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Editorial

Effect of Renal Complications on Brain - An Overview

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The association between chronic kidney disease (CKD) and cardiovascular disease has been extensively studied but, at the same time, little information has been available on the relationship between kidney and brain. In this manuscript, we have evaluated the recent updates on the relation between CKD and neurological disorders based on literature review and survey. A wide spectrum of neurological disturbances and behavioral changes such as fatigue, head ache, sensory clouding, vision impairment, seizures, delirium and coma can be observed in patients with renal complications [1-4].

One of the major problems arising due to renal failures is uremic encephalopathy. Uremic encephalopathy may occur in patients with either acute or chronic renal failure when the glomerular nitration rate falls below approximately 10% of normal. The severity of uremic encephalopathy is often dependent on the type of renal failure. In CKD, the symptoms are usually mild. The patient shows impaired concentration and behavioral changes; these symptoms are however not specific to encephalopathy, and are often associated with other diseases; major depressive disorder, hypertensive encephalopathy, or caused by drug side-effects, as a result, correct diagnosis is rarely possible. In acute renal diseases, the symptoms are more pronounced and more progressive. The severity of uremic encephalopathy correlates with the extent and kinetics of the accumulation of uremic toxins [5,6].

Experiments conducted on uremic animals have shown disturbances of the intermediary metabolism with increased levels of creatine phosphate, adenosine triphosphate and glucose; and decreased levels of monophosphate, adenosine diphosphate and lactate in the brain. We can also notice the inhibition of cerebral sodium potassium ATPase in experimental uremic animals. This results in the elevation of intracellular sodium which is responsible for cerebral dysfunction, particularly seizure activity. While the levels of certain biochemical compounds such as water, magnesium in the brain are normal; the level of calcium is found to be twice the amount. This increase in calcium levels indicates that parathyroid hormone may be involved in some way which facilitates the entry of calcium into the brain, leading to a relevant increase of calcium in the gray and white matter [3,5,6].

Studies have shown that many of the abnormalities that are found in uremic encephalopathy and calcium brain abnormalities can be reduced by parathyroidectomy; conversely these abnormalities can also be reproduced by this hormone to normal animals, while maintaining calcium and phosphate in the normal range. Thus, parathyroid hormone appears to be involved in the pathophysiology of uremic encephalopathy [6].

Dialysis encephalopathy or dialysis dementia is a progressive and often fatal disease, seen in patients of all ages but is common among children and elderly patients. In most of the cases, it leads to death in 6 months. Dialysis encephalopathy was first found by Alfred., *et al*. in the year 1972. It is believed that high levels of aluminum were the cause of dialysis dementia. Aluminum intoxication was probably first implicated in this disorder when studies showed that the aluminum content of brain gray matter in patients with dialysis encephalopathy was markedly elevated as compared with controls. The amount of aluminium in the brain was more than three times greater in patients with dialysis encephalopathy than in patients being treated with chronic hemodialysis who did not have dialysis encephalopathy [6,7].

The initial cases of dialysis encephalopathy were all of the endemic form and usually occurred in patients being treated with chronic hemodialysis for more than 2 years. The initial symptoms include dysarthria, apraxia, and slurring of speech, with stuttering and hesitancy. The patients may also have personality changes, psychosis, dementia, myoclonus, and seizures. The initial symptoms are usually intermittent and often worsen during dialysis. Treatment of dialysis dementia is usually be the chealation therapy whereas the preventive measures include usage of deionized water for the preparation of the dialysate. It has been reported that 20% of patients with chronic renal failure suffer from incapacitating disorder of restless leg syndrome. The carpal tunnel syndrome (CTS) has also been reported in CKD. It is characterized by a numbness of the volar first three fingers and hand, with paresis and atrophy of the thenar muscles [7-10].

The occurrence of subdural hematoma in the patients under hemodialysis is increased more than 20-fold when compared with the general population. Subdural hematoma and intracerebral hemorrhage and micro hemorrhages are discrete or isolated punctuate hypointense lesions smaller than 5 mm on T2∗-weighted MRI. These changes are connected with increased risk of intracranial hemorrhage and polyneuropathy [11-15].

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