## CASE REPORT

# M.M. Sumer • H.T. Atasoy • A. Unal • M. Kalayci • K. Mahmutyazicioglu • O. Erdem Indriven sphenoid wing as a cause of post-traumatic epilepsy

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Abstract Post-traumatic epilepsy is more frequent after severe head injuries, however the severity of the trauma is not always correlated with the injured brain tissue. We report a patient whose seizures developed 4 years after a face trauma. Upward displacement of the sphenoid wing caused a contusion at the orbital surface of the frontal lobe. Computed tomography, magnetic resonance imaging and electroencephalographic findings are presented. The patient responded well to commonly used antiepileptic drugs.

**Key words** Post-traumatic epilepsy • Sphenoid wing • Head injury

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#### Introduction

Post-traumatic epilepsy is an increasing concern with the growing number of traumatic brain injuries. The risk of epilepsy increases with the presence of traumatic brain injuries and the severity of the trauma [1, 2]. Missile injuries, foreign body or bone fragments are frequently reported as causes for epilepsy. Here we report a patient with post-traumatic epilepsy that resulted from the upward displacement of the sphenoid wing.

#### **Case report**

A 20-year-old man with a history of epileptic seizures that had started at 7 years of age was admitted to our clinic. Clinical characteristics of the seizures that he and his parents were reporting were the same: in the beginning he would feel strange epigastric sensations and his head and eyes would turn to the left side; immediately after, he would lose consciousness and fall down on the floor; within 3–4 minutes, he would regain consciousness without postictal confusion. These spells recurred 5- to 8-times per year. His pre-perinatal history was unremarkable. No febrile convulsions were described. However, at 3 years of age he fell from a wall 3 metres high and struck his face to the ground without losing consciousness. No extraordinary condition was observed until epileptic fits occurred 4 years later, as told by his parents.

The patient was in a good health with no complaints except for these spells. General physical and neurological examinations were completely normal. Routine biochemical analyses were within normal limits. Cardiac examination that included 12-channel electrocardiography and echocardiography was normal. A digital electroencephalography (EEG) examination revealed bilateral slow spike and wave



Fig. 1 EEG shows bilateral slow spike and wave discharges, predominantly on the right frontal region

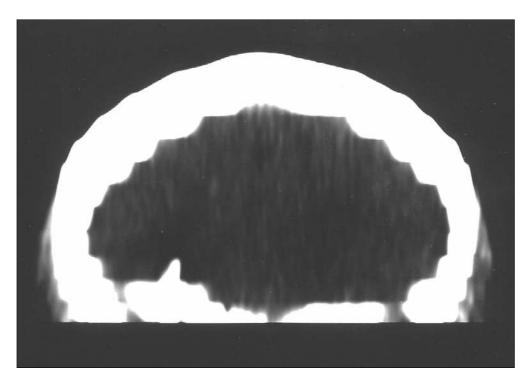


Fig. 2 Axial computed tomogram shows bone fragment localized in right frontal lobe with old infarct

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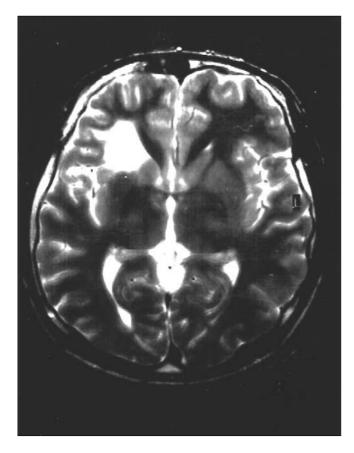


Fig. 3 Coronal cranial reconstruction CT shows upward displacement of the right sphenoid wing

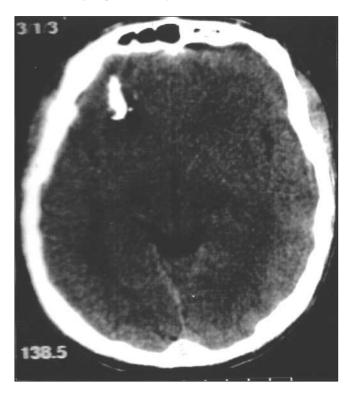


Fig. 4 Cranial MR image shows right Heubner artery territory infarct

discharges, predominantly on the right frontal region (Fig. 1) Axial computed tomography (CT) of the brain showed a bone fragment in the right frontal lobe (Fig. 2). CT also showed an old right Heubner artery territory infarct. Coronal cranial reconstruction CT showed upward displacement of the right sphenoid wing (Fig. 3). Cranial magnetic resonance imaging further delineated the infarcted tissue (Fig. 4).

After diagnosis, carbamazepin (800 mg/day) was given and no spells were observed during the follow-up of 12 months.

#### Discussion

Intraparenchymal lesions increase the risk of developing epilepsy after head injuries [2, 3]. This patient had an infarct as the cause of epilepsy, which may have resulted from contusion of the frontal parenchymal tissue due to the indriven sphenoid wing. Another puzzling mechanism is the upward displacement of sphenoid wing as the cause of the facial trauma, compressing the adjacent Heubner artery while causing an infarct in its territory. However, this is a speculative mechanism. It is also possible that the cavity that is located in the anterior part of the caudate nucleus is a residua of post-traumatic parenchymal haematoma. The clinical pattern of the seizures favours a lateral-paramedial frontal focus or the orbital surface (gyrus orbitales or gyrus rectus) of the frontal lobe. However, an orbital focus generally lacks interictal signs on scalp EEG recordings [4]. Epigastric sensations generally point to a temporal lobe origin [5]. In this patient, epigastric sensations suggest that epileptic discharges from the focus localized in the orbital surface of the frontal lobe spread via the fasciculus uncinatus to the right hippocampal and to the temporopolar cortex, and simultaneously to the right frontal Broadmann area 8, responsible for conjugate eye deviation to the left side. Loss of conciousness suggests bilateral involvement of both hemispheres. The clinical characteristics of the seizures, lack or minimal postictal state and EEG findings are strictly consistent with the radiological data and clearly depicted a right frontal focus.

Bone fragments in lacerated brain tissue have been described as a cause of epilepsy [6, 7]. However, most of these reports derived from war conditions and are generally related to missile injuries. The present case is different in that the trauma is not as severe as in the other studies. To the best of our knowledge, indriven sphenoid wing has not previously been described in the literature as the cause of post-traumatic epilepsy.

Periorbital region trauma may appear superficial and minor at first glance. The unique shape and tiny bony roof of the orbit give these injuries a significant risk of fragmentation. Although they may initially be asymptomatic, scrutinized care is essential.

### References

- Angeleri F, Majkowski J, Cachio G, Sobieszek S, D'Acunto S, Gesuita R, Bacleda A, Polonara G, Krolicki L, Signorini M, Salvolini U (1999) Posttraumatic epilepsy risk factors: oneyear prospective study after head injury. Epilepsia 40:1222–1230
- Pohlmann-Eden B, Bruckmeir J (1997) Predictors and dynamics of posttraumatic epilepsy. Acta Neurol Scand 95:257–262
- 3. Asikainen I, Kaste M, Sarna S (1999) Early and late posttraumatic seizures in traumatic brain injury rehabilitation patients: brain injury factors causing late seizures and

influence of seizures on long-term outcome. Epilepsia 40:584-589

- Salanova V, Morris HH, Van Ness P, Kotogal P, Wyllie E, Luders H (1995) Frontal lobe seizures: electroclinical syndromes. Epilepsia 36(1):16–24
- Kotagal P (1991) Seizure symptomatology of temporal lobe epilepsy. In: Lüders HO (ed) Epilepsy surgery. Raven, New York, pp 143–156
- Rinaldi A, Gazzeri R, Conti L, Schiavone M, Cava M, Tirelli GC (2000) Cranio-orbital missile wound and bullet migration. Case report. J Neurosurg Sci 144:107–112
- Askenasy JJ (1989) Association of intracerebral bone fragments and epilepsy in missile head injuries. Acta Neurol Scand 79:47–52