

## MR imaging of seven presumed cases of central pontine and extrapontine myelinolysis

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**Abstract.** MRI was performed in seven patients with presumed central pontine and extrapontine myelinolysis. The underlying diseases were diabetes, lung cancer, Wilson disease, trauma, alcoholism, renal insufficiency and hemodialysis. CPM was found in four cases (in two of them extrapontine lesions were considered as resulting from Wilson disease), CPM and EPM in three patients. The localization of extrapontine changes included cerebellum, cerebral peduncles, caudate and lentiform nuclei, internal capsules, white matter and cortex of the cerebrum.

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**Key words:** Magnetic resonance imaging, central pontine myelinolysis, extrapontine myelinolysis

Central pontine myelinolysis (CPM) is an acquired myelin disorder affecting alcoholics and accompanying other diseases. In about 10% of cases lesions are found not only in the pons but also in cerebellum and/or cerebrum. They are called extrapontine myelinolysis (EPM). The etiology of this disorder is still uncertain; it is assumed that rapid correction of hyponatremia is the cause of myelin injury so the disease is perceived as an iatrogenic one (Brunner et al. 1990, van der Knaap and Valk 1995,) although there are reports concerning CPM in patients without evidence of hyponatremia (Bernsen and Prick 1999).

We studied seven patients (two females, five males), aged 18 - 67 (see Table I) at a 0.5 T unit (three patients) and at a 1.5 T one (four patients). SE/T1-, PD and T2-weighted images were obtained at 0.5 T; SE/T1-, TSE/T2- and FLAIR/T2-weighted images were performed at 1.5 T.

The clinical and imaging data are shown in Table I. All the patients showed lesions in the pons (Fig. 1). In two cases we found CPM only (Fig. 2, 3) – these were the cases No. 1 (diabetes) and No. 2 (left lung cancer 4 years ago, operated on, chemotherapy, now: right lung cancer, chemotherapy). In five patients we observed CPM and extrapontine lesions. Two of them had Wilson disease so we do not consider changes in the cerebellum in case No. 3 (Fig. 4) and in the lentiform nuclei in case No. 4 as EPM but as resulting from the underlying disease. Other three cases most likely represent EPM with lesions in the caudate and lentiform nuclei (Fig. 5, case No. 5), in the middle cerebellar peduncles, cerebral peduncles (Fig. 6) and posterior parts of the internal capsules (case No. 6) and in the cerebellum, white matter and cortex (Fig. 7) of

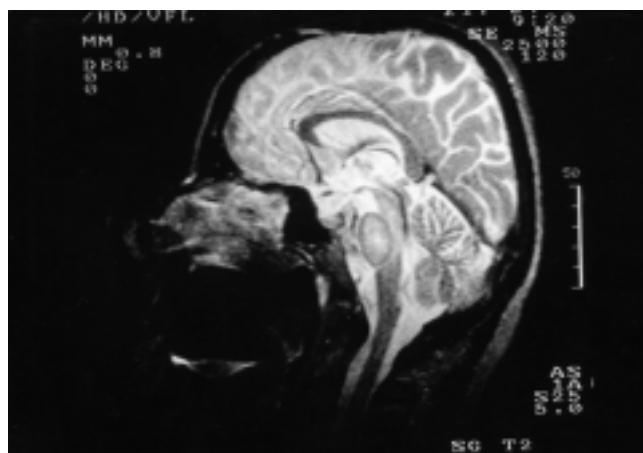


Fig. 1. SE/T2/sag. Case No. 4. Characteristic pattern of CPM with involvement of the central part of the pons and unaffected outer rim.

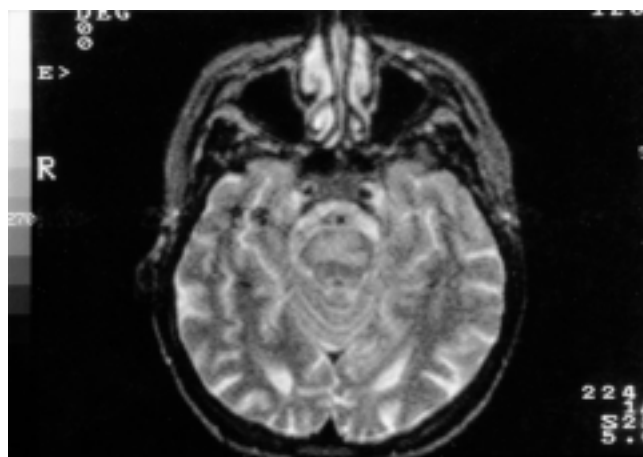


Fig. 2. SE/T2/ax. Case No. 1. CPM.

Table I

Clinical and MRI data						
Case No.	Age	Sex	Underlying condition	Lesions in the pons	Extrapontine lesions	
1	46	M	Diabetes	+	-	-
2	66	M	Lung cancer, chemotherapy	+	-	-
3	38	M	Wilson disease	+	Cerebellum	
4	36	F	Wilson disease	+	Lentiform nuclei	
5	18	F	Trauma	+	Caudate and lentiform nuclei	
6	21	M	Alcoholism	+	Cerebellum, cerebral peduncles, internal capsules	
7	67	M	Renal insufficiency, hemodialysis	+	Cerebellum, cerebral white matter and cortex	

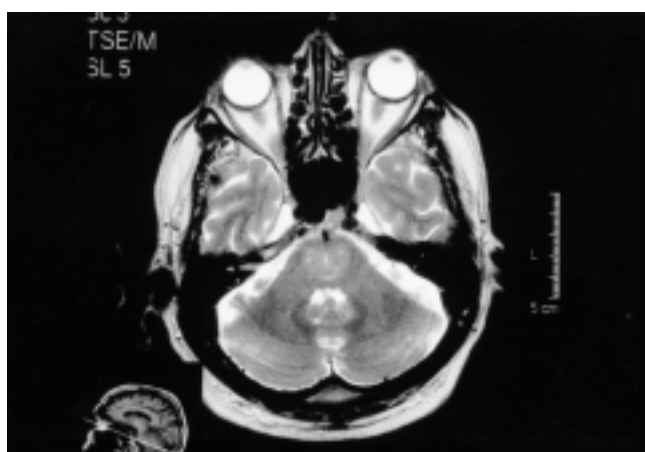


Fig. 3. TSE/T2/ax. Case No. 2. CPM.

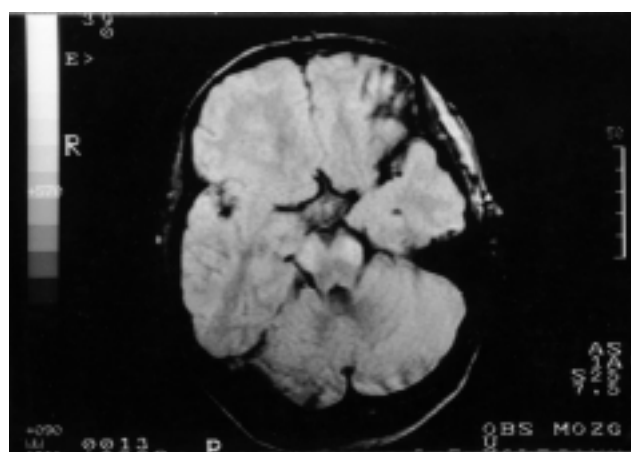


Fig. 6. SE/PD/ax. Case No. 6. EPM: involvement of the cerebral peduncles.



Fig. 4. TSE/T2/ax. Case No. 3. Symmetrical lesions in the cerebellum consistent with Wilson disease.

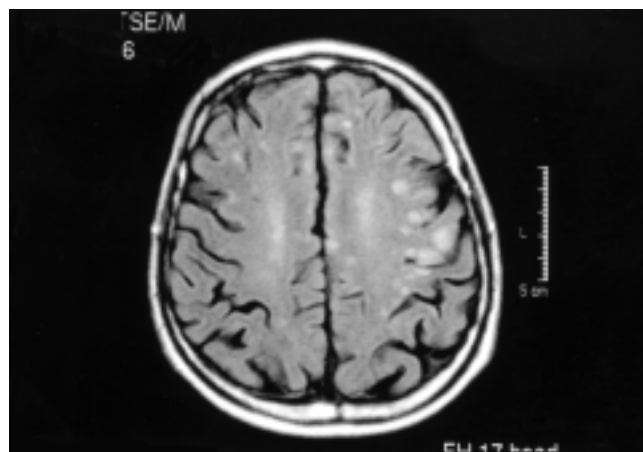


Fig. 7. FLAIR/T2/ax. Case No. 7. EPM: lesions located in the cerebral cortex.

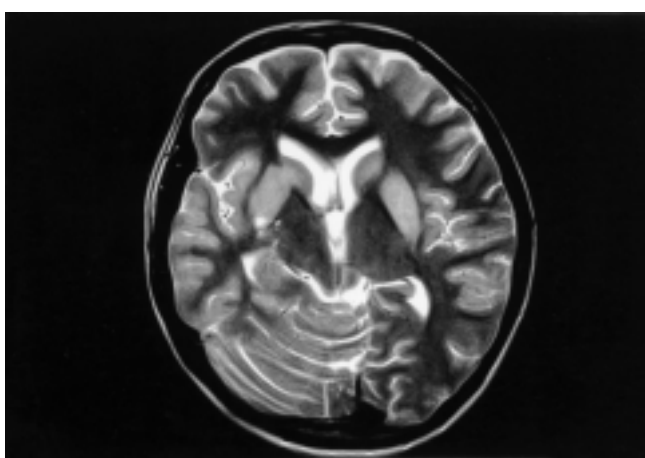


Fig. 5. TSE/T2/ax. Case No. 5. EPM: symmetrical lesions in the caudate and lentiform nuclei.

the cerebrum (case No. 7). In the last case as well as in cases No. 1 and 2 Gd-DTPA was administered to rule out primary tumour or metastases – no contrast enhancement was observed. The patient No. 7 died of the underlying disease. The autopsy was not performed.

The neurological symptoms of CPM and EPM are not typical of this entity. They may result from CPM/EPM or from the primary disorder like in case of Wilson disease. Some patients may be asymptomatic or present only mild clinical manifestations. In our material the spectrum of psychoneurological findings was wide: from behavioral changes without neurological signs in case No. 1 to tetraparesis in case No. 6.

Pathologically, demyelination and loss of oligodendrocytes are observed in the pontine tracts and nuclei in cases of CPM and in the other structures in EPM.

Astrocytosis and macrophages are also identified in the affected regions. These changes result in increased signal intensity observed on PD, T2-weighted and FLAIR images.

The characteristic MRI finding in CPM is a hyperintense focus in the central part of the pons with an unaffected outer rim (van der Knaap and Valk 1995). Five of our patients showed this pattern of demyelination. In two cases two separate foci of high signal intensity were visible in the pons – this pattern has also been shown in the literature (van der Knaap and Valk 1995). In about 90% of cases CPM is present without accompanying EPM. In our material two of seven patients had only CPM. Two others, with Wilson disease, showed lesions in the cerebrum and in the lentiform nuclei. CPM has been described in histologic studies in association with Wilson disease (Shiraki 1968, Goebel and Herman-Ben Zur 1976, Scheinberg and Sternlieb 1984, van Wassenaeer-van Hall et al. 1995). Therefore we considered pontine lesions in these patients as CPM while the extrapontine ones as connected with Wilson disease and resulting most probably from abnormal copper and iron depositions, demyelination and gliosis (van der Knaap and Valk 1995). There was no patient in our material with isolated EPM. We observed CPM and EPM in a patient after multisystem trauma, in an alcoholic and in a patient with renal insufficiency on dialysis therapy. The lesions were located in caudate and lentiform nuclei, in middle cerebellar peduncles, cerebral peduncles and internal capsules and in the cerebellum, white matter and cortex of the cerebrum, respectively. Both underlying conditions and localization of lesions are consistent with the data in the literature (Gallucci et al. 1989, van der Knaap and Valk 1995, Moore and Midha 1997, Agildere et al. 1998, Choe et al. 1998).

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*Received 12 December 2000, accepted 26 February 2001*