Mechanisms of carbamazepine-induced antidiuresis

Carbamazepine may cause water retention and hyponatraemia. Good's Textbook of Medicine, Harrison's Principles of Internal Medicine, The Textbook of Internal Medicine and the Oxford Textbook of Medicine attribute the mechanism to an excessive release of AVP secretion. They also found that both plasma AVP and AVP levels in the nephron were increased.

A reduction in vasopressinase activity is another possibility. These effects need not be mutually exclusive. The two case reports citing a central effect of carbamazepine found an increased AVP level with carbamazepine treatment, and the plasma AVP, AVP levels in the nephron are increased.

Meinders et al. found that the drug may stimulate the central release of AVP either directly or by altering the threshold of the hypothalamic osmoreceptors, while others claim that it is sometimes associated with a reduced AVP level and indicates that the mechanisms of water retention are not clearly understood. However, the Martin dale Extra Hypertensive notes that carbamazepine has an antidiuretic effect that is sometimes associated with a reduced AVP level and indicates that the mechanisms of water retention are not clearly understood. However, the Martin dale Extra Hypertensive notes that carbamazepine has an antidiuretic effect that is sometimes associated with a reduced AVP level and indicates that the mechanisms of water retention are not clearly understood.

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Sorenson et al. found no change in the basal plasma AVP response after carbamazepine exposure, but the plasma AVP response to AVP was decreased. Meinders et al. had previously reported a false plasma AVP with carbamazepine therapy and having found an antidiuretic effect of the drug on patients with cerebral diabetes insipidus, they concluded that the mode of action was renal and not central. When examining the relationships between plasma and urine sodium, osmolality, and AVP levels in normal subjects, Thomas et al. found that there was a close linear relationship between plasma AVP and AVP in urine osmolality. Meinders et al. had previously reported a false plasma AVP with carbamazepine therapy and having found an antidiuretic effect of the drug on patients with cerebral diabetes insipidus, they concluded that the mode of action was renal and not central. When examining the relationships between plasma and urine sodium, osmolality, and AVP levels in normal subjects, Thomas et al. found that there was a close linear relationship between plasma AVP and AVP in urine osmolality.

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Acute transient hydrocephalus in carbon monoxide poisoning: a case report

The pathological changes most commonly seen in carbon monoxide poisoning are in the region of the globus pallidus bilaterally, and less commonly in the medial temporal lobes and the calcarine white matter.
Pallidus hydrocephalus associated fourth ventricle. No involvement has been described.5 The unique anterior cerebral injury by carbon monoxide has hydrocephalus, bilateral cerebellar matter reported. It is since it does not diversion hydrocephalus of, most damage acute intoxication: poisoning. Hart 1962;15(Suppl):186. Graduate Medical Cunningham 1952;22:925. Hydrocephalus, hypoxaemia and acidosis and hypoxaemia which could have been continuous inflammatory changes in her CSF. Many of the relapses have been associated with pyrexia, and she has frequently demonstrated Lhermitte's sign and Uhthoff's phenomenon. There have been numerous separate episodes of neurological signs indicating lesions involving, the cauda equina on two occasions, the brainstem and cerebellum on four occasions, the spinal cord once, and the optic nerves bilaterally twice with three separate attacks of right optic neuritis. She had complex partial seizures and four major seizures controlled by treatment with carbamazepine.

On first admission a 10 day course of ampicillin and tetracycline was prescribed without benefit. Subsequently she has been treated with maintenance low dose prednisolone and pulsed methylprednisolone for relapses. Normal investigations have included: a complete infectious disease screen for all bacterial, viral and fungal causes (HIV antibody testing on two separate occasions was negative); a complete immunological and connective tissue disease screen, serum angiotensin converting enzyme levels, liver biopsy, Kvem biopsy, chest radiographs, cerebral angiography and repeated CSF for IgG, oligoclonal bands and cytology. Her HLA type is A1, A2, B8, B18, DR3 and DR4.

The main abnormality of the CSF was a variable lymphocytic pleocytosis in 11 lumbar punctures (range 6–268 cells). On three occasions the CSF protein was raised (0.60–0.70 g/l). Glucose levels have been normal.

During a 20 month period, five MRI brain scans have been performed. The first, a month after the initial presentation was normal. The second, three months later, showed an area of high signal in the right cerebellar hemisphere on T1 images and correspondingly an area of low signal on the IR scan. At 12 months a third MRI showed several areas of increased signal on T2 images; in the left middle cerebellar peduncle extending into the anterior aspect of the left cerebellar hemisphere, with a corresponding area on the right side, and also on the medial aspect of the right temporal lobe. These areas enhanced with intravenous gadolinium. Sixteen months after presentation a fourth MRI showed widespread T1 increased signal changes localised in the left fronto-parietal area involving both white matter and cortex, the left optic tract, the right temporal lobe, the corpus callosum and the right brainstem, extending into the right cerebellar peduncle. The areas involved were more extensive than those seen previously. Her most recent MRI scan, three months later, showed further new gadolinium enhancing lesions in the right temporal and parietal lobes, with resolution of some of the other lesions seen on earlier scans. The new enhancing right parietal lesion (fig) was biopsied stereotactically. Histology was non-specific with an excess of astrocytes in the cortex and more prominent in the white matter with oedema, narrow perivascular cuffs of myelin pallor and small numbers of foamy macrophages.

Neuropsychological investigation showed that the previously abnormal visual evoked responses (VERs) became normal following the first episode of bilateral optic neuritis. After further attacks of right optic neuritis,
Acute transient hydrocephalus in carbon monoxide poisoning: a case report.

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