

Dialysis Disequilibrium Syndrome: The
Changes of Intracranial PressureKai-Chao Yang¹, Chih-Ju Chang^{1,2} and Cheng-Ta Hsieh^{1,2,3*}¹Division of Neurosurgery, Cathay General Hospital, Taiwan²Department of Medicine, Fu Jen Catholic University, Taiwan³Graduate Institute of Basic Medicine, Fu Jen Catholic University, Taiwan

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Abstract

Dialysis disequilibrium syndrome is a rare but well-known serious complication of dialysis. Cerebral edema associated with increase intracranial pressure has been considered to be main reason of dialysis disequilibrium syndrome. However, the direct evident of intracranial pressure changes in dialysis disequilibrium syndrome has been rarely described. Here, we reported a case of dialysis disequilibrium syndrome in a patient with acute stroke and intermittent hemodialysis. The changes of intracranial pressure levels before and during hemodialysis were analyzed and the results provided a significant evident to support the previous hypothesis.

Introduction

Dialysis Disequilibrium Syndrome (DDS) is a rare but well-known serious complication of dialysis [1]. A clinical deteriorated neurological symptom due to cerebral edema after hemodialysis is defined as the dialysis disequilibrium syndrome, which is similar to symptom that occurs with increased Intracranial Pressure (ICP) [2]. However, the direct evidence of increased ICP is rarely described in the clinical practices [3]. Therefore, we reported a case of DDS in a patient with acute stroke and intermittent hemodialysis. The direct ICP was analyzed and the relevant literature was also reviewed.

Case Presentation

A 47-year-old male developed an acute onset of weakness in the left extremities and slurred speech, following by unconsciousness. His past history revealed a 15-year hypertension with medication and chronic kidney disease on regular peritoneal dialysis for 7 years. At our Emergency Department, his body temperature was 37.5 degrees Celsius; pulse rate, 110 per minute; and respiratory rate, 18 per minute. Blood pressure was 210/110 mmHg. Neurological examinations revealed his Glasgow come score was E1M4V1. No pupil dilation was noted and the Babinski reflex was present in left side.

Laboratory examinations showed the white blood cell count was 5040 per cubic millimeter. Hemoglobin was 9.0 g per deciliter. Platelet count was 120,000 per cubic millimeter. Serum sodium was 131 millimole per liter and potassium was 2.9 millimole per liter. Blood urea nitrogen was 89 milligram per deciliter and creatinine was 12.5 milligram. No coagulopathy was noted.

The non-contrast Computed Tomography (CT) scan of the brain revealed a huge hematoma in right basal ganglion region with mass effect and midline shift (Figure 1A). Depended on the emergent condition, he underwent right decompressive craniectomy and the huge blood clot was subtotally evacuated. The ICP device was also inserted through the frontal parachyma.

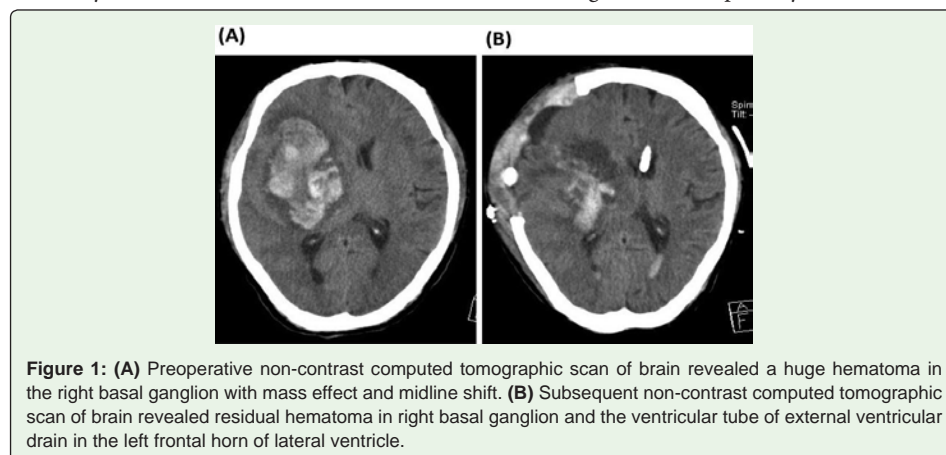


Figure 1: (A) Preoperative non-contrast computed tomographic scan of brain revealed a huge hematoma in the right basal ganglion with mass effect and midline shift. (B) Subsequent non-contrast computed tomographic scan of brain revealed residual hematoma in right basal ganglion and the ventricular tube of external ventricular drain in the left frontal horn of lateral ventricle.

Postoperatively, intermittent hemodialysis was arranged after consulting to neurologist. The therapeutic intervention was guided by ICP monitoring. Initially, the no fluctuated ICP was noted during first 4 time's hemodialysis (Figure 2A). However, since the fifth hemodialysis (one week after surgery), the patient became drowsiness during the hemodialysis and symptom improved after stopping hemodialysis. Otherwise, the increased ICP was obviously revealed during the hemodialysis (Figure 2B). The subsequent CT scan of brain revealed residual hematoma with cerebral swelling (Figure 1B). These ICP data collected from fifth to eighth hemodialysis were analyzed between the time before and during hemodialysis (Figure 2C). Before the hemodialysis, the mean ICP level was 9.9 ± 2.9 mmHg. However, the mean ICP level during hemodialysis increased to 22.7 ± 8.7 mm Hg, which achieved the statistical significance ($p < 0.05$). Otherwise, the decrease rate of urea and water revealed no significant contribution. The phenomenon of DDS was confirmed through the direct evidence of ICP changes.

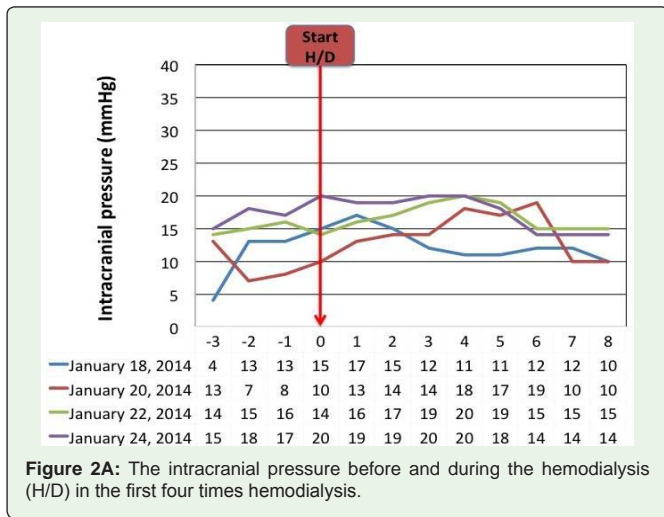


Figure 2A: The intracranial pressure before and during the hemodialysis (H/D) in the first four times hemodialysis.

Because of DDS, the hemodialysis was replaced by the peritoneal dialysis. No deteriorated neurological condition occurred again. The patient had clear consciousness, left facial palsy and left hemiparesis during the six-month follow-up period.

Discussion

DDS, firstly described in 1962 [4], is well-known complication during hemodialysis, which characterizes as nausea, vomiting, headache, visual acuity disturbance, tremor, muscle twisting, disorientation, confusion, seizure or even coma [5,6]. Several predisposing factors have been described to be associated with DDS, including the first hemodialysis, severe uremia, age, pre-existing neurological disorders, and metabolic acidosis [3]. "Reverse urea effect" has been posed as a main theory to induce DDS, because the shift of urea between brain intracellular space and plasma is not immediate but cause a brain intracellular space to interstitial osmotic gradient and leads to cerebral edema [7].

Clinically, the diagnosis of DDS is one of exclusion and should be distinguished from the similar symptoms and signs of intracranial hypertension resulting from subdural hematoma, uremia, nonketotic hyperosmolar coma, acute stroke, dialysis dementia, hypoglycemia or hyponatremia [1]. With the advance of neuro-monitoring, monitoring of ICP is the mainstay of care and helps clinicians to

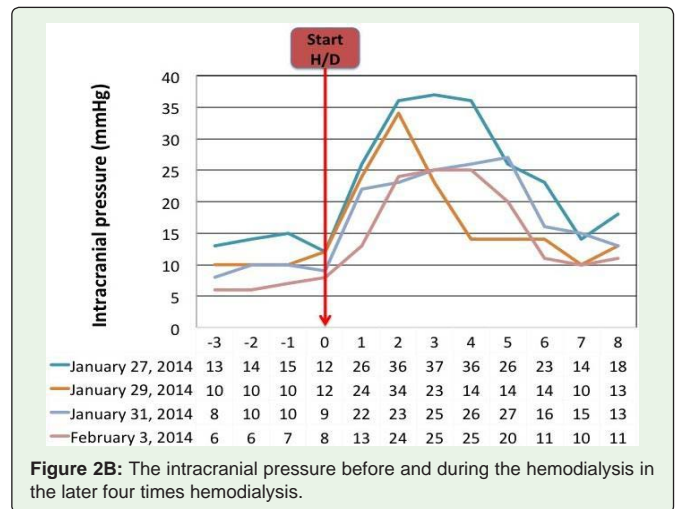


Figure 2B: The intracranial pressure before and during the hemodialysis in the later four times hemodialysis.

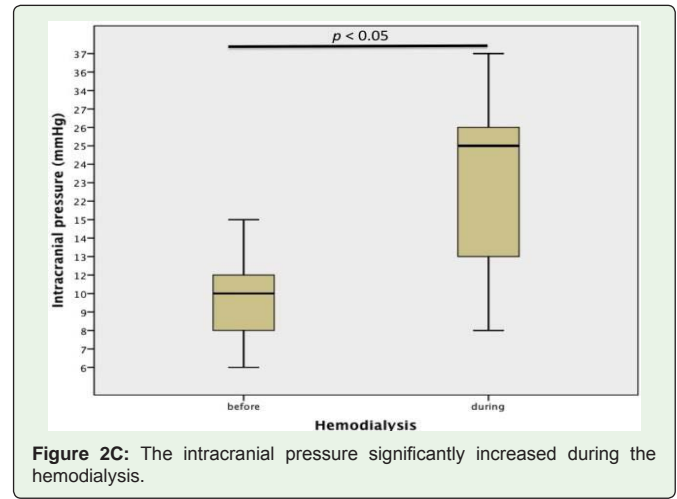


Figure 2C: The intracranial pressure significantly increased during the hemodialysis.

target therapeutic intervention [8]. Although cerebral edema after hemodialysis has been commonly considered as the main reason leading the acute deteriorated neurological condition, the direct evidence of ICP changes in DDS has been rarely reported [3,5,6,9,10]. In the review of literatures, only three spontaneous intracerebral hemorrhage cases developing DDS during hemodialysis have been briefly reported with the direct evidence of ICP changes [6,10], but only the trend of ICP was shown. No statistically analysis was not revealed, like our cases. To our best knowledge, this is first time to analyze the difference ICP levels before and during hemodialysis (Figure 2C).

First hemodialysis has been thought to increase the risk of DDS. In our case, no fluctuated ICP were shown in the first four times of hemodialysis. We thought that decompressive craniectomy might offer the available space for the brain edema in the early stage. Therefore, the DDS didn't occur in our patient during the first time hemodialysis. We also found that the ICP fluctuation during DDS did not increase in the first hour of hemodialysis, but elevated significantly within the second hours (Figure 2B). These results were similar with the study reported by Lin et al in 2008. Our case not only showed the confirmed diagnosis of DDS through the direct increased ICP, but also provided the significant changes of ICP from 9.9 to 22.7 mmHg during the hemodialysis. Our results provided a significant evident

to support the previous hypothesis that the DDS was associated with cerebral edema and increased ICP [11].

In the prevention of DDS during hemodialysis, a slowly gentle start of hemodialysis, increasing dialysate sodium levels, and administration of osmotically active substances have been described [1,3]. Otherwise, the continuous veno-venous hemofiltration or use of sustained low-efficiency dialysis, allowing gradual osmotic movement and mining the gradient between blood and cerebrospinal fluid, is also recommended to replace the hemodialysis and reduce the risk of DDS [3,10]. If the patients developed symptoms suggestive of DDS, slowing or stopping dialysis and the use of glycerol or mannitol to raise the plasma osmolality may be required and effective. Series studies including the CT scan of brain should be performed to exclude the other possible causes as mentioned above.

In conclusion, DDS should be considered as a cause of acute neurological deterioration in patients with acute stroke and intermittent hemodialysis. The ICP monitoring can offer direct evidence and target therapeutic intervention.

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