ORIGINAL ARTICLE

Epilepsy in primary intracranial tumors in a neurosurgical hospital in Enugu, South-East Nigeria

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Abstract

Background: Seizures may be manifestation of intracranial tumor (IT) and demand thorough neurological evaluation. This paper examines epidemiology, lesion characteristics and outcome of seizures associated with primary IT.

Methods: Retrospective analysis of medical records, computed tomography and magnetic resonance imaging of patients diagnosed with IT who presented with seizure from 2003 to 2013 at Memfys Hospital for Neurosurgery Enugu. Postoperative seizure outcome was based on Engel classification and correlated with tumor histology, patient age, anatomical location, time of presentation and extent of tumor resection. Data were analyzed using descriptive and inferential statistics.

Results: Sixty-two patients (34.6%) presenting with seizures were analyzed. Peak age at presentation was in 6th decade. Age of seizure onset had bimodal peak at 4th and 6th decades. Apart from IT located in posterior fossa with mortality of 62.5%, postoperative mortality did not depend on anatomical location of tumor. Postoperative seizure outcome and mortality depend on tumor histology (P = 0.025) and preoperative seizure duration (P = 0.036). Seizure duration shorter than 1 month had poor postoperative seizure outcome and high mortality. Although more patients with meningioma experienced seizures compared to glioma (P = 0.025), there was no difference in proportion of patients with meningioma and glioma who presented with seizure (P = 1.00). Extent of resection predicts postoperative seizure outcome based on meningioma sub-group analysis. Overall, 59.7% of patients had good postoperative seizure outcome, 21.0% had poor outcome and 19.3% died.

Conclusion: Seizures of short duration, IT located in posterior fossa and gliomas are associated with poor postoperative seizure outcome and high patient mortality. Tumor histology does not seem to affect seizure predisposition. Most seizures associated with IT occur in fifth and sixth decades of life and affect frontal lobe most often.

Key words: Intracranial tumors, postoperative seizure outcome, seizures

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Introduction

Seizures may be symptomatic of underlying intracranial tumor (IT). This is particularly likely with first seizures in adults and hence in such patients seizures should be regarded as red flags that demand thorough neurological evaluation and neuro-imaging.[1] Although <5% of people with epilepsy have a brain tumor,[2,3] as many as 30–50% of patients with brain tumor present primarily with seizures and another 10–30% develop seizures in the further course of the disease.[4-6] The exact frequency of seizures in central nervous system tumors depends on factors like tumor location, rate of growth and tumor histology.[7] The reported frequency of seizures with meningiomas is about 25%, but further 20% develop seizures after surgery.[8] Younger patients with a longer survival also have a higher risk of seizures.[9]

Epileptogenicity of brain tumors is multi-factorial resulting from the interplay between the microenvironment, genetics, systemic effects, treatment and other external factors.[10]

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The tumor cells are electrically inert, and the seizures are thought to be due to imbalance between neuronal excitation and inhibition occurring at the adjacent cerebral cortex.[11] Tumors presenting with seizures are, therefore, more commonly cortically placed and may be more amenable to surgical treatment. In addition, slow-growing tumors tend to be more epileptogenic than high-grade malignancies,[9] probably because of longer period of evolution and longer survival compared to high-grade tumors.[4,12,13]

The presence of uncontrolled seizure contributes to an increase in morbidity associated with the tumor and even after removal of the tumor, persistence or development of seizures increases morbidity.[14] In many developing countries, epilepsy is still stigmatized. Earlier studies amongst Nigerians showed very high levels of stigmatization to an extent that the epilepsy patient is more likely to lose a job, lose a spouse or find it difficult to marry and may be driven to suicide or vagrancy.[15] In another study of attitudes toward epilepsy in Eastern Nigeria, it was found that patients and their relatives might shy away from seeking early medical attention because of stigmatization.[16] Secondary epilepsy may be less stigmatized when the cause is apparent. In a recent review of patients with meningiomas, it was found that in spite of generally late presentation, patients with epilepsy tended to present earlier.[17] This, however, may be a reflection of the increased awareness that epilepsy is treatable and not indicative of reduction in stigmatization. It is believed that with the better characterization of the underlying cause, these seizures will become less stigmatized and treatment more desired and accepted.

Few reports have addressed the characteristics of seizures associated with brain tumors in Nigeria. The frequency of seizures in association with brain tumors in Nigeria has been reported as 21.9%.[18] This paper examines the epidemiology, clinical and lesion characteristics as well as outcome of seizures associated with primary ITs.

Methods

This is a retrospective analysis of records of patients diagnosed with ITs who presented with seizures as sole or part of their symptoms between 2003 and 2013 at Memfys Hospital for Neurosurgery Enugu. The medical records, computed tomography, and magnetic resonance imaging findings of all patients who had surgery with histological confirmation were used. Cases of ITs with no seizures, idiopathic seizure conditions and seizures due to structural lesions other than tumors were excluded. Only tumors removed at operation that had histological confirmation were studied further. The patients had antiepileptic drugs (AEDs) commenced preoperatively. Most patients received phenytoin or carbamazepine following diagnosis. Newer generation AEDs were used in patients who can afford their long-term use.

The main indications for surgery were intractable epilepsy and other associated symptoms such as raised intracranial pressure and focal deficits. Following operation patients were continued on anticonvulsants and followed for a minimum of 1-year post surgery. Postoperative seizure outcome was grouped based on the Engel epilepsy basic outcome scale. Good seizure outcome was defined as Engel Class 1 (patients free of disabling seizures) while Class II (rare disabling seizures), Class III (worthwhile improvement) to Class IV (no worthwhile improvement) were grouped under poor seizure outcome. Data analysis was based on both descriptive and inferential statistics facilitated by the Statistical Package for Social Sciences (version 17 SPSS Inc, Chicago, IL) software.

Results

Presentation and demography

There were 179 patients with histology confirmed ITs during the study period and 62 (34.6%) presented with seizure as part of their complaints. Only 24% of the patients had focal seizures. A male to female ratio was 1.7:1.0. Peak age at presentation was in the 6th decade of life while the age of seizure onset showed a bimodal peak at the 4th and 6th decades [Table 1 and Figure 1]. There is a tendency for a delay in presentation in the young adult age group [Figure 1].

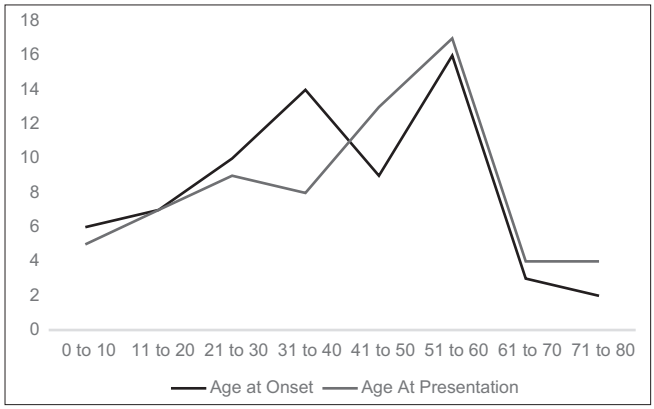

Figure 1: Age at onset of seizures compared to age at presentation

Table 1: Age of patients at time of presentation

Age (years)	Male	Female	Total (%)
0-10	2	3	5 (8.1)
11-20	3	4	7 (11.3)
21-30	6	2	8 (12.9)
31-40	4	3	7 (11.3)
41-50	9	3	12 (19.4)
51-60	9	6	15 (24.2)
61-70	3	1	4 (6.4)
71-80	3	1	4 (6.4)
Total (%)	39 (62.9)	23 (37.1)	62 (100)

Male:female=1.7:1.0

Table 2: Anatomical location of tumor compared with postoperative outcome

Anatomical location	Preoperative frequency (%)	Outcome		
		Postoperative seizure outcome		Mortality
		Good	Poor	Died
Frontal	19 (30.6)	13	5	1
Parietal	10 (16.1)	7	1	2
Temporal	9 (14.5)	4	3	2
Posterior fossa*	8 (13.0)	3	0	5
Sella	5 (8.1)	4	0	1
Pineal*	4 (6.5)	4	0	0
Fronto-parietal	3 (4.8)	2	0	1
Occipital	2 (3.2)	0	2	0
Others†	2 (3.2)	0	2	0
Total (%)	62 (100)	37 (59.7)	13 (21.0)	12 (19.3)

χ^2=6.226, P=0.183>0.05, insignificant. *All tumors located in the pineal region and posterior fossa were associated with hydrocephalus, †Others=Parieto-occipital, fronto-temporal

Table 3: Histology of the tumors and postoperative outcome

Histology	Preoperative frequency (%)	Outcome		
		Postoperative seizure outcome		Mortality
		Good	Poor	Died
Meningioma	31 (50.0)	20	8	3
Glioma	18 (29.0)	6	5	7
Medulloblastoma	3 (4.8)	2	0	1
Pituitary	2 (3.2)	2	0	0
Craniopharyngioma	2 (3.2)	2	0	0
Others*	6 (9.8)	1	0	1
Total (%)	62 (100)	37 (59.7)	13 (21.0)	12 (19.3)

χ^2=11.098, P=0.025<0.05, significant. *Others=Hemangioblastoma, choroid plexus papilloma, germinoma, pineocytoma

Table 4: Relative proportion of seizures between meningioma and glioma

Histology of tumor	Seizure presence		Total
	Present	Absent	
Meningioma			
Count	31	36	67
Percentage	46.3	53.7	100.0
Glioma			
Count	18	21	39
Percentage	46.2	53.8	100.0
Total			
Count	49	57	106
Percentage	46.2	53.8	100.0

χ^2=0.000, P=1.000>0.05, insignificant

Tumor type and location

Frontal area was the most common anatomical location followed by parietal and temporal lobes while posterior fossa lesions accounted for 11.8% and sella tumors, 7.5%.

Table 5: Comparing preoperative duration of seizure and postoperative outcome

Preoperative duration	Number (%)	Postoperative outcome		
		Postoperative seizure outcome		Mortality
		Good	Poor	Died
<1-month	20 (32.3)	9	4	7
1–12 months	19 (30.6)	10	5	4
>1-year	23 (37.1)	18	4	1
Total (%)	62 (100)	37 (59.7)	13 (21.0)	12 (19.3)

χ^2=10.269, P=0.036<0.05, significant

Table 6. Meningioma: Extent of surgery and postoperative outcome

Extent of surgery (meningioma)	Preoperative	Postoperative outcome		
		Postoperative seizure outcome		Mortality
Simpson grade	Frequency (%)	Good	Poor	Died
1	17 (54.8)	12	3	2
2	9 (29.0)	6	3	0
3	2 (6.5)	0	1	1
4	3 (9.7)	1	2	0
5	0	0	0	0
Total	31 (100)	19	9	3

Lesions in the frontal, parietal, sella and pineal regions have a good postoperative seizure outcome. Temporal lobe lesions have poor seizure control while posterior fossa tumors have very poor postoperative outcome with 62.5% of patients dead. Overall, however, the postoperative mortality did not depend on the anatomical location of the tumor (P = 0.183) [Table 2]. There was no hemisphere preference. Meningioma represented 50.0% of cases with 64.5% chance of good outcome. Glioma represented 29.0% and with 33.3% having good outcome and another 38.8% dead (P = 0.025) [Table 3]. Although seizure was experienced in more meningioma cases compared to glioma, there was no significant difference between the proportion of seizures in meningioma and glioma groups [Table 4] (P = 1.00).

Postoperative outcome and duration of seizure

Seizures of <1 month duration had worse postoperative patient survival with 35% dead. It appears that the longer the seizure duration, the better the postoperative seizure outcome (P = 0.036) and the better the postoperative patient survival. Overall, 59.7% of patients had good postoperative seizure outcome, 21.0% had poor outcome and 19.3% died [Table 5]. Of the 54 surviving patients, 40 (74.1%) had good postoperative seizure outcome, with 80% improvement in seizure outcome postoperatively in patients presenting after 1 year. Sub-group analysis of patients with meningioma shows that seizure outcome is related to completeness of tumor removal and location of tumor [Table 6].

Discussion

Presentation

In this series, 34.6% of patients with tumors presented with epilepsy. This is comparable to reports in literature. Only one patient was a known epileptic and in all patients, seizure can be attributed to the presence of the tumor. Although seizures are believed to arise focally in the cerebral cortex adjacent to tumor,[11] only one-quarter of seizures (24%) were clinically focal in our series. Either the patients do not appreciate or place significance on focal components of their seizures or the seizure generalized rapidly. About 33% of patients are known to have a secondary focus distinct from their tumor for their epilepsy.[9] These types of seizures are commoner in younger patients and patients with longer duration of their illness, two conditions that are common in our patients. Morrell and deToledo-Morrell also argue that early treatment of the epilepsy may prevent the development of such secondary focus.[19]

Early presentation is considered a favorable prognostic sign for patients with brain tumor and the presence of seizure as part of clinical presentation may reduce delays in presentation. Regrettably, socio-cultural and religious undertones may make patients with seizures to shy away from seeking early and proper professional diagnosis of the underlying cause. This pattern is changing in Nigeria.[17] In this study, however, significant proportion of patients especially below 50 years still presented late. There is therefore still a need to educate patients about seeking medical advice early, especially in the presence of a red flag like seizure. Patients with seizures should seek treatment early especially in environments without stigma for seizures. It is dangerous to assume that the cause of seizures in an adult presenting with recent onset seizures or a seizure that recently has changed its character is from a nonstructural lesion or metabolic cause for instance. It is important that public orientation even amongst health care workers is expanded in this direction.

Types of tumor

The most common tumor presenting with seizures was meningioma in keeping with the finding that low grade and slow growing tumors are more epileptogenic.[9] The frequency of seizures in our patients with meningioma of 50% is higher than the 25% generally reported. Although this is in keeping with the fact that meningiomas are the commonest primary ITs in our environment, late presentation may also be a factor. The anatomical location of most of the tumors was frontal lobe, and this is also in keeping with other reports.[20] Three patients with medulloblastoma presented with epilepsy. Posterior fossa tumors rarely cause seizures, and we believe that seizure in these patients may be due to associated advanced hydrocephalus.

Surgery

All the patients in this series had surgery, the primary indication being the presence of responsible lesion in a surgically accessible location. Medically refractory epilepsy occurs in 12–50% of patients and is frequently associated with brain tumors and other structural brain lesions.[21] In such patients, seizure control can be achieved in more than 60% with epilepsy surgery.[22] The reasons for refractoriness include loss of receptor sensitivity in some patients with brain tumors, the presence of multidrug-resistance proteins associated (MRP1) with some brain tumors and other nonspecific mechanisms of resistance.[23] Over-expression of multidrug-resistance gene (MDR1) and MRP1 in some glioma tumor cells and areas of cortical dysplasia[24,25] are believed to reduce drug transport into the brain parenchyma.[26] Additionally MDR1 can limit penetration of lipophilic substances like AEDs into the brain. Some commonly used anticonvulsants such as carbamazepine, phenytoin, phenobarbital, lamotrigine and felbamate are substrates for this gene product.[23,27] Although seizure was experienced in more meningioma cases compared to glioma, the relative proportion of seizure between meningioma and glioma based on operated cases is similar - 46.3% and 46.2% respectively.

Outcome

It has been shown that in patients with intractable epilepsy associated with a brain tumor, up to 70–90% improve or become seizure free if surgical excision include total removal of the epileptogenic zone.[28,29] This, however, requires that the epileptogenic zone be excised together with the tumor. This series, limited by its retrospective nature did not give specific attention to the epileptogenic zone, and this may account for the most modest control of seizures achieved. Outcome was better in patients with more extensive surgical removal of their tumors, and this may be an indication of the contribution of pressure on adjacent cortical tissue on epiletogenicity.

Patient presenting with seizures of late onset and shorter duration fared worse following intervention. As much as 61% of the postoperative mortalities in this study had seizure of <2 months duration presenting as the last complaint. The poorer prognosis with late onset seizures can be explained on the basis that high-grade tumors are less epileptogenic than low-grade tumors[9] and thus are more likely to cause late-onset seizure. These patients may have had other clinical evidence of tumor but did not present until seizure disorder forced them. In keeping with this, it has been established that brain tumor patients tend to present late to hospital in Nigeria although in patients with meningiomas the occurrence of seizure may lead to a relatively earlier presentation.[17]

Tumors in the sella and pineal regions appear to have a very good postoperative outcome probably because the underlying mechanism of seizure is related only to the associated hydrocephalus and not the characteristic of the tumor *per se* since the anatomical location of the tumors are not cortical. Development of hydrocephalus may be an indicator of advanced disease and in these cases, early diversion of the cerebrospinal fluid may be all that is needed to control the seizure. On the other hand, both the intrinsic nature of the tumor and the presence of hydrocephalus may explain the poor overall outcome experienced in posterior fossa tumors presenting with hydrocephalus as seen in this study.

Seizure treatment and prophylaxis

For the patient with brain tumor, a single seizure is sufficient to establish a diagnosis of epilepsy, because the risk of repeated seizures is increased.[30] For these patients, long-term anti-convulsant treatment is indicated unless the lesion can be removed completely and does not recur.[10] For patients with brain tumors who present with seizure, anticonvulsants have been shown to limit functional and emotional morbidity.[31]

Even in patients who do not have seizures as part of their presentation we routinely give AEDs. Although there is no clear guidelines supporting seizure prophylaxis for brain tumors,[32] the prophylactic use of AEDs are still common. We have routinely used carbamazepine and phenytoin for both treatment and prophylaxis in tumor patients to limit cost, but this practice is changing. Prophylactic use of phenytoin has not been shown to be effective in randomized studies of patients with intracerebral tumors.[33,34] Also phenytoin interacts in a complex manner with dexamethasone. Dexamethasone is often used to control peri-tumoral edema, and concurrent use with phenytoin shortens the half-life and the activity of dexamethasone.[35] Dexamethasone on the other hand may increase (by competition for protein binding) or decrease (by interference with hepatic metabolism) the blood concentration of phenytoin.[36] Concurrent use of both drugs, therefore, requires that phenytoin concentrations should be monitored closely, particularly during withdrawal of dexamethasone, to avoid toxic levels of phenytoin.[35]

Rossetti and Stupp[10] following a review of AED in current use suggest that pending prospective studies, levetiracetam and/or pregabalin should be used for the treatment of seizures in brain tumor patients. They suggest that newer generation AED should be given for brain tumor patients undergoing surgery until approximately 1 week, and that long-term prophylaxis is not required. In developing countries where cost considerations are important, the use of traditional AED is still a standard practice and among these valproic acid is considered to have the best pharmacokinetic profile.[10]

Socio-cultural

The occurrence of seizure in a patient represents a difficult physical and emotional challenge and diagnosis of epilepsy still carries significant stigmatization in Nigeria.[16,17] This study suggests that patients with seizures should seek treatment early irrespective of socio-cultural attitudes as early presentation results in a more favorable prognosis for both seizure and tumor control. Regrettably, a lot of socio-cultural and religious undertones make patients with seizures to shy away from seeking early and proper professional diagnosis of the underlying cause. Targeted education of the public and, in particular, first line health providers are likely to change practice and needs to be encouraged.

Neurosurgery in Nigeria is still evolving and as such, local research that would highlight the relationship between IT and seizure conditions in our locality and form the basis for public orientation are needed. This study should provide such a basis.

Conclusion

Seizures of recent onset especially in an adult patient should be properly investigated as intracranial tumors, especially meningiomas, are frequently the underlying cause in this environment. However, tumor histology does not seem to primarily affect tumor predisposition. The extent of tumor resection has a positive relationship with postoperative seizure outcome. Seizures of short duration, posterior fossa tumors presenting with seizure and gliomas predict worse postoperative patient survival. Temporal lobe tumors have poor seizure outcome. Most seizures occur in the fifth and sixth decades of life and affect the frontal lobe most often. It is, therefore, dangerous to assume that the cause of seizures in a patient presenting with recent onset seizures especially in adults or a seizure that recently has changed its character is caused by a nonstructural lesion. It is important that public orientation even in the health circle is expanded in this direction.

References

1. Naymee JV, Joshua PK. Neuro-imaging in the evaluation of epilepsy. Semin Neurol 2012;32:361-73.
2. Olafsson E, Ludvigsson P, Gudmundsson G, Hesdorffer D, Kjartansson O, Hauser WA. Incidence of unprovoked seizures and epilepsy in Iceland and assessment of the epilepsy syndrome classification: A prospective study. Lancet Neurol 2005;4:627-34.
3. Bromfield EB. Epilepsy in patients with brain tumors and other cancers. Rev Neurol Dis 2004;1 Suppl 1:S27-33.
4. Herman ST. Epilepsy after brain insult: Targeting epileptogenesis. Neurology 2002;59:S21-6.
5. Wen PY, Marks PW. Medical management of patients with brain tumors. Curr Opin Oncol 2002;14:299-307.
6. Glantz MJ, Cole BF, Forsyth PA, Recht LD, Wen PY, Chamberlain MC, *et al.* Practice parameter: Anticonvulsant prophylaxis in patients with newly diagnosed brain tumors. Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2000;54:1886-93.
7. Hauser WA, Annegers JF, Kurland LT. Incidence of epilepsy and unprovoked

seizures in Rochester, Minnesota: 1935-1984. Epilepsia 1993;34:453-68.
8. Lieu AS, Howng SL. Intracranial meningiomas and epilepsy: Incidence, prognosis and influencing factors. Epilepsy Res 2000;38:45-52.
9. van Breemen MS, Wilms EB, Vecht CJ. Epilepsy in patients with brain tumours: Epidemiology, mechanisms, and management. Lancet Neurol 2007;6:421-30.
10. Rossetti AO, Stupp R. Epilepsy in brain tumor patients. Curr Opin Neurol 2010;23:603-9.
11. Wolf HK, Roos D, Blümcke I, Pietsch T, Wiestler OD. Perilesional neurochemical changes in focal epilepsies. Acta Neuropathol 1996;91:376-84.
12. Cascino GD. Epilepsy and brain tumors: Implications for treatment. Epilepsia 1990;31 Suppl 3:S37-44.
13. Riva M. Brain tumoral epilepsy: A review. Neurol Sci 2005;26 Suppl 1:S40-2.
14. Taphoorn MJ. Neurocognitive sequelae in the treatment of low-grade gliomas. Semin Oncol 2003;30:45-8.
15. Osuntokun BO. Epilepsy in the developing countries. The Nigerian profile. Epilepsia 1972;13:107-11.
16. Mezue WC, Mezue AA. Early detection of attitudes towards epilepsy. A rational basis for positive attitudinal change. Orient J Med 1989;1:30-3.
17. Ogungbo B, Finkel MF, Ogun Y. Building foundations for improving health opportunities in sub-Saharan Africa. Neurology 2013;81:2134-5.
18. Owolabi LF, Akinyemi RO, Owolabi MO, Sani UM, Ogunniyi A. Epilepsy Profile in Adult Nigerians with late onset epilepsy secondary to brain tumor. Neurol Asia 2013;18:23-7.
19. Morrell F, de Toledo-Morrell L. From mirror focus to secondary epileptogenesis in man: An historical review. Adv Neurol 1999;81:11-23.
20. Sirven JI, Wingerchuk DM, Drazkowski JF, Lyons MK, Zimmerman RS. Seizure prophylaxis in patients with brain tumors: A meta-analysis. Mayo Clin Proc 2004;79:1489-94.
21. Sisodiya SM, Lin WR, Harding BN, Squier MV, Thom M. Drug resistance in epilepsy: Expression of drug resistance proteins in common causes of refractory epilepsy. Brain 2002;125:22-31.
22. Britton JW, Cascino GD, Sharbrough FW, Kelly PJ. Low-grade glial neoplasms and intractable partial epilepsy: Efficacy of surgical treatment. Epilepsia 1994;35:1130-5.
23. Löscher W, Potschka H. Role of multidrug transporters in pharmacoresistance to antiepileptic drugs. J Pharmacol Exp Ther 2002;301:7-14.
24. Aronica E, Gorter JA, Jansen GH, van Veelen CW, van Rijen PC, Leenstra S, *et al.* Expression and cellular distribution of multidrug transporter proteins in two major causes of medically intractable epilepsy: Focal cortical dysplasia and glioneuronal tumors. Neuroscience 2003;118:417-29.
25. Aronica E, Gorter JA, Redeker S, van Vliet EA, Ramkema M, Scheffer GL, *et al.* Localization of breast cancer resistance protein (BCRP) in microvessel endothelium of human control and epileptic brain. Epilepsia 2005;46:849-57.
26. Calatozzolo C, Gelati M, Ciusani E, Sciacca FL, Pollo B, Cajola L, *et al.* Expression of drug resistance proteins Pgp, MRP1, MRP3, MRP5 and GST-pi in human glioma. J Neurooncol 2005;74:113-21.
27. Rogawski MA. Does P-glycoprotein play a role in pharmacoresistance to antiepileptic drugs? Epilepsy Behav 2002;3:493-95.
28. Luyken C, Blümcke I, Fimmers R, Urbach H, Elger CE, Wiestler OD, *et al.* The spectrum of long-term epilepsy-associated tumors: Long-term seizure and tumor outcome and neurosurgical aspects. Epilepsia 2003;44:822-30.
29. Zentner J, Hufnagel A, Wolf HK, Ostertun B, Behrens E, Campos MG, *et al.* Surgical treatment of neoplasms associated with medically intractable epilepsy. Neurosurgery 1997;41:378-86.
30. Fisher RS, van Emde Boas W, Blume W, Elger C, Genton P, Lee P, *et al.* Epileptic seizures and epilepsy: Definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). Epilepsia 2005;46:470-2.
31. Hildebrand J, Lecaille C, Perennes J, Delattre JY. Epileptic seizures during follow-up of patients treated for primary brain tumors. Neurology 2005;65:212-5.
32. Chang BS, Lowenstein DH, Quality Standards Subcommittee of the American Academy of Neurology. Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury: Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2003;60:10-6.
33. Cohen N, Strauss G, Lew R, Silver D, Recht L. Should prophylactic anticonvulsants be administered to patients with newly-diagnosed cerebral metastases? A retrospective analysis. J Clin Oncol 1988;6:1621-4.
34. De Santis A, Villani R, Sinisi M, Stocchetti N, Perucca E. Add-on phenytoin fails to prevent early seizures after surgery for supratentorial brain tumors: A randomized controlled study. Epilepsia 2002;43:175-82.
35. Rüegg S. Dexamethasone/phenytoin interactions: Neurooncological concerns. Swiss Med Wkly 2002;132:425-6.
36. Lackner TE. Interaction of dexamethasone with phenytoin. Pharmacotherapy 1991;11:344-7.

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