A CASE OF SUBACUTE BERI-BERI POLINEUROPATHY ASSOCIATED WITH WERNICKE'S ENCEPHALOPATHY?

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BACKGROUND

Peripheral neuropathy(PN) and Wernicke's encephalopathy(WE) are common typical manifestations of chronic alcoholism [1]. Generally, PN presents as a slowly progressive disease with chronic course, whilst WE has typically an acute, rapidly progressive course.

CASE REPORT

We describe the case of a 53-year-old, alcoholic male patient with simultaneous symptoms of subacute axonal polyneuropathy and less prominent clinical manifestations of WE. Symptoms including numbness of limbs, muscle weakness, gait impairment, and mental confusion had started one month earlier and then progressively worsened. Preceding gastrointestinal or airway infection was denied. At the time of clinical examination patient was unable to stand and walk and restricted to wheelchair.

Electromyography and electroneurography examination showed a subacute axonal sensory-motor polyneuropathy with evidence of florid active denervation in proximal and distal muscles in all extremities in the absence of significant reinnervation. A reduction in the amplitude of sensory(SAP) and compound motor action potentials(cMAP) was observed in the upper limbs, whilst both SAP and cMAP were unelicitable in the lower limbs (Table1, 2). Findings from laboratory tests(including folate and vitamine B12) and CSF were unremarkable. On MR imaging of the brain increased T2 signal of paraventricular regions of the thalamus and of periaqueductal regions of the midbrain were observed (Figure 1).

Based on clinical and instrumental findings of both axonal PN and WE, the hypothesis of a thiamine deficit was advanced. The patient was treated with administration of thiamine and clinical condition improved rapidly. In addition, intravenous immunoglobulin were also started during hospitalization as a dysimmune etiology could not be excluded. To date the patient remains under observation.

DISCUSSION

The patient here described presented a rare association of WE and subacute sensory-motor axonal PN. This clinical presentation is very rare and, to our knowledge, very few cases has been reported so far, and only one of these was due to alcohol abuse[2]. However, patients have been described in whom the peripheral nervous system involvement mimicked a Guillain Barrè Syndrome. This may pose a problem for the treatment choice as an immune-mediated pathophysiology cannot be rule out at all. In our case, according to previous reports, intravenous immunoglobulin treatment was done[3]. Another peculiarity of the present case refers to the subacute course of the polyneuropathy that occurred in strict temporal relationship to the other symptoms of WE.

CONCLUSION

The neuropathy seen in most alcoholics is usually chronic, with insidious onset, the physician needs to remember that neuropathy due to thiamine deficiency(TD) can also present as an acute process. Furthermore, though in developed countries the most common causes of TD are alcoholism and chronic disease, such as cancer, other subjects should be considered at risk. Among these patients with poor nutritional status on total parenteral nutrition, pregnant women with prolonged hyperemesis, patients after bariatric bypass surgery or suffering from anorexia. In all these cases, an early diagnosis of TD followed by adequate thiamine replacement is imperative to prevent and treat life threatening conditions such as WE and severe axonal polyneuropathy.

MOTOR NERVES	Lat[ms]	Amp [mV]	VC [m/s]	Amp % [%]	F-M [ms]
Sinistr Medianus					
Wrist-ABP	3,1	4,9			
Be Elb-Wrist	8,3	3,4	45,2	31	33,4
Sinistr Tibialis					
Ankle-AHB	5,9	0,3			
Knee-Ankle	14,7	0,5	43,2	43	48,4
Sinistr peroneus					
Ankle-EDB	---	---			

Table 1: Electroneurographic examination of the upper and lower limbs.

SENSORY NERVES	/Lat [ms]	Amp [µV]	VC[m/s]	Amp %[%]	Lat onset [ms]
Sinistr Medianus					
Dig II-Wrist	---	---			---
Sinistr Ulnaris					
Dig II-Wrist	1,94	7,0	54,1	43	2,9
Sinist Suralis	---	---			---

Table 2: Electroneurographic examination of the upper and lower limbs.

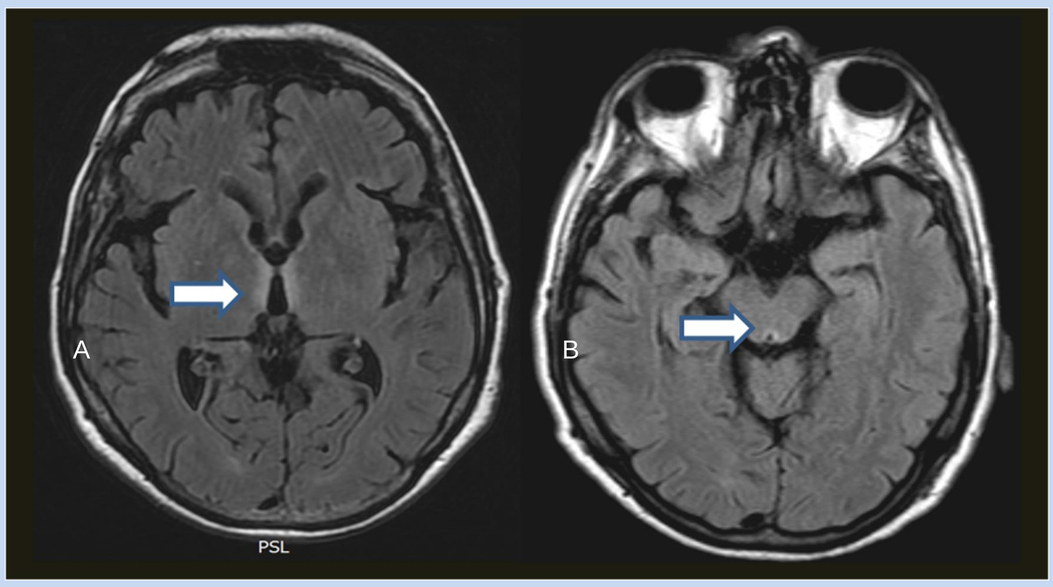

Figure 1: Axial RMI brain sections. A) abnormal hyperintensity in the mesial dorsal thalami. B) hyperintense signal in the periaqueductal gray matter.

References

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