

Central Pontine Myelinolysis: Correlation between CT and Electrophysiologic Data

J. Sztencel,¹ D. Baleriaux,¹ S. Borenstein,^{2,3} E. Brunko,³ and D. Zegers de Beyl²

Because of the nature and location of the lesion in central pontine myelinolysis, considerable difficulties in diagnosis may arise. Computed tomography (CT) and brainstem auditory-evoked potentials are useful in following the spread and regression of the pontine lesion. The correlation between clinical evolution, serial CT scans, and brainstem auditory-evoked potentials is considered in a patient with central pontine myelinolysis and subsequent complete recovery with special emphasis on the evolution of the CT scan after recovery.

Central pontine myelinolysis (CPM) is the most likely diagnosis when a patient with a background of metabolic nutritional and electrolyte disorders develops quadriplegia and pseudobulbar signs over several days [1]. Although once thought to be invariably fatal, survival after CPM has been reported in recent years [1-3]. In the absence of pathologic proof, it can be difficult to diagnose the disease because of the location and nature of the lesion. Brainstem auditory-evoked potentials (BAEP) are useful in detecting the lesion in the pontine tegmentum: the latency difference between waves I and V reflects the conduction velocity in the pontine segment of the auditory pathway, and reversible prolongation of the I-V interpeak latency has been shown to occur in patients with CPM who recover [2]. High-resolution computed tomography (CT) may demonstrate the radiolucent pontine lesions of CPM [4, 5]. Little, however, is known on the radiologic evolution when recovery occurs.

We closely followed the disease process of CPM in one patient for 8 months with serial CT scans and BAEP. The patient made a

full recovery. We will concentrate on the correlation between clinical, radiologic, and electrophysiologic data and on the radiologic evolution over several months after recovery.

Case Report

A 28-year-old man with a long-standing history of excessive alcohol intake was admitted because of dysarthria. He developed a febrile illness 3 days before admission. On admission his temperature was 38°C, and he was severely dehydrated, dysarthric, and drooling. There was a slight quadriparesis with extensor plantar reflexes. Gross myoclonic jerks affected his limb and head on attempted movement. Serum electrolytes, liver function tests, and lactate were normal. Lumbar puncture showed normal cerebrospinal fluid (CSF). The patient developed quadriplegia with bilateral facial palsy and paralysis of tongue movements and swallowing over the next ten days.

CT 3 weeks after admission showed an extensive hypodense lesion in the pons (fig. 1A). Contrast injection did not change the density. BAEP at about the same time showed an increase of the I-V interpeak latency on right ear stimulation and desynchronization of waves IV and V (fig. 2). An earlobe-vertex subcutaneous needle electrode derivation was used. Clicks of 80 dB intensity were administered monaurally with alternating polarity and a stimulus frequency of 10/sec. The analysis time was 10 msec and band pass was 32-3,000 Hz. No reproducible wave pattern was seen after left ear stimulation because of severe congenital left ear

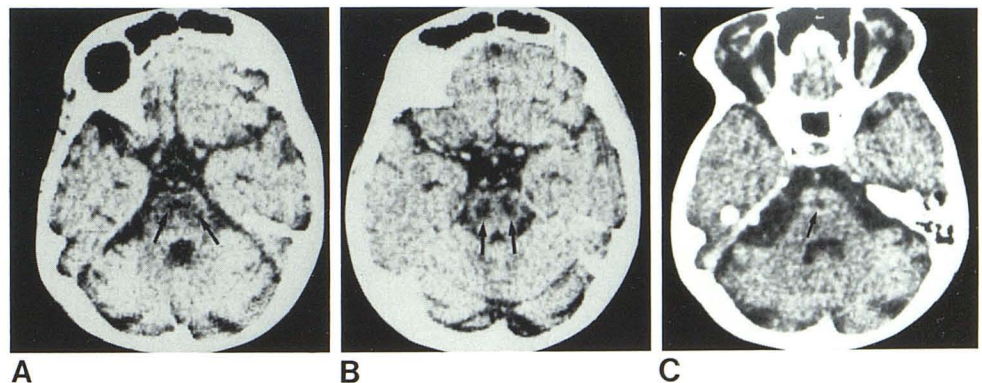


Fig. 1.—A, 3 weeks after admission. Large hypodense lesion extends throughout pons (arrows). B, 4 weeks later. Two distinct well delineated hypodense lesions (arrows). C, 32 weeks after admission. Small hypodense lesion is still present (arrow).

¹ Department of Neuroradiology, Hôpital Erasme, Route de Lennik 808, B-1070 Brussels, Belgium. Address reprint requests to J. Sztencel.

² Department of Neurology, Hôpital Erasme, B-1070 Brussels, Belgium.

³ Unité de Recherche sur le Cerveau, Faculté de Médecine, Brussels, Belgium.

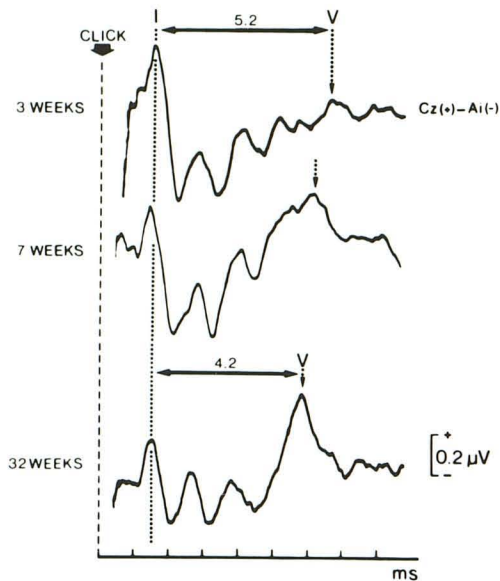


Fig. 2.—Evolution of BAEP I-V latency in right ear stimulation. Successive tracings with same recording conditions show progressive shortening of I-V interpeak latency. Cz (+) = vertex reference electrode; A₁ (-) = earlobe active electrode.

deafness. At 4–7 weeks quadriplegia, dysarthria and dysphagia gradually improved. CT 7 weeks after admission showed two distinct hypodense pontine lesions (fig. 1B). BAEP I-V interpeak latency, although still well beyond normal limits, correlated with the clinical improvement (fig. 2).

By week 20, recovery was complete. The lesion appeared smaller on CT with irregular margins. By week 32, CT showed a remaining small hypodense pontine lesion (fig. 1C), whereas BAEP showed a normal I-V interpeak latency and a high-amplitude well synchronized wave V (fig. 2). The patient was examined 18 months after admission. He was well and leading a normal active life.

Discussion

The diagnosis of CPM was suggested by the history of chronic alcoholism with a superimposed febrile illness and the subsequent development of progressive paralysis of the muscles deriving innervation at and below the pontine level. The diagnosis was supported by the finding of an extensive hypodense pontine nonenhancing lesion and the electrophysiologic evidence of slowing of conduction and desynchronization of the afferent volley in auditory structures between the lower pons and midbrain. During the patient's gradual recovery the serial CT scans showed slow regression of the hypodense pontine lesion. The CT scan was still very abnormal at the stage of complete recovery and remained abnormal 8 months after admission. The BAEP I-V interpeak latency correlated closely with clinical improvement and became strictly normal only 3 months after complete recovery. CT and BAEP appear to be complementary methods to confirm the clinical diagnosis of pontine involvement in CPM. Both are useful to follow the spread and eventual regression of the disease process, and BAEP seems to correlate more closely with the clinical evolution.

REFERENCES

1. Messert B, Orrison WW, Hawkins MJ, Quagliari CE. Central pontine myelinolysis. *Neurology (NY)* 1979;29:147–160
2. Stockard JJ, Mossiter VS, Wigbert CW, Kobayashi RM. Brain stem auditory-evoked responses in suspected central pontine myelinolysis. *Arch Neurol* 1976;33:726–728
3. Yufe RS, Hyde ML, Terbrugge IC. Auditory evoked responses and computerized tomography in central pontine myelinolysis. *Can J Neurol Sci* 1980;7:297–300
4. Thompson DS, Hutton WJT, Stears JC, Sung JH, Norenberg M. Computerized tomography in the diagnosis of pontine and extrapontine myelinolysis. *Arch Neurol* 1981;38:243–246
5. Telfer RB, Miller EM. Central pontine myelinolysis following hyponatremia demonstrated by computerized tomography. *Ann Neurol* 1979;6:455–456