FRONTAL LOBE SYNDROME FROM BILATERAL GLOBUS PALLIDUS LESIONS

A COMPLICATION OF WERNICKE'S ENCEPHALOPATHY

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SUMMARY — A 38 year-old man developed the classical clinical picture of Wernicke's encephalopathy as a consequence of prolonged total parenteral nutrition. As a late complication he developed a frontal lobe syndrome. Bilateral globus pallidus lesions were observed in the CT-scan examination. Some aspects related to the cortical syndromes caused by subcortical lesions are discussed.

Síndrome de lobo frontal por lesões de globus pallidus bilaterais: complicação da encefalopatia de Wernicke.

RESUMO — Relata-se um caso de encefalopatia de Wernicke que ocorreu em paciente masculino de 38 anos, como complicação de alimentação parenteral total. Houve boa recuperação clínica com suplementação de tiamina mas, como complicação neurológica tardia, o paciente desenvolveu quadro amnésico associado a síndrome de lobo frontal caracterizada por apatia, perseveração e alteração de testes de função cortical frontal. TC revelou presença de lesões bilaterais de globo pálido. São discutidos alguns aspectos relacionados ao desenvolvimento de síndromes corticais frontais em decorrência de lesões estruturais subcorticais.

Wernicke's encephalopathy (WE) is characterized by a classic triad of ocular abnormalities (eg. nystagmus, ophthalmoplegia, pupillary changes, ptosis, retinal hemorrhages, and papilledema), ataxia and abnormal mental state, and is caused by deficiency of thiamine 10. Strub[12] recently described a patient suffering frontal lobe syndrome as a consequence of bilateral globus pallidus lesions of unclear etiology. We describe a patient who developed WE during total parenteral nutrition 1.3,6,9. As a late complication, bilateral globus lesions leading to a frontal lobe syndrome was detected.

CASE, REPORT

OS, a 38-year-old man was admitted in May 3, 1988 with hematemesis. He was known to drink alcoholic beverages daily, the amount ignored. An endoscopic examination disclosed a duodenal ulcer with active hemorrhagic fundi. In May 12, 1988, he was submitted to a partial gastrectomy. Seven days later he was reoperated due to the formation of a duodenal-cutaneous fistula and a right subphrenic abscess. He received appropriate antibiotics, and the next day parenteral hyperalimentation was initiated (Aminoplasmal + glucose 50% 4- potassium chloride + acid potassium phosphate -f- calcium gluconate + sodium acetate + folic acid. Total daily volume: 1000 mL). Five days later the daily volume of i.v. parenteral solution was increased to 2000 mXi. In June 18, 1988, the piatient was found confused and obnubilated. Four days later his mental condition worsened, and nystagmus to all directions, right abducens palsy, sluggish pupillary reflexes to light, intentional tremor with dysmetria in the upper limbs were observed. A CT scan and a CSF exam were performed,

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and were normal. He was admitted to the Intensive Care Unit with arterial hypotension (80/40mmHg) and sinusal tachycardia *(120* bpm). At this time, he was found somnolent, desorientated, untalkative and uncooperative. Nystagmus to all directions, right abducens palsy, sucking and grasping reflexes in both upper limbs, and global hypotonia were present. Muscle power wias normal in all limbs. Cutaneous plantar reflexes were flexor. The diagnosis of Wernicke's encephalopathy was made and thiamine lOOmg bid, i.v., was initiated. The next day (June 19), he was comatous and irregular breathing developed. He was entubated and mechanically ventilated for seven days, when his level of consciousness begun to improve. During this time his blood pressure, progressively returned to normal levels. Thereafter his neurological deficits progressively improved. During his admission the following blood laboratory tests were regularly performed and were normal: sodium, potassium, phosphorus, magnesium, hepatic transaminases, creatinine and glucose. He was discharged from the Intensive Care Unit in June 30, 1988, and delivered home in August 5, 1988. At this time he was fully conscious, giving short answers when (asked and without spontaneous speech. His thought content was normal, but there was a marked recent memory impairment. His speech was normal, but truncal and limbs ataxia were present, and he could walk only with support. All deep reflexes were present and normal, except the achilleus. Global hypotonia was unchanged. Grasping and sucking reflexes disappeared. Nystagmus could be elicited but was not marked, and the complaints of diplopia and sixth nerve palsy disappeared. Three months later the patient was reviewed, and his wife complained of his lack of general and sexual interest, tendency to be isolated, short periods of irritability, without euphoria or bouts of crying. He gained weight and was going to be overweight, but hyperphagia could not be diagnosed. He was not properly sad and his thoughts were not melancholic. Retrograde amnesia and confabulation were absent, but his ability to learn, or to form new memories was greatly impaired. Alternating sequencing tasks were bilaterally impaired 11. Although the diagnosis of depression could not be established with certainty, imipramine 75mg ad was tried for three months without success. In December 1989, a CT scan disclosed bilateral small low density areas in the region of globus pallidus (Fig. 1). The patient was reexamined at this time, and his mental status and neurological examination were unchanged.

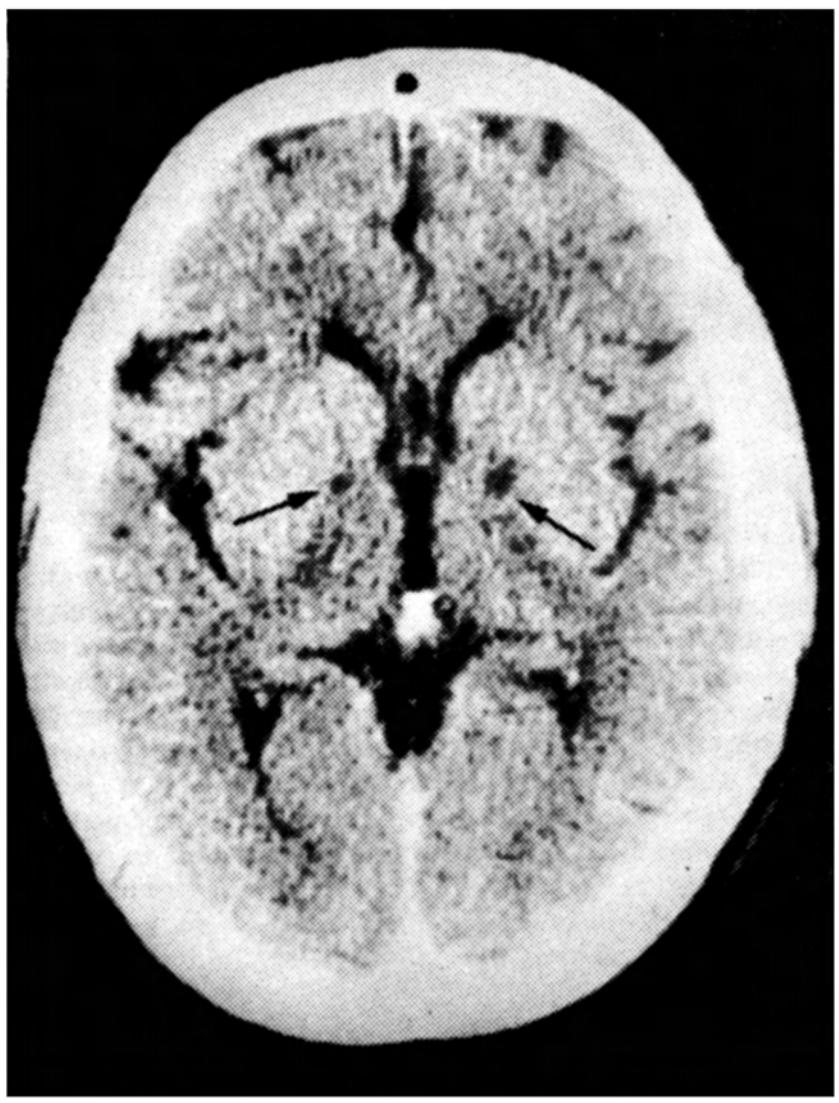

Fig. 1 — Case OS. CT scan: bilateral globus pallidus lesions (arrows).

COMMENTS

Most reports of CT findings in WE have been those of diffuse cerebral atrophy, bilateral low density lesions in the thalamic region, hypothalamus, and around the aqueduct[5,7,8]. In the acute stage, these lesions may appear as contrast-enhancing areas due to focal dilatation of capillaries. In the case reported by Strub[12], the bilateral hemorrhagic lesions in the globlus pallidus detected by MRI may be an example of this stage although clinical diagnosis of WE could not be established in that case. The lesions observed in our case, not observed in an early stage, may be due to tissue necrosis as occurs in the late stages of WE.

The symptoms of frontal lobe syndrome without direct damage to the frontal lobes may be caused by damage of subcortical brain structures that have strong interconnections with the frontal lobes[12-14]. More recently, Wolfe et al.[14] found that patients with multiple lacunes, mostly involving the basal ganglia, were impaired in several tests sensitive for frontal lobe dysfunction. In addition, most of these patients showed an apathetic behavior.

We also should keep in mind that the classical clinical picture of WE, as observed in our case, is rare[4]. Coma is an importante but unusual feature of acute WE 2,13. In fact, WE diagnosis should be suspected in alcoholics presenting in coma without localizing signs. Such patients should receive empirically intravenous thiamine while other causes of coma are excluded 13. Hypotension and hypothermia, as observed in our case, may be related to hypothalamic involvement, causing impaired autonomic regulation 3.

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