Thalmic Vaculation in Acute Wernicke’s Encephalopathy

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Two patients with acute Wernicke’s encephalopathy, with the diagnosis confirmed pathologically at autopsy, showed substantial vacuolation and neuronal degeneration in discrete nuclei of the thalamus. Thalamic vacuolation has not been described previously in acute Wernicke’s encephalopathy. The use of frozen sections to minimize processing artifact was fundamental in demonstrating this pathology. The pathogenic mechanism underlying this change appears to be different to that seen in the more typical periventricular, mamillary body and brainstem lesions. We hypothesize that a specific neural pathway may be involved and suggest that this pathway could be the ascending nitric oxide-containing cholinergic pathway from the brainstem.

KEY WORDS: Wernicke’s encephalopathy, excitotoxic lesions, thalamus, vacuolation

INTRODUCTION

The neuropathological changes of Wernicke’s encephalopathy (WE) have been described in a number of large human studies with the topography of lesions and microscopic changes demonstrated in paraffin-embedded sections (Cravioto et al., 1961; Harper, 1979; Harper, 1983; Torvik, 1985; Victor et al., 1989). In this study, two well-documented cases of acute WE were chosen for serial section analysis of diencephalic and brainstem regions. The use of frozen sections revealed previously undescribed thalamic vacuolation expanding the neuropathological descriptions of acute WE.
MATERIALS AND METHODS

Case 1

This 51 year old man was admitted to a Sydney teaching hospital confused, agitated and tremulous. He had a long history of excessive alcohol intake (100-160 grams of alcohol per day) although three years previously he had been abstinent for 14 months. On examination he was disoriented in time and place, and had ophthalmoplegia, dysdiadokinesis and past pointing on finger nose testing. He was unable to stand alone so that gait disturbance could not be tested. The clinical diagnoses considered included acute WE and an alcohol withdrawal syndrome. He was commenced on intravenous fluids (4% dextrose and N/5 saline) with supplementary thiamine (100 mg intravenously). He was also given 30 mg diazepam over 3 hours. Investigations showed a normal blood profile and normal electrolytes. Liver function tests were slightly abnormal with elevated bilirubin and transaminases. Over the next two days he improved in that he was better oriented in time, person and place. He then began to vomit blood and bile and had a respiratory arrest, probably as a result of inhalation of vomitus. He died four days after admission.

Further questioning of the family and the general practitioner revealed that he had been an alcoholic most of his adult life. His diet was poor although he had taken multivitamin tablets from time to time. He had never previously demonstrated signs or symptoms of the Wernicke-Korsakoff syndrome.

At autopsy, the patient was found to have gastric erosions, fatty liver and evidence of inhalation with an early acute bronchopneumonia. Other abnormalities were restricted to the central nervous system.

Neuropathological Examination: - The brain was removed at 30 hrs post-mortem, weighed (1260g) and examined prior to fixation in 10% formalin for two weeks. After fixation, coronal brain slices were examined macroscopically and routine blocks of the left mammillary body, hypothalamus, thalamus, hippocampus, cerebellar vermis, frontal superior, parietal and temporal cortices were embedded in paraffin, sectioned at 8μm and stained with haematoxylin and eosin, cresyl violet and luxol fast blue. In addition, the whole diencephalon, basal forebrain and brainstem were sectioned on a freezing CO2 microtome into 50μm ‘thick’ sections. Four sections every 750μm were stained with cresyl violet, haematoxylin and eosin, luxol fast blue and the modified Bielschowsky silver stain.

Case 2 (Harper, 1981)

This 45 year-old woman was admitted to a Perth teaching hospital following a period of abnormal mentation, short-term memory loss and confusion. She had a two to three year history of heavy drinking (250-350 grams of alcohol per day), although her alcohol intake had been substantially reduced in the last three months. On examination, the patient was dehydrated and speaking in a confabulatory fashion, although her speech was well formed. Apart from tachycardia (pulse 120 beats/min) and a temperature of 38.5°C, no other abnormalities were detected. The clinical diagnoses considered included herpes simplex encephalitis and acute WE. She was commenced on intravenous fluids (5% dextrose) with no supplementary vitamins and was treated with adenine arabinoside and dexamethasone.