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Mild Central Pontine Myelinolysis: A Frequently Undetected Syndrome

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Summary. Over a period of 1 year we diagnosed central pontine myelinolysis (CPM) in five patients all of whom survived, two of them with complete functional recovery despite extensive lesions on cranial computerized tomography and magnetic resonance imaging.

Diagnosis was based upon the combination of an acute brainstem dysfunction with typical neuroradiological features; a history of chronic alcoholism or a preceding hyponatremia may serve as a diagnostic hint.

The spectrum of symptoms ranged from severe tetraplegia and cranial nerve palsies to latent signs of pyramidal tract lesions and discrete ocular motor abnormalities. In two patients pontine and extrapontine manifestations of demyelination were confirmed neuroradiologically; in one patient a solely extrapontine manifestation was present.

Thus it is reasonable that: (1) the incidence of comparatively mild forms of CPM as well as extrapontine manifestations are more frequent than hitherto assumed, (2) the clinical outcome of the syndrome is better than expected from earlier fatal case reports and is quite independent of the extent of the lesion as it appears with brain imaging methods.

Key words: Central pontine myelinolysis – Hyponatremia – Alcoholism

Introduction

In 1959 Adams et al. first presented a pathohistological description of symmetrical demyelinative lesions in the central region of the basis pontis with relative sparing of axon cylinders and neurons in three alcoholics and one patient with malnutrition (Adams et al. 1959). They used the term central pontine myelinolysis (CPM) whereas later reports on the syndrome showed that similar histopathological changes with demyelination, neuronal damage and gliosis can also be found in other parts of the brain, especially in the basal ganglia, cerebellum, cortex, and subcortex (Finlayson et al. 1973; Wright et al. 1979; Goldman and Horoupian 1981; Thompson et al. 1981; Zegers de Beyl et al. 1983). Zwick et al. (1985) reported on four cases with central spinal myelinolysis with midline lesions of the funiculus gracilis.

CPM occurs predominantly in patients with alcoholism, but also in liver diseases, malnutrition, and Wilson's disease (Lüthy 1932; Goebel and Herman-Ben Zur 1976). Clinically it frequently follows a delirium tremens and combines with Wernicke's encephalopathy (Goebel and Herman-Ben Zur 1976) and very rarely with Marchiafava-Bignami syndrome (Ghatak et al. 1978).

Impairment of consciousness including coma and dysfunction of the basis pontis (locked-in syndrome, tetraparesis, pseudobulbar palsy) are prominent clinical features. If regions beyond the pons are affected, particularly the basal ganglia, then extrapyramidal motor symptoms may occur.

Precise etiology and pathogenesis of the disease are still unknown. Clinical findings and animal experiments have revealed the importance of a preceding electrolyte imbalance. In particular, the rapid correction of a preexisting hyponatremia plays an important role in the development of CPM (Kleinschmidt-De Masters and Norenberg 1981; Norenberg et al. 1982; Laureno 1983; Norenberg and Papendick 1984).

For many years CPM has been regarded as a fatal disease which can only be diagnosed at postmortem. With the introduction of computerized tomography however it has become possible to diagnose CPM *intra vitam* and several single case reports have since been published (Anderson et al. 1979; Telfer and Miller 1979; Yufe et al. 1980; Loizou and Rokos 1981; Thompson et al. 1981; Gerber et al. 1983; Hazratji et al. 1983; Sztencel et al. 1983; DeWitt et al. 1984; Larrue et al. 1984; Marra 1984; Nakada and Knight 1984; Rosenbloom et al. 1984; Schroth 1984; Stam et al. 1984; Yeow and Tija 1984; Alberca et al. 1985).

The purpose of this paper is to stress our recent clinical experience that CPM is not a rare and not an obsolete condition if one considers mild forms with relatively benign courses.

Patients and Clinical Findings

At the Neurological Clinic of Munich we have observed five patients with the clinical and neuroradiological criteria of CPM within the last year. During the acute phase of the condition a cranial computerized tomography (CCT) was performed in all patients, and magnetic-resonance (MR) imaging in four of them. Investigation of CSF included cell count, total protein, analysis of immunoglobulins; the basic myelin protein was determined in the CSF from two of the patients. Somatosensory and auditory brainstem evoked responses were recorded in four patients.

In two patients the disease developed after a preceding alcoholic delirium (see Table 1), one of these patients had suffered from grand mal seizures for several years. Two patients had a history of chronic alcoholism but without delirium

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Table 1. Clinical data in 5 patients with central pontine myelinolysis

Patient	Age	Sex	Concomitant diseases				Neurological symptoms and signs	Serum sodium (mmol/l)	CSF (n/3 cells/µl-mg/dl protein)	CCT hypodense area	MR pathologic area	Clinical outcome
			Alcoholism	Delirium	Hepatopathy	Others						
1	63	♀	+	–	+	Wernicke's encephalopathy, Korsakoff's psychosis	Cerebellar ataxia, horizontal gaze evoked nystagmus, abducens paresis left	127→137	4–22	Pons	Pons	In need of care
2	39	♀	–	–	–	Suprasellar meningioma, hypophyseal insufficiency	Spastic tetraparesis, downward gaze palsy	143→175 124→151	4–168 (ventricular shunt)	Pons	Not done	In need of care
3	42	♀	+	–	–	–	Latent signs of pyramidal tract lesion, vertigo	Normal	2–19	Pons	Pons + mesencephalon	Complete recovery
4	54	♂	+	+	+	Diabetes mellitus	Spastic tetraplegia, facial palsy bilateral, dysphagia, pathologic crying, myoclonus	113→140	1–34	Pons + paraventricular bilateral	Pons + paraventricular bilateral	In need of care
5	42	♂	+	+	+	Seizures	Spastic tetraparesis, rigor of all extremities	139→150	8–37	Thalamus right	Normal	Complete recovery

tremens immediately before the manifestation of CPM. The only patient who was not a chronic alcoholic suffered a relapse after operative treatment of a suprasellar meningioma with accompanying hypophyseal insufficiency.

Four patients had signs of pyramidal tract lesions, three of them showed severe spastic tetraparesis associated with downward gaze palsy, dysphagia, bilateral facial palsy, pathologic crying, myoclonus, and rigor. The fourth patient with signs of pyramidal tract lesions had only latent tetraspasticity without functional deficit, which in combination with vertigo were the sole symptoms of the disease. One patient was admitted with the typical signs of Wernicke's encephalopathy with cerebellar ataxia and horizontal gaze evoked nystagmus. Additionally this patient had Korsakoff's psychosis with confabulation, memory impairment, and disorientation. Left abducens paresis suggested a pontine lesion.

In two patients the disease appeared completely cured with respect to the neurological deficit. At the time of discharge or transfer three patients had obvious residual symptoms requiring nursing care and assistance.

Cerebrospinal Fluid. The cell count in the CSF of all the patients was normal. The CSF total protein was normal in four patients. The fifth patient, who had a ventricular shunt because of hydrocephalus, exhibited an increase in total protein of 168 mg/dl with a disturbance of the blood-brain barrier but without evidence of autochthonous immunoglobulins in the CSF. When two patients were examined 6 and 8 weeks after onset of the disease, there was no evidence of basic myelin protein in the CSF.

Retrospective reconstruction of serum sodium levels revealed that two patients had hyponatremia before the development of neurological symptoms (113 and 127 mmol/l), which was rapidly corrected. A third patient had an increase in serum sodium from 143 to 175 mmol/l before manifestation of the neurological deficit which in the further course of the disease fell to 124 mmol/l and was rapidly corrected to 151 mmol/l. Another patient had a mild elevation of serum sodium to 150 mmol/l after having a normal serum sodium (139 mmol/l). One patient had normal serum sodium at admission and during the entire hospital stay. The level of serum sodium before the development of clinical symptoms remained unknown.

Cranial Computerized Tomography (performed by the Department of Radiology, University of Munich). In four patients CCT showed a hypodense area in the pons. In one of these patients additional hypodense paraventricular areas were demonstrated bilaterally. One patient with no hypodense pons lesion only had a hypodense area in the right thalamus.

Magnetic-Resonance Imaging (performed by the Institute of Radiology, Dr. Wallnöfer, Dr. Weber, Dr. Scheid, Munich). MR imaging was performed using spin-echo and inversion recovery techniques (Picker International, 0.15 tesla). In three patients there was an area of increased signal intensity in spin-echo sequence analysis in the pons (Figs. 1 and 2), in one patient also in the lower mesencephalon, and in another patient in the paraventricular areas on both sides. In the fourth patient, in whom CCT revealed a hypodense area in the right thalamus, MR imaging was normal; however this examination

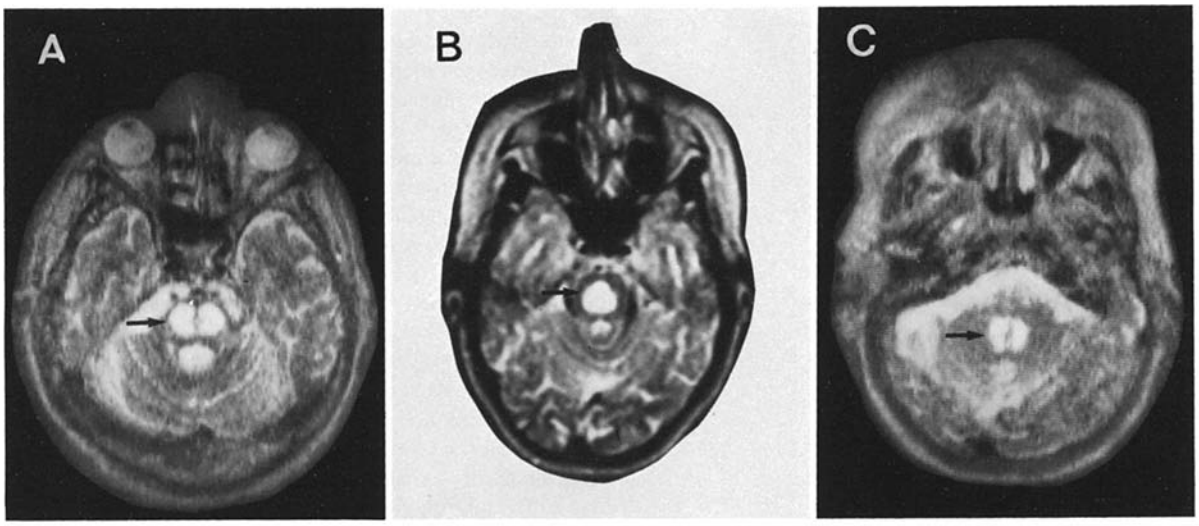

Fig. 1A–C. Magnetic resonance imaging in three patients with central pontine myelinolysis. MR imaging (spin-echo technique) shows an area of increased image intensity *(arrows)* occupying the basis pontis extending in **B** to the lower mesencephalon. In **A** and **C** the median raphe is well visualized between the two symmetrical bean-like areas in the basis pontis. (**A** = patient 4, repetition time 4260 ms, time echo 120 ms; **B** = patient 3, repetition time 3000 ms, time echo 120 ms, **C** = patient 1, repetition time 3000 ms, time echo 120 ms)

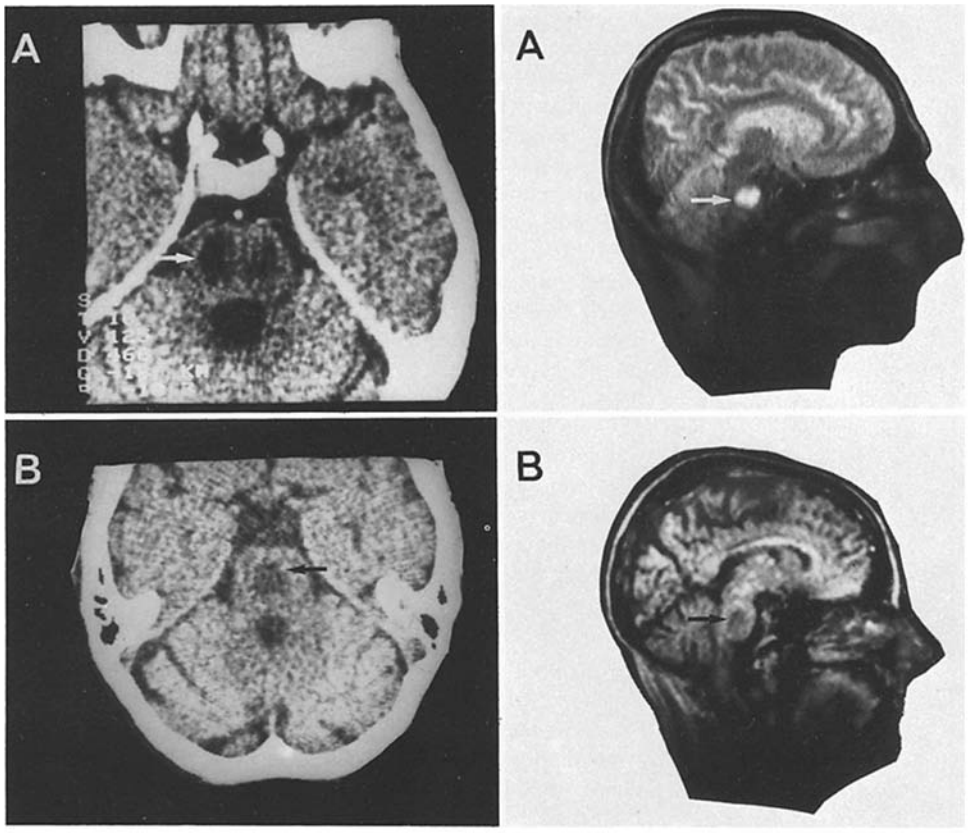

Fig. 2A, B. Computerized tomography and magnetic resonance (sagittal imaging) in two patients with central pontine myelinolysis. CCT and MR (spin-echo technique) show pathologic areas in the basis pontis *(arrows)*. (**A** = patient 4, contrast CCT, MR: repetition time 1500 ms, time echo 120 ms; **B**= patient 3, native CCT, MR: repetition time 500 ms, time echo 24 ms)

was performed 7 weeks after CCT, when the patient had fully recovered clinically.

Brainstem auditory evoked responses were normal in all patients. The somatosensory evoked potentials were normal in three patients, in the fourth patient no cortical responses could be recorded after stimulation of the median nerve bilaterally.

Discussion

Clinical Diagnosis

Our observation of five cases with CPM within a 1-year period suggests that this disease is more frequent than has been assumed so far. It is the improvement in radiological methods

such as CCT and MR, which has enlarged the clinical spectrum of the disease, whereas in the past diagnosis could only be made at autopsy.

Single reports in the literature on nonfatal cases with CPM describe severe clinical syndromes with tetraparesis and multiple cranial nerve palsies or locked-in syndrome. We also observed patients with mild clinical symptoms despite large hypodense symmetrical pontine areas which were discovered in CCT by chance. In one patient with the clinical manifestation of Wernicke's encephalopathy and Korsakoff's psychosis CCT showed a pathological pontine area typical for CPM but with no severe clinical symptoms. There was only a mild pontine oculomotor disturbance with horizontal gaze evoked nystagmus and incomplete abducens palsy. Since these symmetrical hypodense pontine areas in CCT are not found in patients with Wernicke's encephalopathy, one must assume the coincidence of CPM and Wernicke's encephalopathy. According to Goebel and Herman-Ben Zur (1976) 28% of 112 autopsy-proven cases with CPM had Wernicke's encephalopathy. The enlargement of the clinical spectrum of CPM makes it desirable to establish reliable diagnostic criteria.

Of the four main features pontine dysfunction must be regarded as the cardinal clinical sign. Even in patients with mild CPM pontine dysfunction can always be demonstrated clinically or electrophysiologically. The three other important diagnostic features, the typical pathological findings in CCT or MR, a history of chronic alcoholism, and electrolyte disturbances are optional.

There is a marked disassociation between the severity of neurological dysfunction and the extension of the CCT lesion. In our patients with a similar large symmetrical hypodense area in CCT the clinical picture ranged from tetraplegia to only a discrete disturbance of ocular motility. Part of the hypodense pontine area in CCT can therefore be explained by edema which has not yet led to structural damage.

Since CPM areas in the pons in CCT are highly characteristic—two circumscribed bean-like hypodense symmetrical areas in the center of the pons without space occupying effect —CPM can be diagnosed nowadays even in less typical clinical syndromes and diseases without a history of chronic alcoholism. On occasions differential diagnoses such as multiple sclerosis, lacunar infarction, Binswanger's encephalopathy, or progressive multifocal leucencephalopathy must be ruled out.

If CCT is normal in clinically suspected cases of CPM (Messert et al. 1979; Kandt et al. 1983), brainstem evoked responses and MR imaging may be helpful investigations (Stockard et al. 1976; Wiederholt et al. 1977; Yufe et al. 1980; Thompson et al. 1981; Szentcel et al. 1983; DeWitt et al. 1984; Schroth 1984). All our patients had normal auditory evoked responses, one of them had pathological somatosensory evoked responses. Until now there has only been one reported case with CPM in the literature in whom MR was carried out (DeWitt 1984). As in the case of DeWitt, in three of our patients MR yielded no further information on the degree and extension of the lesion as compared with CCT results. In one patient, however, MR demonstrated a mesencephalic manifestation which could not be shown in CCT.

In follow-up studies the clinical improvement of the patients correlated well with the normalisation of brainstem auditory evoked responses (Stockard et al. 1976; Wiederholt et al. 1977; Yufe et al. 1980; Sztencel et al. 1983; DeWitt et al. 1984; Zegers de Beyl 1985). On the other hand the reduction in the size of the affected pontine area in CCT lags behind clinical improvement (Telfer and Miller 1979; Thompson et al. 1981; Gerber et al. 1983; DeWitt et al. 1984; Larrue et al. 1984). Only Sztencel et al. (1983) were able to demonstrate one case in which slow resolution of the pathological CCT area corresponded to the gradual improvement of clinical symptoms.

With the further development of the resolution of brain imaging techniques it may become possible to detect extrapontine areas affected by the disease more frequently. Two of our five patients had extrapontine manifestations in CCT (Fig. 3), and the MR of another patient showed involvement of the mesencephalon. In a review of the literature Wright et al. (1979) found extrapontine alterations in 12 of more than 150 patients with CPM diagnosed at autopsy. Of the 23 patients with CPM reported in the literature in whom CCT was performed (Thompson et al. 1981; Hazratji et al. 1983) extraponto-mesencephalic manifestation of the disease was demonstrated in only 3. The localisation of the hypodense paraventricular areas (patient 4) required the exclusion of encephalomyelitis disseminata. Our patient with these paraventricular areas in CCT was 54 years old, had no history of disseminated demyelinative disease and developed clinical symptoms after an alcoholic delirium. The CSF was normal without autochthonous production of immunoglobulins, and multiple sclerosis could thus be excluded. One of our patients (patient 5) had an extrapontine hypodense area in the region of the thalamus on the right and a corresponding rigor of the contralateral extremities. The incidence of lacunar infarcts with demyelinisation of the white matter, basal ganglia, and the pons is typical of Binswanger's encephalopathy which however is correlated with arterial hypertension. None of our patients had hypertension.

Progressive multifocal leukencephalopathy is associated with a malignoma or cytostatic therapy and presents no diagnostic difficulties.

The demonstration of extrapontine areas in CPM suggests that Marchiafava-Bignami syndrome, the degeneration of corpus callosum in chronic alcoholism, is probably an isolated extrapontine variant of the same process (Wright et al. 1979).

Hyponatremia

Conger et al. (1969) were the first to stress the possible association of a preceding hyponatremia with the development of CPM. Since then several authors have described patients with hyponatremia as the initial pathogenetic factor (Tomlinson et al. 1976; Burcar et al. 1977; Messert et al. 1979; Wright et al. 1979; Norenberg et al. 1982).

In 1981 Kleinschmidt-DeMasters and Norenberg demonstrated demyelinative pontine and extrapontine lesions in rats treated with hypertonic saline after 3 days of vasopressin-induced hyponatremia.

In 1983 Laureno experimentally induced a central pontine and extrapontine myelinosis in dogs after rapid correction of vasopressin-induced hyponatremia. Hyponatremia alone or a slowly corrected hyponatremia did not produce the corresponding clinical and histological alterations.

In 1984 Norenberg and Papendick found more severe demyelinative lesions in rats after rapid correction of hyponatremia which lasted for 3 days than in a hyponatremia of 1 day's duration. Perhaps the rapid increase in serum sodium from normal to hypernatremia levels may also be important in the

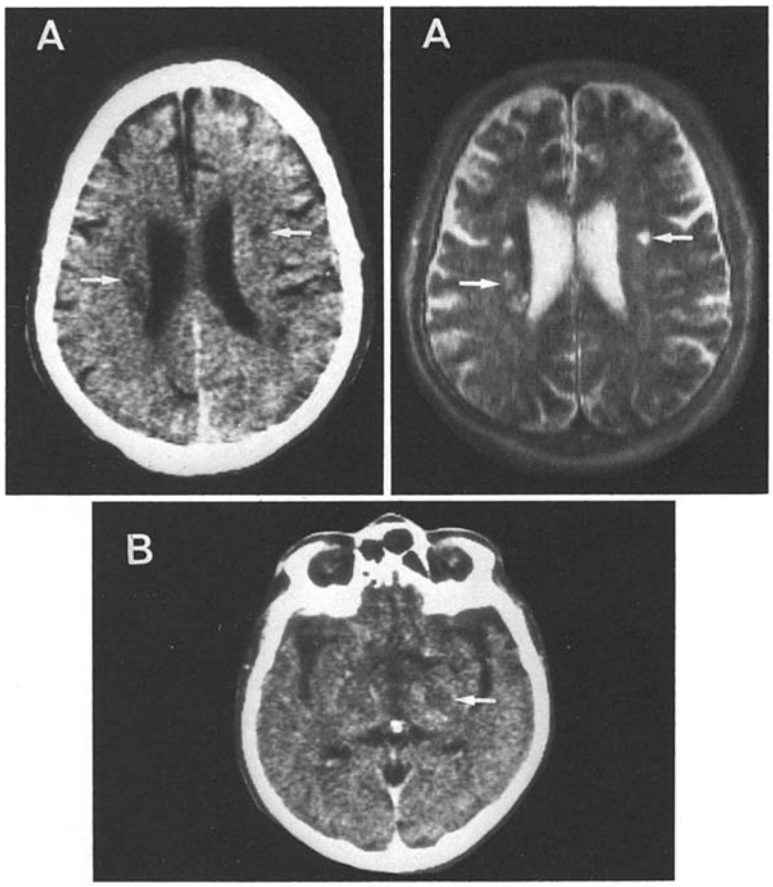

Fig. 3A, B. Computerized tomography and magnetic resonance imaging in two patients with extrapontine manifestation of central pontine myelinolysis. CCT in **A** shows an area of low density with a corresponding increased image intensity in MR paraventricular bilaterally *(arrows)*. CCT in **B** shows an area of low density in the right thalamus; MR imaging, performed 7 weeks after CCT examination; was normal. (**A** = patient 4, native CCT, MR: repetition time 4260 ms, time echo 120 ms; **B** = patient 5, contrast CCT)

pathogenesis of the disease (Laureno 1983). This may have occurred in two of our patients (2 and 5).

Hypothetically a rapid increase in serum sodium may cause an osmotic endothelial lesion with the release of myelinotoxic factors and the production of a vasogenic edema with subsequent demyelinating processes (Norenberg 1983). The main localisation of the lesion in the pons could be explained by the proximity of grey and white matter in this area; myelinotoxic factors of the well vascularized grey matter may thus cause a lesion of adjacent myelinated fibers.

In contrast Messert et al. (1979) suggested that brain edema following hyponatremia and volume expansion may be an important factor in the pathogenesis of the disease.

Chronic Alcoholism

Chronic alcoholism can be found in the history of 60% (Goebel and Herman-Ben Zur 1976) to 78% (Messert et al. 1979) of the patients with CPM. There are no reports in literature on the frequency of delirium tremens preceding CPM. Four of our five patients were alcoholics. The high incidence of alcoholism in the history of patients with CPM may be caused either by a direct toxic effect of alcohol or the pathogenetically important electrolyte disturbances, which are frequent in alcoholism. Alcohol has an antidiuretic hormone (ADH)-blocking effect. Alcohol withdrawal with following delirium tremens may lead to a rapid return of ADH function and thus cause hyponatremia, which predisposes for CPM (Messert et al. 1979).

On the other hand CPM has been observed only rarely in patients who suffer from other metabolic disorders with hyponatremic states, such as inappropriate secretion of ADH (Conger et al. 1969; Finlayson et al. 1973) and patients with Addison's disease (Kandt et al. 1983). There have been no reported cases of CPM in patients with Guillain-Barré syndrome, who frequently develop hyponatremia due to hormone dysfunction. Possibly mild pontine symptoms in those patients remain undetected so far. The high incidence of alcoholism and hyponatremia in the history of patients with CPM suggests that hyponatremia leads to the manifest disease of CPM when associated with a postulated pathological cofactor on the basis of alcoholism.

Both hypo- and hypernatremia may lead to CNS symptomatology such as confusion, lethargy to coma, muscle weakness or myoclonus, and seizures (Arieff and Guisado 1976).

A causative therapy for CPM is still unknown. Prevention must therefore be the focus of clinical efforts. Thus the careful control of electrolyte disturbances is very important especially in predisposed patients and electrolyte disturbances should be corrected gradually.

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