tually no psychiatric complications have been described. Psychiatric disorders in HHT may develop shortly after adolescence and present with schizophrenia-like symptoms with or without accompanying liver cirrhosis.[3] In addition, a paradoxical cerebral embolism may occur from silent pulmonary arteriovenous malformations, especially in cases with the *HHT1* type. Furthermore, transient ischemic attacks may occur regardless of the degree of respiratory symptoms. Finally, a disturbed angiogenesis with fragility of small vessels may be involved in brain dysfunction and neuropsychiatric symptomatology.[4]

Recently, it was suggested that the neurovascular manifestations show age-related penetrance with an increased prevalence of cerebral manifestions over the lifespan.[5]

Although a causal relationship between HHT and psychopathology cannot be demonstrated in the presented cases, it is worthwhile to consider brain manifestations of the disease, including neuropsychiatric complications, that manifest via different pathways. It is therefore highly remarkable that no neuropsychiatric complications of HHT are reported in the literature.

Harold J.H. Kuijpers, M.D.
F.M.M.A. van der Heijden, M.D., Ph.D.
S. Tuinier, M.D., Ph.D.
Vincent van Gogh Institute for Psychiatry, the Netherlands
W.M.A. Verhoeven, M.D., Ph.D.
Vincent van Gogh Institute for Psychiatry, and Erasmus University Medical Centre, Department of Psychiatry, Rotterdam, the Netherlands

References

1. Begbie ME, Wallace GMF, Shovlin CL: Hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome): a view from the 21st century. Postgrad Med J 2003; 79:18–24
2. Letteboer TGW, Zewald RA, Kamping EJ, et al: Hereditary hemorrhagic telangiectasia: ENG and ALK-1 mutations in Dutch patients. Hum Genet 2005; 116:8–16
3. Watanabe A: Portal-systemic encephalopathy in non-cirrhotic patents: classification of clinical types, diagnosis and treatment. J Gastroenterol Hepatol 2000; 15:969–979
4. Fernandez LA, Sanz-Rodriguez F, Zarrabeitia R, et al: Blood outgrowth endothelial cells from hereditary haemorrhagic telangiectasia patients reveal abnormalities compatible with vascular lesions. Cardiovasc Res 2005; 68:235–248
5. Krings T, Ozanne A, Chng SM, et al: Neurovascular phenotypes in hereditary haemorrhagic telangiectasia patients according to age: review of 50 consecutive patients aged 1 day–60 years. Neuroradiology 2005; 47:711–720

Posttraumatic Parieto-Occipital Epilepsy

SIR: The prevalence of epilepsy after traumatic brain injury (TBI) is about 4%,[1] parietal and occipital seizures being considered a rare outcome.[2,3] We report a case of a patient where posttraumatic parieto-occipital seizures were misidentified as psychiatric symptoms, and stress the importance of the neuropsychological evaluation in the diagnosis, especially when imaging and electrophysiological exams show no anomalies.

Case Report

A 39-year-old right-handed woman, with no psychiatric history, suffered a TBI after being hit by a car, presenting a Glasgow Coma Scale score of 15 upon hospital admission. The cranial computed tomography scan revealed epidural left temporobasal hematoma and sinking left zygomatic arch fracture. More than a year after TBI, she started complaining of tingling and numbness in the right leg and inability to move despite no loss of power in the limb. These episodes lasted 1 to 2 minutes without loss of consciousness, but she had to interrupt ongoing tasks. They were preceded by a sense of malaise and followed by fatigue and disorientation, and their frequency had increased to an episode every 2 weeks. She also complained of poor concentration, forgetfulness, sleep disturbances, headache, dizziness, anxiety and sadness, and being medicated with antidepressants, without response. Magnetic ressonance imaging (MRI) 2 years after the accident and the EEG revealed no anomalies. During the neuropsychological evaluation, when drawing a three-dimensional figure (cube), she complained of difficulty in the task, dizziness and tingling of the right leg, followed by involuntary rapid tremor-like movements on the same side for about a minute, with no loss of consciousness. On the same day the EEG revealed paroxysmal bilateral temporo-occipital activity with left accentuation and contra-lateral homonymous propagation. The suggestion test was negative. She was medicated with a regimen of sodium valproate, 1600 mg daily, and reevaluated after 6 months, but when drawing a three-dimensional figure she developed a seizure with the same characteristics.

Comment

Our patient presented few risk factors for late seizures,[4] the first seizure occurring more than a year after TBI, similar to the 20 months' mean time previously reported.[5] Parietal and occipital seizures are mainly characterized by the presenting auras, including somatosensory phenomena, although the clinical manifestations may spread beyond and overshadow the focal origin. The paresthesia may spread in a Jacksonian manner, with motor activity in the affected body mem-

ber following the sensations in about 50% of cases. Some parietal seizures may resemble panic attacks and be misdiagnosed as a psychiatric disorder, but showing poor response to antidepressants, as with our patient. Parieto-occipital seizures may be provoked by stimuli involving the receptive, interpretive, and connective function of the parietal and occipital lobes. The EEG may show focal discharges in the posterior regions, but the seizures are often electrically silent.[3] Even though MRI and functional imaging often reveal underlying pathology,[3] a normal exam, as in our patient, should not exclude the presence of organic lesion. The constructional apraxia, revealed as a disturbance in drawing without correct spatial relationships, existed despite the absence of apraxia for single movements.[6] The copying deficit was particularly evident in drawing three-dimensional figures, and was not simply one of formulating the sequence of steps necessary to produce a complex abstract form. This may be due to a difficulty in encoding the pictorial structure of a model depicting a three-dimensional object, suggesting the existence of a parietooccipital lesion. In summary, although a rare outcome from TBI, posttraumatic parieto-occipital epilepsy may occur even after minor head trauma and few risk factors, and respond poorly to anticonvulsants. Symptom presentation is pleomorphic, and can be misdiagnosed as psychiatric. The neuropsychological evaluation is useful in the diagnosis, especially when functional and neuroimaging exams show no anomalies, and may help establishing a causative role for TBI in producing neuropsychiatric disorders, which is important from clinical, scientific, and legal perspectives.

Sofia Brissos, M.D.
Department of Psychiatry, Júlio de Matos' Psychiatric Hospital, Lisbon, Portugal
Vasco Videira Dias, Psy.D.
Department of Psychology and Sociology, Autonomous University of Lisbon, Portugal
Teresa Paiva, M.D., Ph.D.
Department of Neurology, CENC, Lisbon, Portugal

References

1. Carvajal P, Almárcegui C, Pablo MJ, et al: Crisis parciales postraumáticas. Rev Neurol 2001; 33:737–739
2. Siegel AM, Williamson PD: Parietal lobe epilepsy. Adv Neurol 2000; 84:189–199
3. Sveinbjornsdottir S, Duncan JS: Parietal and occipital lobe epilepsy: a review. Epilepsia 1993; 34:493–521
4. Temkin NR: Risk factors for posttraumatic seizures in adults. Epilepsia 2003; 44:18–20
5. Salazar AM: Posttraumatic epilepsy: pathogenesis. Epilepsia 1998; 39:29–35
6. Marshall RS, Lazar RM, Binder JR, et al: Intrahemispheric localization of drawing dysfunction. Neuropsychologia 1994; 32:493–501

Patau Syndrome

SIR: Patau syndrome is a congenital disorder which was reported by Dr. Klaus Patau in 1960.[1] The syndrome is caused by presence of an extra copy of chromosome 13. Other changes in chromosome 13, such as translocation, can also result in the characteristics classified as Patau syndrome. The syndrome causes serious physical and mental abnormalities, including neurological impairment, structural facial defects, heart defects, and mental retardation.

The incidence of Patau syndrome is approximately one per 12,000 live births.[2–4] The median survival age for children with the syndrome is 2.5 days. The most common cause of death is cardiopulmonary complications. Forty-five percent of the patients die within the first month of life and 70% die by 1 year of age. Survival to adulthood is extremely rare.

Case Report

We would like to report a 51-year-old Caucasian woman with Patau syndrome. Her chromosomal analysis showed partial translocation of chromosome 13 (unbalanced rearrangement between chromosome 13 and chromosome 14).

She had had 17 siblings in all. Eight of them died within the first few weeks of life. Another five died between the ages of 10 and 34 as a result of the medical complications of the syndrome; these siblings had mental retardation and psychosis. One living sibling, a 30-year-old man, suffered from mental retardation and psychosis. Three other siblings, two men and one woman, had no chromosomal abnormalities and are healthy.

The patient was born at full term with six toes on each foot, a small head, small eyes, low-set ears, and a cleft lip. Although all of her developmental milestones were delayed, she graduated from a special education high school. Her Wechsler Intelligence Scale for Children at 4 years old showed an IQ of 61 and moderate mental retardation. Her Wechsler Adult Intelligence Scale (WAIS) at 18 years old showed an IQ of 74 and educatable mental retardation while Rorschach testing showed severe immaturity. Her WAIS at 34 years old showed an IQ of 72 and borderline intellectual capabilities. The Halstead-Reitan Neurophysiological Battery showed serious neurological impairment. Her EEG showed complex partial seizures, and her computed tomography (CT) scan showed blunting of the frontal horns of the ventricular system.