Case report

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Large haemorrhage within glioblastoma mimicking haemorrhagic stroke and coexistance of meningioma: a case of collision tumours

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Abstract

Intracranial collision tumours are rare pathologies in which two distinct neoplasms are found in the same location. We present an unusual case of an intracranial collision tumour composed of meningothelial meningioma (CNS WHO G1) and glioblastoma (IDH-wildtype, CNS WHO G4). This collision tumour was found in a 64-year-old man. This patient was hospitalized urgently due to left-sided hemiparesis. The computed tomography (CT) revealed large multilobar intracranial haemorrhage located in the right hemisphere. The history of hypertension and obesity pointed to the misdiagnosis of a typical haemorrhagic stroke. Despite extensive physiotherapy after initial improvement, the magnetic resonance imaging (MRI) showed signs of a marginal contrast enhancement with a suspicion of a brain tumour. Moreover, the meningioma in the same location was suspected. The neuropathological findings confirmed two neoplasms with fragments of the dura mater infiltrated by malignant glioma cells and small nests of meningothelial cells with psammoma bodies. The presented case is extremely rare showing that more malignant tumour may infiltrate a meningioma. Moreover, this case highlights the clinical observation that glioblastoma may mimic a haemorrhagic stroke. In such cases when pharmacological treatment is not effective, suspicions should be raised about a possible underlying brain tumour.

Key words: *glioblastoma, meningioma, intracerebral haemorrhage, collision tumour.*

Introduction

Glioblastoma (GB) presents classically with symptoms of raised intracranial pressure and gradual progressive neurological deficits [29]. In rare cases, however, an acute presentation, with intracerebral haemorrhage and rapid clinical deterioration may occur [5,9,10,28]. Contemporary imaging modalities mainly computed tomography (CT) do not reliably reflect underlying mass lesions in parenchymal large brain haemorrhage at first presentation [10]. The history of hypertension, obesity and cardiac insufficiency with a typical presentation of a large haemorrhagic stroke may delay the final diagnosis. The progressive neurological deficits despite extensive physiotherapy in patients after intracerebral haemorrhage may indicate other pathology mainly an underlying tumour [9,10]. In such cases MRI with contrast enhancement is warranted. In patients presenting with intracerebral haemorrhage, the incidence of previously undiagnosed brain tumours has been estimated to be 0.6% [28]. Moreover the MRI may reveal the presence of the coexistence of different histologic tumours

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at adjacent sites, so called collision tumours [24]. Collision tumours consisting of meningioma and GB in a patient without prior radiation therapy or family tumour history are extremely rare. In the literature, less than 20 cases of such collision tumours have been reported [1,3,6,11-13,17,18,20,21,23-27,30-32]. The pathogenesis of concurrent or collision brain tumours remains unknown [25].

We present here the case of a 64-year-old man without a previous history of malignancy, radiation therapy, trauma who had an unusual presentation of two different types of intracranial brain lesions – a meningothelial meningioma (CNS WHO grade 1) and GB (IDH-wildtype, CNS WHO G4) [14]. Interestingly, the meningioma was infiltrated by GB cells in the neuropathological specimen. To our knowledge, this is the first case of a haemorrhage within GB and concomitant meningioma.

Case report

A 64-year-old male was referred to the Neurological Department due to speech impairment and left-sided hemiparesis. These symptoms although mild were present a few days before admission. Urgently performed computed tomography (CT) revealed the massive haemorrhagic stroke in the right hemisphere, localized mainly in the temporal and frontal lobes as well in the right basal ganglia (Fig. 1A). The angio-CT was done to exclude a ruptured middle cerebral artery aneurysm. The intracranial hematoma caused significant perilesional cerebral oedema with mass effect producing the midline shift to the left by 5 mm. The patient had a history of hypertension, cardiac insufficiency, obesity and alcohol dependence syndrome. He had been drinking alcohol for a couple of months before the neurological symptom's onset. There was no head trauma history.

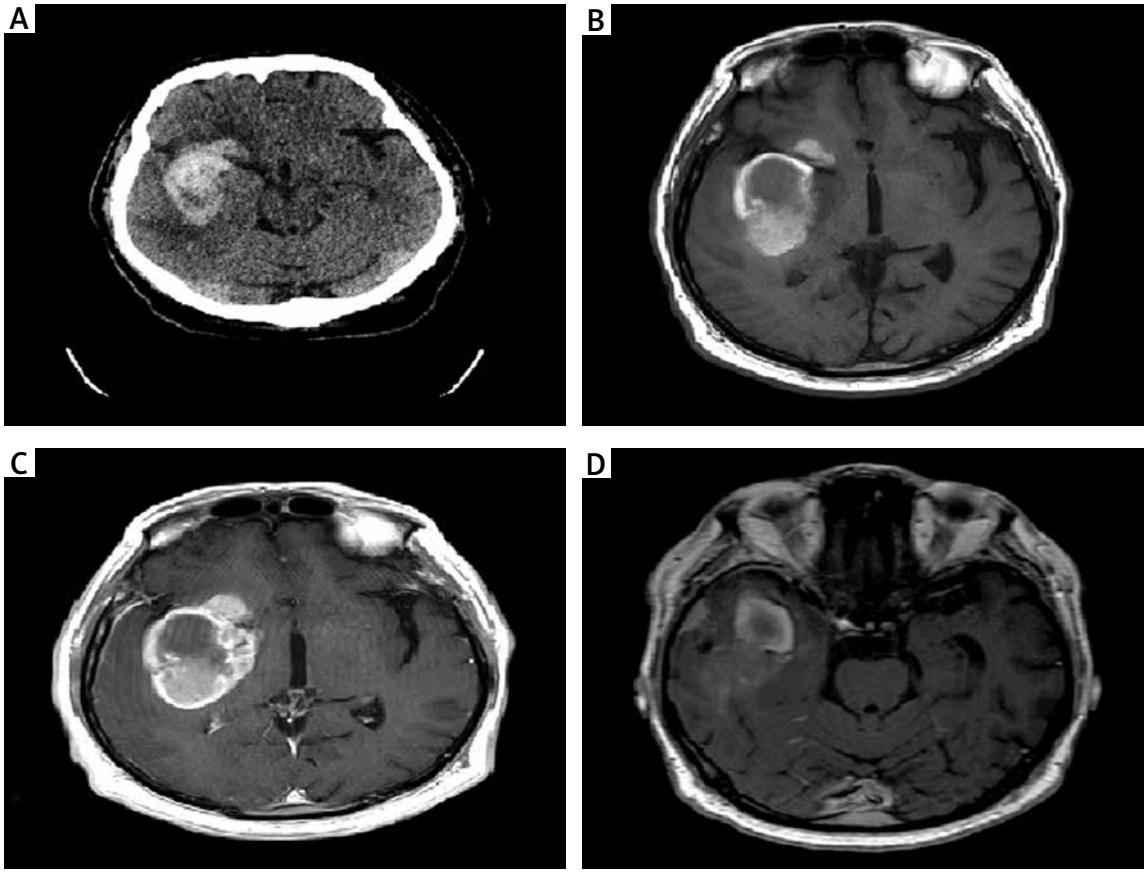

Fig. 1. The computed tomography (CT) revealed the massive intracranial haemorrhage in the right hemisphere, localized mainly in the temporal and frontal lobes (**A**). The magnetic resonance imaging (MRI) without contrast showed a heterogeneous lesion in the right hemisphere (**B**). The MRI with intravenous contrast showed signs of peripheral contrast enhancement with intratumoral haemorrhage (**C**). Additionally, there was a homogeneous and well-circumscribed mass between the right temporal lobe and a skull with a broad dural base and strong enhancement indicating a small meningioma located in the right temporal region (**D**).

The alcohol withdrawal syndrome was diagnosed in this patient. Additionally, a sore on the coccyx was observed.

The first clinical diagnosis was haemorrhagic stroke. During the hospitalization the patient underwent a physiotherapy course with an improvement in his clinical state, especially in left limbs' strength. The patient was able to sit on the bed as well as to walk for a short distance with the help of the physiotherapists.

Consequently, the magnetic resonance imaging (MRI) was done showing a heterogeneous lesion in the right hemisphere (Fig. 1B). The contrast enhanced MRI sequences showed signs of haemorrhage within the lesion as well as peripheral enhancement (Fig. 1C). This lesion exerted the mass effect causing still midline shift to the left by 5 mm. The MRI examination was done 3 weeks after the first CT and suspicion of a brain tumour with intratumoral haemorrhage was raised.

Additionally, there was a homogeneous and well-circumscribed mass between the right temporal lobe and a skull with a broad dural base and strong enhancement indicating a meningioma located in the right temporal region (Fig. 1D). Based on neuroimaging examination collision tumours (double pathology) were suspected: malignant glioma with intratumoral haemorrhage accompanied by a meningioma. The patient was consulted by a neurosurgeon and the surgical one-stage excision of both tumours was proposed to the patient. Thereafter, the patient was transferred to the Neurosurgical Department. A day before the planned neurosurgical procedure a repeated MRI examination was done. The MRI revealed the growth of the tumour indicating the presence of the malignant glioma (Fig. 2A). The intravenous administration of the contrast agent showed the typical enhancement for primary brain tumour (Fig. 2B) producing a larger mass effect than in the previous

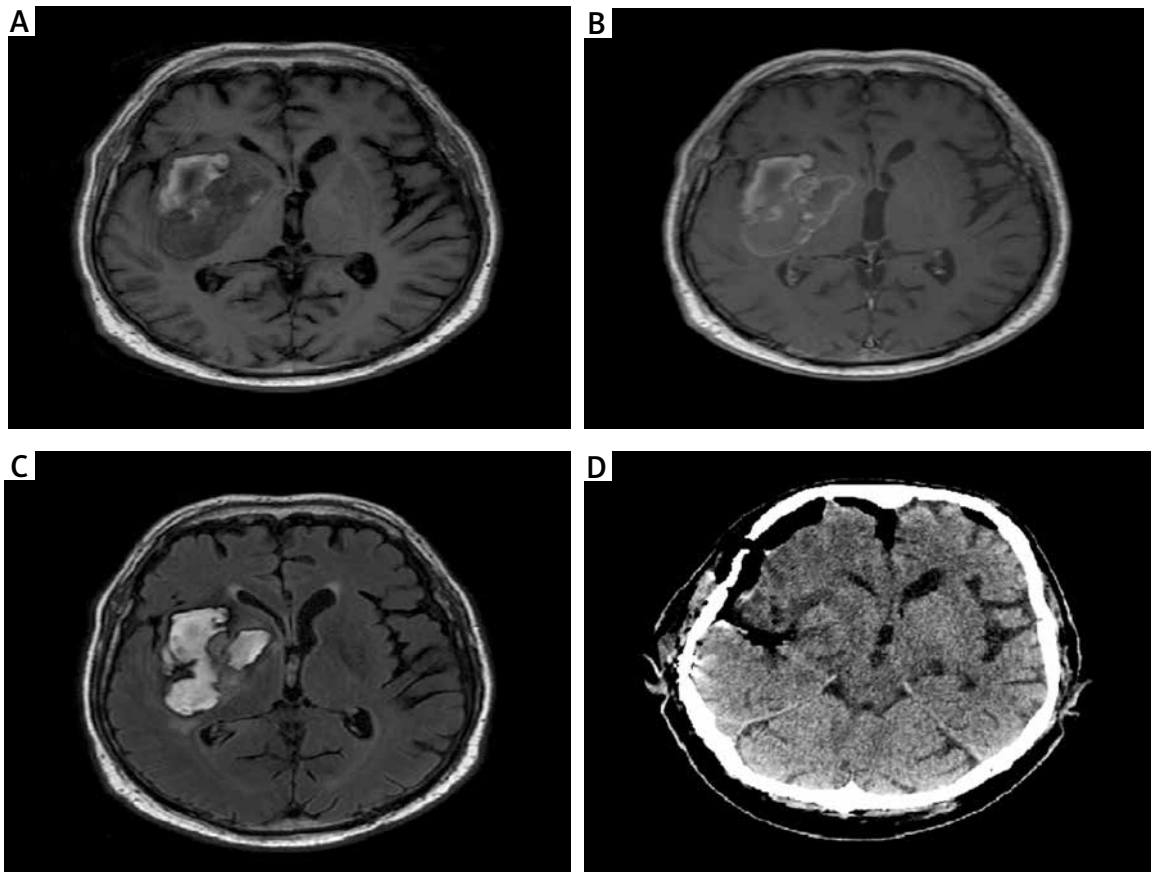

Fig. 2. The MRI examination revealed the growth of the tumour indicating the presence of the malignant glioma (**A**). The intravenous administration of the contrast agent showed the typical enhancement for primary brain tumour (**B**, suspicion of malignant glioma) producing a larger mass effect that in the previous MRI examination. The MRI flair sequence showed still the presence of intratumoral haemorrhages in different stages of absorption (**C**). The postoperative CT showed a typical appearance after removal of both pathologies (**D**).

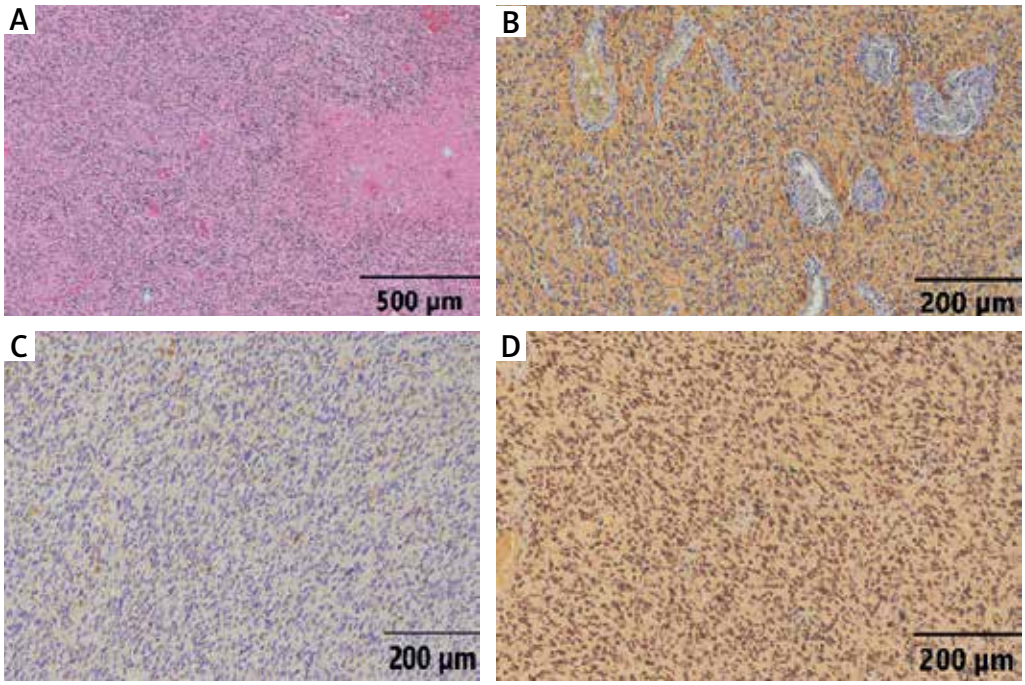

Fig. 3. Histological evaluation of the lesion located in the right subcortical parieto-temporal region. Hematoxylin and eosin (H&E) staining showed diffuse infiltration of high-grade glioma with microvascular proliferation and foci of necrosis (**A**). GFAP immunostaining revealed strong cytoplasmic expression in the neoplastic cells (**B**). Negative immunostaining for IDH1 R132H (**C**) and retained nuclear immunoreactivity for ATRX (**D**) within tumour cell nuclei consistent with the diagnosis of glioblastoma IDH-wildtype, CNS WHO grade 4.

MRI examination. The MRI flair sequence showed still the presence of intratumoral haemorrhages in different stages of absorption (Fig. 2C).

After signing the informed consent, the patient underwent standard right fronto-temporal craniotomy. During surgery, the dura with a typical appearance of meningioma was totally removed (Simpson grade 1). Thereafter, the intraparenchymal tumour was approached through the middle temporal gyrus. The intraoperative appearance suggested the presence of a cavernous hemangioma. The lesion had septum with greenish tint. There were no areas of necrosis or the presence of an excessive number of pathological vessels. The lesion had clear boundaries in relation to the surrounding gliosis. The lesion could be gross totally excised. Removal of the tumour was easy due to its relatively low vascularity. The postoperative CT showed a typical postoperative appearance (Fig. 2D). The postoperative course of the patient in the Neurosurgical Department was uneventful. Both neuropathological specimens were sent for further examination. The patient was transferred back to the Neurological Department for further physiotherapy. Interestingly, although the neurological symptoms completely subsided, the patient became apathetic and uncooperative during rehabilitation. Additionally, the sore of his coccyx complicated his rehabilitation program.

The neuropathological examination revealed indeed two tumours. The lesion located subcortically in the right temporal, frontal and parietal lobes revealed diffuse infiltration of malignant astrocytic glioma with a nuclear pleomorphism, microvascular proliferation, foci of necrosis and haemorrhages. Neoplastic cells showed positive immunostaining for GFAP, Olig2, ATRX and negative for IDH1 R132H mutation (Fig. 3A-D). Ki67 immunolabeling index was about 40%. The diagnosis of GB, IDH-wildtype, CNS WHO G4 was established according to the current guidelines (Central Nervous System Tumours, WHO Classification of Tumours, 5th Edition, 2021) [14]. The second lesion, taken from the dura mater and removed completely, showed fragments of the dura mater with malignant glioma infiltration and small nests of neoplasmic hyperplasia of meningothelial cells with psammoma bodies (Fig. 4A-D). Meningothelial cells demonstrated positive immunostaining for vimentin and EMA, and negative for GFAP (Fig. 4C, D). Pathological findings were indicative for the diagnosis of meningothelial meningioma, CNS WHO G1. No fur-

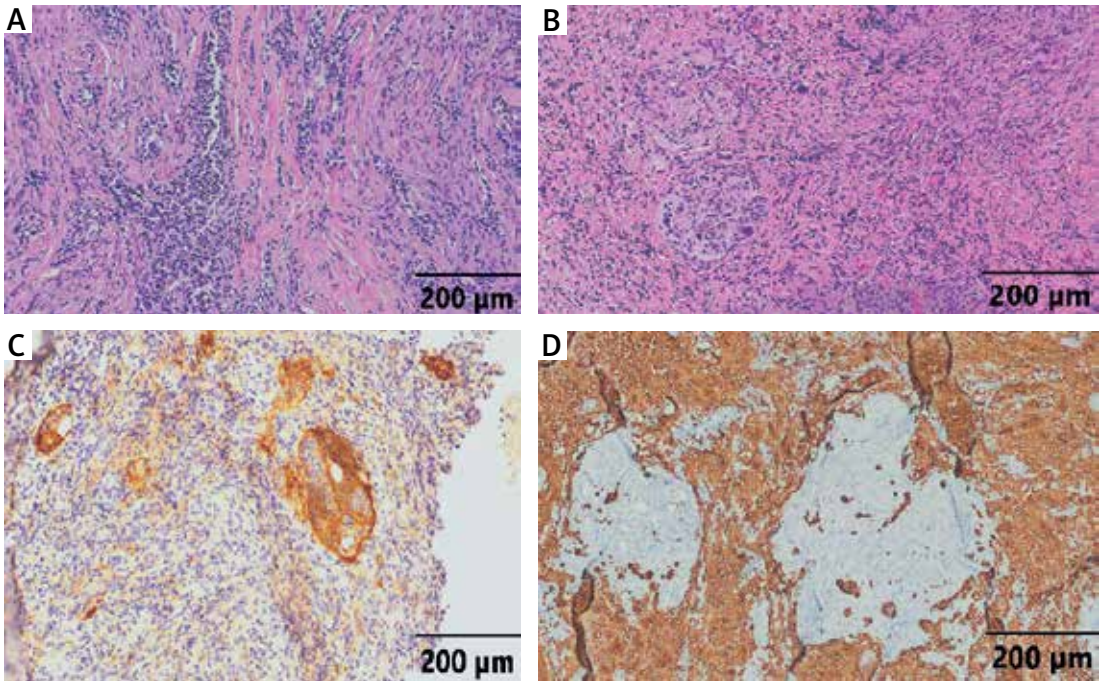

Fig. 4. Histological evaluation of the lesion located in the dura mater above the right temporal lobe. H&E staining revealed dural infiltration of the high-grade astrocytic glioma (**A**). Lobules of neoplasmic meningothelial cells with single psammoma bodies surrounded by malignant glioma (H&E) (**B**). Immunohistochemistry demonstrated positive immunoexpression of EMA (**C**) and negative immunoexpression of GFAP (**D**) in meningioma cells.

ther molecular analysis of both tumours was done in the presented case.

After obtaining the neuropathological examination, the neuro-oncology consultation took place. The patient, due to his uncooperativeness and ongoing surgical treatment of the large sore over his coccyx, was not classified for further radiation and chemotherapy. Despite repeated physiotherapy and surgical management of his sore in the area of his coccyx the patient became weaker and ceased walking or sitting on the bed. Finally, the patient was discharged from the Neurological Department and transferred to a home hospice. The patient passed away 2 months after the onset of first neurological symptoms.

Discussion

Glioblastoma is the most common primary malignant brain tumour in adults and meningioma is the most common benign brain tumour but the coincidence so called "collision" of these two distinct types of tumour occurrence in the same location is extremely rare [2,13,15,24]. The term "collision" has been used for the cases in which two tumours are being intermixed or appear close to each other. Gliomas and meningiomas are the most common frequently reported combination of collision tumours in adults [1,3,4,6,11-13,17,18,20,21,23-27,31,32]. In the literature, less than 20 cases of such collision tumours were reported [1,3,6,11,13,17,18,21,23-27,31,32]. Table I summarizes the clinical characteristics of collision tumours consisting of a meningioma and high-grade glioma. In our case, GB presented clinically as an haemorrhagic stroke causing left-sided hemiparesis and dysarthria.

The natural history of disease progression, and the exact mechanism of collision brain tumours are not elucidated [4-6,13,16,19,20,25,26,30,31]. Some authors indicate that collision brain tumours are coincidental [13]. Other researchers suggest that the initial tumour can trigger development of the other tumour [5,13,27]. Therefore, there are at least three different hypotheses for the presence of collision tumours [8,26]: 1) the original meningioma induced the oncogenesis of the glioma [26]; 2) the glioma promotes the growth of the meningioma [25]; 3) the two distinct tumours are co-stimulated by each other [8].

As mentioned above intracranial primary collision tumours of different histologic types are extremely rare, and their occurrence is still unclear. Local tissue

Table I. Clinical characteristics of patients operated for collision tumours consisting of meningioma or high-grade glioma (anaplastic astrocytoma or glioblastoma)

Authors, year of publication [reference number]	Age/ Sex	Clinical presentation	Meningioma location	GBM/ anaplastic astrocytoma location	Invasion of both tumours	Surgical management	Follow-up (months)
Lin *et al*., 2022 [13]	56/F	General weakness, dizziness, severe headache	Left frontal CNS WHO grade 2	Left frontal IDH mutant glioblastoma	CTM Invasion of atypical meningioma	One-stage tumour removal	Glioblastoma recurrence at 6 months
Zhang *et al*., 2018 [31]	66/F	Headache	Right parietal	Right parietal	No CTM	One-stage tumour removal	NR
Hasimu *et al*., 2016 [7]	48/F	Generalized epileptic seizure, left hemiparesis	Right parietal lobe	Right temporal lobe	No CTM	Two-staged tumour removal	5 months lost for further follow-up
Hasimu *et al*., 2016 [7]	65/M	Dysphasia, walking instability	Right frontal	Left frontal	No CTM	One-stage tumour removal	8 months – death
Ruiz *et al*., 2015 [21]	86/M	Left hemiparesis	Right frontotemporal	Right frontotemporal	No CTM	One-stage tumour removal	Alive at 15 months after surgery
Khalatbari *et al*., 2010 [11]	12/M	Left lateral ventricle, trigone	Left lateral ventricle, trigone	Headache, seizure, right hemiparesis III astrocytoma	No CTM	One-stage tumour removal	At 22 months, death due to recurrence of malignant glioma
Chen *et al*., 2010 [1]	63/F	Left frontal	Left frontal	Headache, arm weakness	No CTM	One-stage tumour removal	NR
Suzuki *et al*., 2010 [25]	75/F	Left temporal	Left temporal	Headache	CTM	One-stage tumour removal	NR
Mitsos *et al*., 2009 [17]	73/F	Right sphenoid wing	Right temporal	N/A	CTM	Two-stage tumour removal, first meningioma, thereafter glioma.	NR
Nestler *et al*., 2007 [18]	49/M	Disorientation, dysphasia	Left frontal falx	Left frontal	No CTM	One-stage tumour removal	14 months – death
Tugcu *et al*., 2006 [27]	42/M	Right hemiparesis, dysarthria	Left parietal	Left parietal	No CTM	One-stage tumour removal	NR
Drlicek *et al*., 2004 [3]	51/M	Headache, facial palsy	Left frontal	Left frontal	CTM	One-stage tumour removal	NR
Goyal et al., 2003 [4]	72/M	Headache seizure	Right sphenoid wing	Right temporal	No CTM	One-stage tumour removal	N/R
Prayson *et al*., 2002 [20]	87/F	Left hemiparesis	Right frontal lobe	Right frontal lobe Astrocytoma III	CTM	One-stage tumour removal	NR
Spallone *et al*., 1991 [23]	48/M	Headache and confusion	Left cavernous sinus	Left paraventricular Astrocytoma III	No CTM	Stereotactic biopsy	10 months death due to recurrence of anaplastic astrocytoma

Table I. Cont.

Authors, year of publication [reference number]	Age/ Sex	Clinical presentation	Meningioma location	GBM/ anaplastic astrocytoma location	Invasion of both tumours	Surgical management	Follow-up (months)
Vaquero *et al*., 1990 [30]	75/F	Dysphasia and facial dysesthesia	Left parietal lobe	Left parietal lobe	CTM	One-stage tumour removal	2 weeks – death due to pulmonary embolism
Marra *et al*., 1977 [16]	63/M	Left arm paresis	Right parietal lobe	Right parietal lobe	No CTM	One-stage tumour removal	8 months death due to glioblastoma progression
Strong *et al*., 1976 [24]	56/F	Left hemiparesis, headache, gait disturbance	Right frontoparietal falx	Right frontoparietal	No CTM	Two-stage tumour removal	NR
Strong *et al*., 1976 [24]	53/M	Headache, dysphasia, right hemiparesis	Left sphenoid wing	Left temporal lobe	No CTM	One-stage tumour removal	NR
Present case	64/M	Headache, left hemiparesis, dysarthria	Right temporal lobe	Right temporal lobe	CTM Invasion of meningioma by glial cells	One-stage tumour removal	1 month

F – female, M – male, CTM – common tumour matrix, No-CMT – no common tumour matrix, NR – not reported

irritation from the perilesional oedema caused by original meningioma can potentially cause the local tissue irritation leading to astrocyte cells transformation, and consequently neoplastic proliferation [8]. This hypothesis is supported by a case of collision convexity meningioma and glioma reported by Vaquero *et al*. [30]. In this case the transient area between meningioma and GB was observed and two types of tumour cells were mixed in some areas [30]. Other similar cases were presented by Drlicek *et al*. and Prayson *et al*. [3,20]. In both cases meningioma and peripheral GB invaded each other on the histopathological examination [3,20]. Some authors found that especially meningothelial and fibrous meningiomas may affect the growth of the adjacent glioma [15]. It is well known that patients with neurofibromatosis have a predilection to develop various gliomas [15].

Our case showed the invasion of meningioma by GB cells. This situation is explained by the fact that GB as more malignant brain tumour may invade a less invasive tumour such as meningioma. However, collision tumours without histological invasion have also been reported [18,26,27], Nestler *et al*., as well as Tugcu *et al*. and recently Truong *et al*. found that there was no infiltration of meningioma and GB tissue proven in histological examination [18,26,27]. This transformation hypothesis fails to explain why adjacent glial transformation does not occur in most of intracranial meningioma. It also fails to explain the occurrence of two distinct tumours in different brain areas [26].

Other hypotheses suggest a genetic pathway role, including the p53 disruption and receptor tyrosine kinase signalling molecules with an expression of platelet-derived growth factor receptors (PDGFRs) [5,25]. PDGF is one of the growth factors, playing a significant role in angiogenesis and promoting tumorigenesis [25]. PDGF and its three subunits (PDGF-AA, PDFG-BB and PDGF-AB) are secreted by glioblastoma cells [25]. The receptors of PDGFs are also present in meningioma and may stimulate division of meningioma cells [26]. As a result, glioma may stimulate adjacent meningioma formation in arachnoid cells by production of a common growth factor. One such case was presented by Suzuki *et al*. [25]. It was found that both tumours (GB and meningioma) were strongly positive for PDGFR expression seen in immunohistochemistry [25]. However, this hypothesis is not supported by clinical observations that meningioma formation does not happen in most cases of glial tumours.

There are some chromosomal and genetic studies on the subject of coincidence of the different tumours, mainly meningioma and GB but the results are not strictly defined [2,5,14,19,22]. The association of multiple, primary brain tumours has not been definitely supported by genetic results till now and the coincidence may play a significant role [2,14,15]. We cannot

rule out that genetic studies, including next-generation sequencing, will provide new genetic mutations in cases of collision tumours reported in the future. An obstacle is the relatively rare coexistence of collision tumours and the fact that in many countries access to genetic testing including next-generation sequencing is still limited [5,19,22].

Careful preoperative radiological evaluation and appropriate surgical strategy for collision tumours are of great importance in these patients. In our patient, the intratumoral haemorrhage delayed the final diagnosis. As in the other cases reported in the literature, the contrast enhanced MRI remains the diagnostic tool of choice for establishing the diagnosis of collision tumours [2,3,7]. This was the case in our patient that the haemorrhagic stroke was excluded and a suspicion of collision tumours was raised. However, low-density areas surrounding meningioma usually represent peritumoral oedema and exceptionally low-grade astrocytoma [7]. An intraoperative biopsy in doubtful cases should be performed. Another option is a susceptibility-weighted perfusion MR imaging showing hemodynamic differences between two unique tissue types [7]. The presence of meningioma and high-grade glioma including astrocytoma IDH-mutant G 2-3 or GB is easier to ascertain than low-grade astrocytoma in the vicinity of a meningioma. As mentioned above, in our case the haemorrhage obscured the typical appearance of GB on first MR imaging. The repeated MR examination showed still large haemorrhagic changes inside an enlarging invasive tumour.

The neurosurgical removal is usually the treatment of choice for collision tumours. One-stage tumour removal remains the preferred neurosurgical treatment especially when collision tumours are spatially contiguous [6,12,13,21,24-27,31,32]. In most of reported cases, one-stage tumour removal was performed [1,3,7,11, 13,16,18,20,21,24,25,30,31]. In our presented case the meningioma was en route to the large haemorrhagic tumour, and both entities were removed. The meningioma was dissected with dural base achieving Simpson 1 meningioma removal and GB was resected nearly totally leaving a small remnant in the region of basal ganglia. The safe gross total removal is indicated when both tumours produce a marked mass effect. Postoperative CT showed a typical appearance after excision of both tumours with a visible reduced mass effect. The preferred one-stage tumour removal is also advocated by other authors who performed two-stage approach in collision tumours [7]. Hasimu *et al*. found a haemorrhage inside astrocytoma after removal of meningioma in a patient without comorbidities like hypertension or coagulopathy [7]. This means that the causal relationship between surgery and haemorrhage was highly probable [7]. Among all reported cases of collision tumours, in only three patients two-stage tumour removal was undertaken pointing to the fact that one-stage surgery is the preferred surgical approach [7,23,24].

The prognosis of the collision tumours consisting of meningioma and high-grade glioma is unfavourable. In most reported cases the mean life expectancy is less than one year or is not reported [1,3,4,6,11-13,17,18,20-25,27,31,32]. The death is usually related to the recurrence of high-grade glioma. Our patient, although his neurological status improved, was not qualified for further radio and chemotherapy and deceased 1 month after surgery.

Conclusions

Haemorrhagic events in patients with a history of obesity and hypertension point to the diagnosis of a haemorrhagic stroke. The careful examination and close follow-up with MR imaging may suggest the underlying cause of haemorrhage as an GB. MR imaging is the preferred diagnostic option in establishing the radiological diagnosis of collision tumours. Our described case showed the infiltration of meningioma by malignant glial cells. The intensive analysis of any clinical features and neuroimaging signs in MRI may lead to faster diagnosis with potentially better prognosis. When two lesions are closely situated, the best way of surgical management is to remove them in one-stage surgery.

Disclosure

The authors report no conflict of interest.

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