Intracranial Meningioma Manifesting As Transient Ischemic Attack: A Case Report

L C Meguins, G B Sampaio, E Cintra Abib, R F l Hossain Ellakkis, M A Fernandes Teixeira, S R Martucci J, R A Rocha da Cruz Adry, D Freitas de Morais

Citation

L C Meguins, G B Sampaio, E Cintra Abib, R F l Hossain Ellakkis, M A Fernandes Teixeira, S R Martucci J, R A Rocha da Cruz Adry, D Freitas de Morais. *Intracranial Meningioma Manifesting As Transient Ischemic Attack: A Case Report*. The Internet Journal of Neurosurgery. 2013 Volume 9 Number 1.

Abstract

Meningiomas are the most common intracranial primary neoplasm in adults. They present a wide variety of clinical manifestation and interpreting the symptomatology of meningiomas according to their location is one of the most fascinating topics that explore the full potential of the clinical neurologic examination. Epileptic seizures are reported as the very first symptom in most patients. However, cerebrovascular events, particularly transient ischemic attacks (TIA), are uncommon presentation of meningiomas. We describe the case of a patient with an intracranial meningioma manifesting initially as TIA and make a brief review upon the possible explanations of the event.

INTRODUCTION

Meningiomas are neoplasms of meningothelial cells of the arachnoid layer (1). They are estimated to constitute 13% to 26% of all intracranial tumors with a prevalence of 97.5/100,000 in the United States (2-4). Patients with intracranial meningiomas may present a wide variety of symptoms regarding the location of the tumor, such as headache, epileptic seizure and impaired vision (5, 6). However, transient ischemic attack (TIA) is an uncommon presentation of meningiomas (7, 8). We describe the case of a patient with an intracranial meningioma manifesting initially as TIA and make a brief review upon the possible explanations of the event based on current available data.

CASE REPORT

A 56-years-old woman was admitted on the Emergency Department with a complaining of a transitory weakness on the right arm lasting 30 minutes with complete strength recover. Her past medical history was remarkable for diabetes mellitus type 2 on use of subcutaneous insulin three times daily. On clinical assessment of the patient, we found blood pressure 130/90mmHg, puses 80/min and temperature: 37.3oC. The neck was supple without abnormal carotid artery sounds, lungs were clear and the heart rate was regular with no murmurs. Neurological examination was essentially normal. Laboratorial blood investigation, chest radiography and electrocardiogram were within normal range. Brain magnetic resonance image (MRI) showed an extra-axial large mass lesion, with 3.91cmx6.47cm on diameter, compressing the left fronto-parietal lobe and with homogeneous gadolinium enhancement (Figure 1, 2 and 3). No signs of cervical or cranial arterial stenosis or occlusions were identified. As no other structural abnormality was found, a diagnosis of an extra-axial intracranial tumor was made and the symptoms were attributed to it. Based on this diagnosis, the patient underwent surgical removal of the lesion. Complete mass and dural excision was performed without major complications. Histological investigation revealed meningothelial meningioma. The patient presented an uneventful recovery without neurologic deficits and was discharged home in good clinical condition. She is currently being follow-up on outpatient appointments.

Figure 1

Axial T2-Weighted TSE MRI showing an extra-axial large mass lesion compressing the left fronto-parietal lobe.

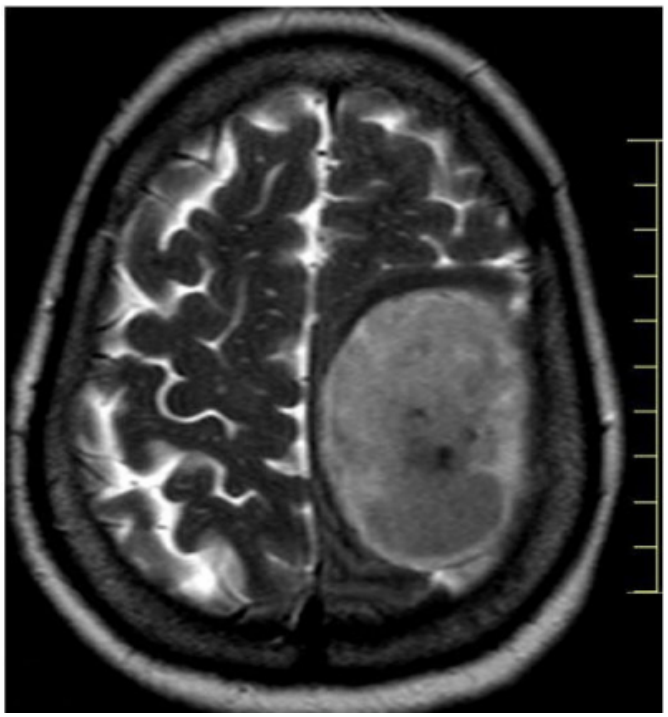


Figure 2

Sagital T1-Weighted MRI showing an extra-axial large mass lesion compressing the left fronto-parietal lobo.

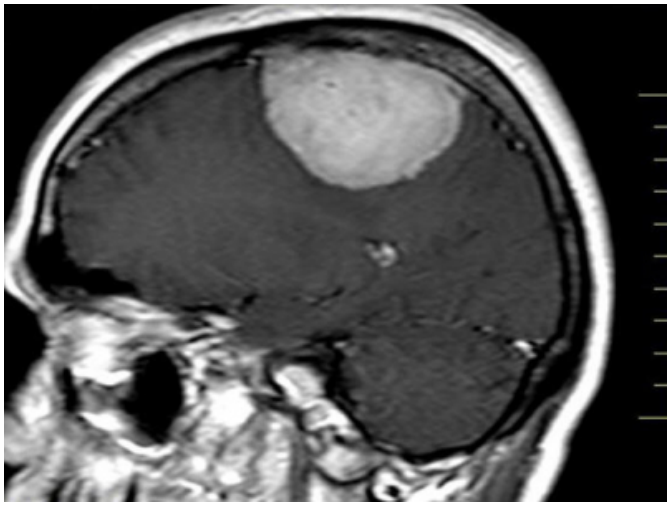
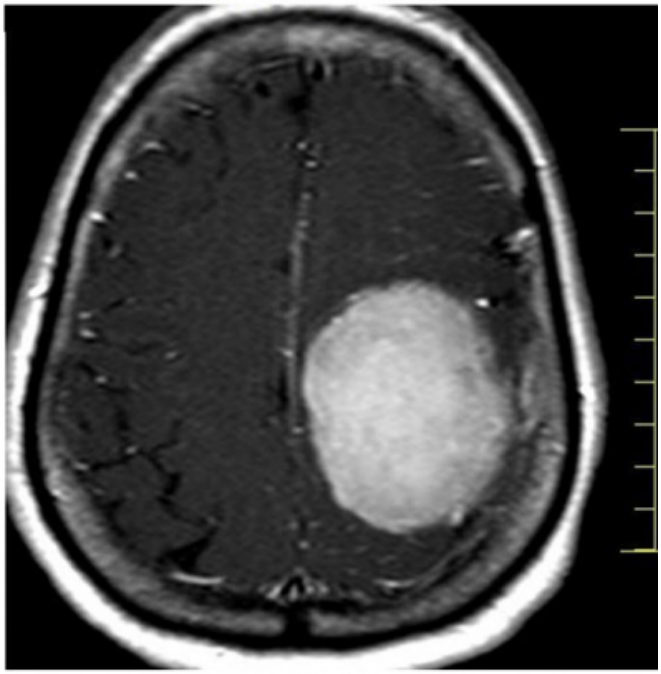


Figure 3

Homogeneous gadolinium-enhance mass lesion compressing the left fronto-parietal lobo.



DISCUSSION

Meningothelial cells tumors are among the most common neoplasms of central nervous system. The majority of cases have a benign clinical course, and some are asymptomatic. Meningiomas are generally well circumscribed, slow-growing lesions that are often amenable to total surgical resection and account for 13% to 26% of all intracranial tumors with a prevalence of 97.5/100,000 in the United States (2-4). They usually manifest as slowly progressive signs of neurological deficits, headache or focal seizures (2). However, transient ischemic attack (TIA) is an uncommon presentation of meningiomas and few is known about the pathophysiological pathways that lead to ischemia (7, 8). According to Kondziolka and colleagues (1988) (8), between all pathologically confirmed cases of brain tumor, only 2.76% of patients had an initial presentation suggestive of occlusive cerebrovascular disease.

A possible explanation for neurologic deficits simulating TIA in intracranial tumors is that the mass causes compression on cerebral blood vessels and edema on the surrounding brain tissue to a degree sufficient to cause partial impairment of cerebral blood flow (CBF) (7, 9). When systemic blood pressure falls or tumor size increases critically the CBF reduces, causing temporary cortical ischemia and paralysis of functions. Whether a readjustment of CBF is possible, the cortical function returns (10).

Additionally, some biological substances, particularly vascular endothelial growth factor (VEGF), produced by cell tumors are known to increase arterial vascular supply. VEGF plays an important role in meningiomas as an angiogenic factor, inducing additional pial supply and corresponding penetration of blood-brain barrier (11). Therefore, it has also been suggested that vascular flow and shunting through the neoplasm could lead to a "steal" phenomenon on adjacent cortex and produce clinical neurological deficits (8, 12). Sawaya and colleagues (1984) (13), hypothesized that certain intracranial tumors may escape host antitumor fibrinolytic activity by producing plasmin inhibitors and that plasmin inhibitors may play a role in the thromboembolic complications frequently seen in patients with intracranial neoplasms. Moreover, hypercoagulability state has been demonstrated in patients with brain tumor (14). Based on this data, it seems reasonable to believe that some patients with meningioma may present thromboembolic episodes causing reduction of CBF and neurological disfunction. However, more studies are still needed to ensure this possibility.

CONCLUSION

In conclusion, the present report reinforces the need for a careful clinical and neuro-radiological investigation of patients who present with symptoms of TIA to ensure that such potentially treatable lesions are not missed. Meningiomas are a possible cause of TIA and must be remembered and evaluated in patients manifesting transitory neurologic deficits.

References

1. Hadidy AM, Nadi MM, Ahmad TM, Al-Hussaini MA, Al-Abaddi AA, Musharbash AF, Maani WS. Descriptive epidemiological analysis, MRI signals intensity and histopathological correlations of meningiomas. Neurosciences (Riyadh). 2010 Jan;15(1):11-4.
2. Marosi C, Hassler M, Roessler K, Reni M, Sant M, Mazza E, Vecht C. Meningioma. Crit Rev Oncol Hematol. 2008 Aug;67(2):153-71.
3. Wiemels J, Wrensch M, Claus EB. Epidemiology and etiology of meningioma. J Neurooncol. 2010 Sep;99(3):307-14.
4. Campbell BA, Jhamb A, Maguire JA, Toyota B, Ma R. Meningiomas in 2009: controversies and future challenges. Am J Clin Oncol. 2009 Feb;32(1):73-85.
5. Rohringer M, Sutherland GR, Louw DF, Sima AA. Incidence and clinicopathological features of meningioma. J Neurosurg. 1989 Nov;71(5 Pt 1):665-72.
6. Solero CL, Giombini S, Morello G. Suprasellar and olfactory meningiomas. Report on a series of 153 personal cases. Acta Neurochir (Wien). 1983;67(3-4):181-94.
7. Ueno Y, Tanaka A, Nakayama Y. Transient neurological deficits simulating transient ischemic attacks in a patient with meningioma--case report. Neurol Med Chir (Tokyo). 1998 Oct;38(10):661-5.
8. Kondziolka D, Bernstein M, Resch L, Tator CH. Brain tumours presenting with tias and strokes. Can Fam Physician. 1988 Feb;34:283-6.
9. Masuoka J, Yoshioka F, Ohgushi H, Kawashima M, Matsushima T. Meningioma manifesting as cerebral infarction. Neurol Med Chir (Tokyo). 2010;50(7):585-7.
10. Launay M, Fredy D, Merland JJ, Bories J. Narrowing and occlusion of arteries by intracranial tumors. Review of the literature and report of 25 cases. Neuroradiology. 1977 Dec 14;14(3):117-26.
11. Schmid S, Aboul-Enein F, Pfisterer W, Birkner T, Stadek C, Knosp E. Vascular endothelial growth factor: the major factor for tumor neovascularization and edema formation in meningioma patients. Neurosurgery. 2010 Dec;67(6):1703-8.
12. Oluigbo CO, Choudhari KA, Flynn P, McConnell RS. Meningioma presenting with transient ischaemic attacks. Br J Neurosurg. 2004 Dec;18(6):635-7.
13. Sawaya R, Cummins CJ, Kornblith PL. Brain tumors and plasmin inhibitors. Neurosurgery. 1984 Dec;15(6):795-800.
14. Weinberg S, Phillips L, Twersky R, Cottrell JE, Braunstein KM. Hypercoagulability in a patient with a brain tumor. Anesthesiology. 1984 Aug;61(2):200-2.

Author Information

Lucas Crociati Meguins
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S
lucascrociati@libero.it

Gustavo Botelho Sampaio
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

Eduardo Cintra Abib
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

Richam Faissal E l Hossain Ellakkis
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

Marco Aur Fernandes Teixeira
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

S Robinson Martucci J
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

Rodrigo Ant Rocha da Cruz Adry
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S

Dionei Freitas de Morais
Department of Neurological Sciences. Hospital de Base da Faculdade de Medicina de S
S