中文題目：以連續性腎臟替代療法成功治療顯影劑引發之神經毒性案例分享

英文題目：Successful Treatment of Contrast Neurotoxicity by Continuous Renal Replacement Therapy in a Critical Uremic Patient

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Introduction

Contrast neurotoxicity is an uncommon complication after intravascular contrast media administration. Here we report a 73-year-old woman with end stage renal disease, under regular hemodialysis who was admitted for management of ruptured aneurysm at right subclavian artery with the common carotid artery fistula occlusion. After angiography with a 10x40 mm-sized stent placement, tonic-clonic seizure developed with loss of consciousness 11 hours after the procedure. Due to unstable vital signs, continuous venovenous hemofiltration was arranged to avoid hemodynamic instability. Emergent brain CT revealed increased leptomeningeal enhancement in the right fronto-temporal region and subsided without evidence of intracranial hemorrhage (ICH) or aneurysm only 4 hours after CVVH, which supported our diagnosis of contrast-induced neurotoxicity. Though this case we highlights that physician should beware of contrast induced encephalopathy, especially those who has chronic renal failure, and hemofiltration via continuous renal replacement therapy was a reliable procedure to remove contrast media rapidly and help patient to recovered from contrast-induced neurotoxicity.

Case Presentation

A 73 year-old woman with a history of end stage renal disease under maintenance hemodialysis (HD) for 3 years was admitted due to a ruptured aneurysm at right subclavian artery adjacent to the junction with the common carotid artery, which was

diagnosed by angiography. On the second day of hospitalization, a 10x40 mm-sized stent was placed to repair this lesion. On day 3, however, tonic-clonic seizure started from the left extremities, with loss of consciousness; while high blood pressure (200/110mmHg) was noted within a few hours after the stenting procedure. Her initial Glasgow coma scale was E2V2M4, and the immediate non-contrast computerized tomography (CT) of the brain showed increased leptomeningeal enhancement in the right fronto-temporal region (Figure. 1).

Because of her unstable condition, CVVH rather than HD was arranged to avoid hemodynamic instability on day 4. The following CT angiography of the brain showed no evidence of intracranial hemorrhage (ICH) or aneurysm (figure. 2) after only 4 hours of treatment by CVVH, supporting the diagnosis of contrast neurotoxicity. Remarkably, the patient's consciousness became clear without any neurological deficit and she was transferred from ICU to nephrology ward on day 7. Finally, she was discharged under stable clinical condition on day 10 and had an uneventful follow-up period at our outpatient department for more than 2 years.

Figure. 1

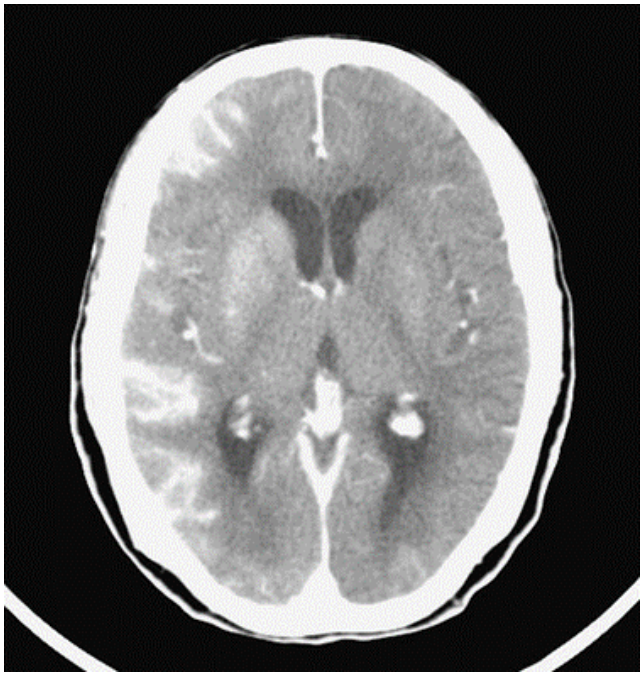
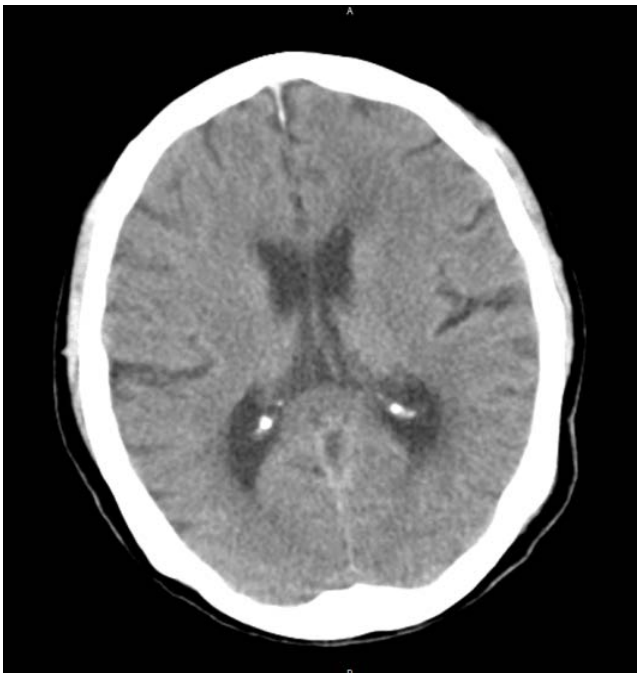


Figure. 2



Discussion

Contrast-related neurotoxicity may mimic ICH and subarachnoid hemorrhage (SAH) both clinically and radiologically. Several situations may present as "pseudo-SAH" in images, including hyponatremia, anoxic injury, diabetic ketoacidosis with metabolic encephalopathy, diffuse cerebral edema and septic shock1-2. Pseudo-SAH usually manifests as reversible neurological deficit such as cortical blindness, amnesia, seizure attack, hemiplegia or unconsciousness because of the stimulation of neural cells caused by the extravasation of contrast media which may result from the increase in the permeability of blood brain barrier on account of shrinkage of cells and opening of tight junctions due to the hyperosmolality of the contrast agents 3-4. However, the volume of contrast medium needed to induce such changes is unknown, as the amount administered varies widely in published reports. Restricting the amount may reduce the risk of extravasation5, while renal insufficiency may increase it. In our patient, the unilateral leptomeningeal enhancement resulted from uremia-related vascular hyperpermeability superimposed by the large dose (150 ml) of contrast medium (iohexol) administered via the right common carotid artery during the stenting procedure. Furthermore, prolonged contrast accumulation was caused by concurrent stasis of venous flow due to the compression of the right internal jugular vein by hematoma coming from the ipsilateral ruptured pseudoaneurysm, leading to neurological symptoms on account of contrast-induced hyperosmolality. Fortunately, CVVH can remove iohexol, a water soluble non-ionic contrast medium with a molecular weight (MW) of 821 Daltons in a very efficient way similar to vitamin B12 (MW=1335 Daltons) with a sieving coefficient and clearance of which at 100% and 37 ml/min respectively by the hemofilter we used under a blood flow rate of 200 ml/min6.

In conclusion, the differential diagnosis between contrast neurotoxicity and ICH/SAH is a major clinical challenge. Hopefully, the increased awareness of this unusual adverse event will not only reduce unnecessary radiological investigations but also facilitate prompt and adequate treatment of this serious clinical complication.

References

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