英文題目：Contrast-Induced Encephalopathy in Patients with End-Stage Renal Disease
中文題目：血液透析病患發生顯影劑導致之腦病變
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Case Presentation

A 64-year-old man of hypertension, diabetes mellitus, coronary artery disease and end stage renal disease (ESRD) was transferred for suspicious peripheral vascular disease. He had been receiving regular hemodialysis (HD) through the left forearm arteriovenous graft. He received twice coronary angiographies 10 months previously and no neurological abnormality was recorded. He underwent amputation of his left toes a month previously, but condition of wound did not improve. Hence, he was admitted for survey through lower-extremity angiography. In addition, he reported recently having unsatisfactory HD sessions with venous pressure elevation and difficult hemostasis, which implied graft occlusion.

He was clear and oriented during admission. Glasgow Coma Scale (GCS) score was E4V5M6. Laboratory data were within normal reference ranges. We performed computed tomography angiography of the lower extremity after infusion of 100 mL of iobitridol. Angiography showed total occlusion in left anterior tibial artery and severe stenosis in left superior femoral artery. Accordingly, he received balloon dilatation for these arteries. Angiography of left arteriovenous graft was performed after infusion of 150 mL of iohexol sequentially, which stenosis of left cephalic and innominate vein was shown. Thus, he received another balloon dilatation for central venous stenosis. A stable vital sign with a GCS score of E4V5M6 was recorded.

He exhibited irritation and disorientation with a GCS score of E2V2M4 two hours after procedures. We immediately performed neurological examinations, which showed symmetric undilated pupils, bilateral normal corneal and vestibulo-ocular reflex, negative Kernig's and Brudzinski's signs, absence of convulsion, and bilateral symmetric muscle power of extremities. Laboratory examinations revealed normal except mild hyperglycemia. Thus, we suspected contrast induced encephalopathy (CIE) and arranged for emergency HD two hours after his neurologic dysfunction.

HD was performed with dialysate calcium level of 3.0 meq/L for two hours. His consciousness improved to a GCS score of E3V3M4 after the first HD session. The second HD session was arranged on the third day of hospitalization. His GCS score before the second HD session was E2V3M4, which completely recovered to a score of E4V5M6 at the third hour of HD. We performed neurological examinations again and did not note any positive finding.

Discussion

CIE is defined as newly-onset cerebral dysfunction after exposure to a contrast agent. Diagnosis is made on basis of neurological symptoms, such as altered consciousness, seizure, blindness, or focal neurologic deficits, and exclusion of structural abnormalities. It is a rare event after intra-arterial administration of contrast agent. Studies have reported that factors such as old age, male sex, hypertension, diabetes mellitus, renal insufficiency, drug, and prior contrast exposure can be related to CIE. Mechanisms of CIE favored hyperosmolarity and chemotoxicity of contrast media, which may damage the blood–brain barrier and alter neuronal excitability in the brain. Imaging of CIE, which computed tomography or

magnetic resonance imaging is the preferred modality, would reveal subarachnoid hyperattenuation, cerebral edema, or localized prominent vascular markings initially and resolved in the follow-up imaging. HD is considered a standard therapy for CIE because it can efficiently remove contrast agents. However, dialysis regimen and recovery time had not been clearly defined due to rarity of reported cases.

Muruve and Steinman described the first CIE episode of a patient with ESRD in 1996. Previous reports of CIE in uremic patients are summarized in Table 1. These patients aged from 44 to 70 years, male sex predominant, and half of CIE episodes were reported after coronary angiographies. Onset of presenting neurologic dysfunction ranged from immediateness to 14 hours later. Initial symptoms included confusion, seizure, tonic deviation, agitation, headache and hemiplegic Dosage, osmolarity, and iodine content of contrast agents causing CIE ranged widely. Only one-third of these patients had reported prior exposure to a contrast agent, which might indicate CIE as an idiosyncratic reaction. All patients were eventually treated with HD, and symptoms of most patients resolved completely within several days.

In summary, we described a patient exhibiting irritation and disorientation 2 hours after receiving iobitridol and iohexol. He was afebrile without any infectious focus. Regular dialysis and laboratory examinations excluded uremia and electrolyte imbalance. On basis of neurological examination results, CIE was considered as the most possible diagnosis. His consciousness completely recovered after 2 HD sessions within 2 days. With advancement in caring, survival of patients with ESRD can be extended and more enhanced procedure including angiography may be performed for their comorbidities and complications. CIE is a rare and unexpected complication after intra-arterial contrast administration. Physicians should be cautious regarding patients' neurological status after their contrast exposure to imaging studies and provide appropriate management.