.

occurred. The extent of the neurological deficit could only be detected by neuropsychological test battery. Depressive symptoms may reach up to 50%[4–6] among the patients with a primary brain tumour. However, to the best of our knowledge, depressive symptoms before the diagnosis of the tumour has not been reported except by D'Angelo *et al*,[7] who reported that 9.7% of patients with a primary brain tumour were diagnosed with depression before surgery. Depending on its localisation, a tumour may affect functional connections of this specific region,[8] it may change the diffuse brain modulatory systems or it may influence hypothalamus pathways and lead to vegetative symptoms and depression. Tumours may also secrete cytokines,[9] which may cause depression. Depression may be the final common pathway for several different aetiologies, and suspecting a tumour in a case of depressive symptoms is not common until another neurological deficit is recognised. In addition, the symptoms of tumour may overlap with depressive symptoms, and thinking of the tumour in differential diagnosis may not be a priority.

We believe that the first depressive episode of this patient was not related to the current depressive symptoms and GBM localisation, since it was triggered by a major stressor, was quickly resolved within 2 months of antidepressant treatment and the patient was completely symptom-free until the past 6 months. However, the depressive episode during our examination bore differences compared to the previous one; depressive symptoms started without a significant trigger, her symptoms increased gradually and visual memory deficits were added on. These characteristics point to a tumour emergence, progression and mental state change in a 6-month period. On the other hand, the aetiology of depressive symptoms is multifactorial and since this patient had another episode in the past, the depressive symptoms concurrently observed with the tumour may not be directly linked to the tumours' presentation and localisation. Since the tumour could not be resected totally, we could not observe if the depressive symptoms would subside after resection of the tumour.

This case also has an unusual location and cystic formation of GBM. Although, the cystic mass seen in MRI slides was not a common presentation for GBM, MR spectroscopy findings and pathology report confirmed the GBM diagnosis. Gliomas are commonly located in the cerebral lobes, mainly frontal and temporal areas, and rarely in the ventricules.[10 11] There are a few studies that show PCC as the origin of GBM.[12] Therefore, the location of GBM in this case is a rare presentation. In addition, most of the GBM cases are reported to be non-cystic.[13–15] Cystic GBM has an unknown exact prevalence and unclear mechanism of formation. Among the several hypotheses that try to explain the underlying mechanisms are necrotic degeneration, central haemorrhage and entrapment of cerebrospinal fluid or local disruption of the blood–brain barrier.[16] Earlier studies suggested the formation of a cyst within GBM to be a favourable prognostic factor and to be related to higher survival and lower recurrence rates,[17 18] However, in 2011 Kaur *et al*[19] reported that primary cystic GBM was not associated with better overall survival according to the largest series of cases.

The role of PCC in mood regulation and memory functions is also not well known. Previous studies have reported decreased splenium white matter,[20] and decreased age-related volume increase in schizophrenia,[21 22] and morphological disturbances[23] and reduced area[24–26] in bipolar disorder. However, two studies reported that splenium volume was not related to depression.[27 28] Tissue loss of PCC has also been linked to lower mini mental test scores and to self-perceived memory impairment.[29] Our case is in accordance with the literature for the presentation of visual memory disturbance after disrupted PCC tracts and it also adds to the knowledge about depressive symptoms. However, other depressogenic effects of tumours, such as changing functional connections, secreting cytokines or changing the function of hypothalamic pathways, cannot be excluded in our case.

In conclusion, primary brain tumours' presentation with psychiatric symptoms and memory disturbances, in addition to headaches and seizures should be kept on mind. PCC might affect the mood and it could also rarely be the origin of a GBM. The structural connections of PCC with other limbic and memory-related areas should be investigated in further studies to replicate our observation and hypothesis.

## Learning points

- Posterior corpus callosum may rarely be the primary location for glioblastoma multiforme.
- A cystic mass, which may resemble an infection in MRI, observed in posterior corpus callosum may be the representation of glioblastoma multiforme.
- Visual memory deficits, which can be recognised with detailed neuropsychological tests, could be related to posterior corpus callosum pathologies and it should be assessed in patients with brain tumours.

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**Contributors** HYE performed the patient's psychiatric interview and also collateral interview with the family for additional information, wrote the first draft of the article and is responsible for the overall content. AO has evaluated the radiological images and contributed mainly to the discussion of the GBM localisation and its cystic formation. ÖÖ-Ç performed the neurological examination and ENVY has applied the neuropsychiatric test battery. EE and IS have evaluated the patient from the neurosurgical point of view and performed the neurosurgery. All authors contributed significantly to the discussion of the patient and revising of the first draft. All authors approved the final article and assured all the questions regarding the accuracy of the article.

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