Opinion

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Dialytic Approach in Hemodialysis (HD) Patients with Hydrocephalus

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ABSTRACT

Keywords: Hydrocephalus; Hemodialysis; Urea; Blood Flow Rate; Dementia; Gait Disturbance

Opinion

Cognitive dysfunction and neurological disturbances are common in Hemodialysis (HD) patients [1] and may be induced by chronic processes, acute events and a combination of both. Patients starting HD who develop acute neurologic disturbances, though measures to prevent the disequilibrium syndrome [2] were implemented, deserve special attention. Possible acute causes include ischemic and hemorrhagic strokes due to intra-dialytic hypotension, high blood pressure and/or anticoagulation on the background of the increased frequency and severity of diabetes mellitus, hypertension, atherosclerosis, arteriolosclerosis and atrial fibrillation in incident HD patients [3]. Falls, trauma, vascular disease, anticoagulation and polycystic kidney disease predispose to subdural and subarachnoid hematomas [4]. An additional acute cause for neurological symptoms, which is infrequent though reported to be more common in dialysis patients, is hydrocephalus [5]. Acute events at start of hemodialysis due to hydrocephalus may occur in patients with previous diagnosis of hydrocephalus or in previously undiagnosed patients.

Hydrocephalus consists of widening of the ventricles due to excessive accumulation of the Cerebrospinal Fluid (CSF). Obstructive hydrocephalus is due to disturbed CSF circulation and communicating hydrocephalus is due to impaired CSF absorption or uncommonly excessive production. Hydrocephalus can be with high or normal CSF pressure. Normal pressure hydrocephalus is a chronic adult-onset communicating hydrocephalus, usually caused by impaired CSF absorption that develops gradually usually in elderly patients [6]. Chronic hydrocephalus in adults can manifest as a triad of gait disturbance, the most common initial symptom, urinary incontinence and dementia, with or without symptoms and signs of increased intracranial pressure, such as headache, papilledema and localizing signs [6]. In addition to acute events at start of HD, increased risk of hydrocephalus, obstructive and even more common communicating, has been reported in Taiwanese dialysis patient cohort [5]. Explanations were that hydrocephalus may develop after subarachnoid hemorrhage and meningitis, which are more frequent in ESRD patients and may cause inflammation and arachnoid granulation fibrosis interfering with CSF absorption [7]. In addition, periventricular ischemic lesions associated with increased frequency of hypertension and vascular disease may cause weakening and dilation of the ventricles and increased local venous resistance, resulting in reduced CSF absorption [8]. Treatment of hydrocephalus consists of surgical ventriculo-peritoneal or lumbo-peritoneal shunts [9]. In HD patients, we suggest combining the surgical therapy with specific dialytic approach. Urea removal in HD induces a gap between blood and brain cells urea levels leading to increased brain cell water, which is typical in the disequilibrium syndrome during HD initiation [2].

Patients with hydrocephalus may be more susceptible to this adverse effect as previously reported [10]. In another patient

shorter and more frequent dialysis sessions along with intravenous mannitol resulted in satisfactory clinical response [11]. We reported a similar effect of correlation between the increase in intraocular pressure during HD and rate of urea removal manifested by post dialysis urea rebound and suggested to reduce blood flow and thus urea removal rates in HD patients with glaucoma [12]. We assume that even urea removal with only mild gap and increase in brain cell water during chronic HD, can exacerbate clinical picture in susceptible HD patients including those with hydrocephalus. Possible putative mechanism may be that even small increases in brain cell water may further interfere with CSF absorption and augment widening of the ventricles with excess CSF. Thus, in addition to the surgical procedures, reducing blood flow and urea removal rates and thus possibly the urea gap and the increase in cell brain water may be helpful to prevent and ameliorate symptoms in such patients.

Recently, we observed dialytic exacerbation of headache, vomiting and/or neurologic symptoms in 3 incident HD patients with known or newly suspected normal pressure hydrocephalus. Those patients were dialyzed after appearance of symptoms of headache and /or vomiting with blood flow of 200-225 instead of 300 ml/minute prolonging time of dialysis to assure adequacy of dialysis delivery. 1 patient was diagnosed with normal pressure hydrocephalus and underwent lumbar-peritoneal shunt placement 2 years prior to HD. 1 of the newly diagnosed patients underwent ventricular-peritoneal shunt placement, which was considered but not performed in the third patient. Reduced blood flow rate of 200-225 ml/minute was helpful in reliving symptoms. In my opinion, this approach may be favorable to using short frequent dialysis as previously reported [11], since the increase in brain cell water may be prevented, and not just stopped after a shorter time of HD.

In Conclusion, normal pressure hydrocephalus needs to be considered in HD patients with exacerbated headache, vomiting or the typical neurologic picture during HD. In addition to surgical procedures, reduced blood flow and urea removal rates may be helpful to prevent and ameliorate symptoms in these patients.

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