Intradialysis stroke

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case report

Abstract:

This case report showed an unusual complication arising from managing kidney failure patient. Haemodialysis procedure in most patients with kidney failure lowers their blood pressure. However, some patients exhibit paradoxical increase in BP during dialysis. There is considerably higher prevalence of clinical cardiovascular disease in the dialysis patients compared with the general population; this had been documented way back in 1974. Chronic kidney disease is associated with accelerated atherosclerosis, and high cardiovascular mortality in this group of patients may be attributed to this atherosclerosis. There is also vascular remodelling, which is characterized by: proportional increase in arterial diameter and wall thickness, this may be due to pressure overload which leads to wall hypertrophy and an increase in wall to lumen ratio or flow overload.
Ureamic vasculature predisposes dialysis patients to further injury by the presence of Framingham risk factors such as anaemia, inflammation, oxidative stress, metabolic alterations, sympathetic over activity, electrolyte disturbances and vascular calcification, and thus predisposing them to stroke. Intra dialysis cardiovascular stroke is not common in this environment; we therefore presented a case of stroke during dialysis in a patient with chronic kidney disease.

Keywords: Haemodialysis, Chronic kidney disease, Hypertension, atherosclerosis.

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Intra dialyse course

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rapport de cas

Résumé:

Ce rapport de cas a révélé une complication inhabituelle résultant de la gestion une insuffisance rénale du patient. Procédure L'hémodialyse chez la plupart des patients avec une insuffisance rénale abaisse la pression artérielle. Cependant, certains patients présentent paradoxal augmentation de BP au cours de la dialyse. Il est considérablement plus forte prévalence clinique des maladies cardiovasculaires dans les patients en dialyse par rapport à la population générale, ce qui a été documenté en 1974. Maladie rénale chronique est associé à l'athérosclérose accélérée, et la haute mortalité cardiovasculaire dans ce groupe de patients peut être attribuée à cette l'athérosclérose. Il y a aussi remodelage vasculaire, qui est caractérisée par : augmentation proportionnelle de diamètre artériel ou l'épaisseur de la paroi, cela peut être dû à une surcharge de pression qui mène à mur hypertrophie et une augmentation de mur de lumen ratio débit ou surcharge.
Ureamic vascularisation prédispose les patients dialysés à d'autres blessures par la présence de Framingham facteurs de risque comme l'anémie , de l'inflammation, stress oxydatif, altérations métaboliques, compatissant à activité, perturbations électrolytiques et calcification vasculaire, et donc les prédispose à la course. Intra dialyse course cardio-vasculaires est pas commun dans cet environnement; nous avons donc présenté un cas de course au cours de la dialyse dans un dossier de patient avec maladie rénale chronique.

Mots-clés: l'hémodialyse, maladie rénale chronique, l'hypertension, l'athérosclérose.

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Case Report

W.U was a 43 year old man, known hypertensive, diagnosed ten years ago. He was referred from a secondary medical facility, on account of generalized body weakness of three weeks duration, progressive abdominal and leg swelling, progressive reduction in urinary output of two weeks duration and persistent vomiting and hiccups of 7 days duration. There was history of poor drug compliance. He was not previously diagnosed to have diabetes mellitus. There was positive family history of hypertension in the mother and one of his siblings. There is no family history of kidney disease. There was significant history of herbal medicine intake. He also took 36-42g of alcohol daily for fifteen years before he stopped five years ago on religious ground.

Examination revealed a middle aged man in altered level of consciousness, pale, in obvious respiratory distress and has bilateral pitting pedal oedema extending to the knee. The blood pressure was 180/120mmHg. There was fine bibasal crepitation and also there was asterixis.

The full blood count showed leucocytosis white blood cell (WBC) 12.2x 10^9, packed cell volume 19%. The urine was turbid, and there was 2+ proteinuria. Urine microscopy, culture and sensitivity revealed WBC 8-10/hpf, pus cells 5-8/hpf but the culture yielded no growth. The results of investigations are depicted in Table 1 The kidneys were normal in their positions. Right kidney 10.4 x 5.4cm, left kidney 10.4 x 4.3cm they have regular outline. The parenchymal echogenicity was increased. The corticomedullary differentiation was poor.

. An assessment of ureamic encephalopathy, secondary to chronic kidney failure was made, precipitated by urinary tract infection.

He had two sessions of haemodialysis within three days, and got better. Patient subsequently discharged himself against medical advice because he did not want blood transfusion on religious ground. He was managed on outpatient basis.

A week after, he represented in emergency room symptomatic of uraemia. His PCV then was 18%. He was managed with tabs amlodipine 10mg daily, methyl dopa 500mg B.D, intravenous furosemide 40mg B.D, s.c. erythropoietin 4,000i.u thrice weekly and was also dialysed. He was discharged home a week after. He represented a month after with breathlessness, general body weakness all of a week duration and recurrent vomiting of two days duration. The results of the electrolytes and urea showed Na 147mmol/L, potassium 4.2mmol/L, HCO_3 15mmol/L, urea was 73.9mmol/L and creatinine 1007.8μmol/L . The blood pressure was 180/120mmHg.Tabs aldomet 500mg was given and an hour later his blood pressure was 160/100mmHg. While on dialysis, he was noticed by the nurses to have suddenly become restless and agitated, and developed right sided weakness.

On examination, he was conscious and alert, no sign of meningeal irritation. The power in the right upper limb was zero while right lower limb was 3. There was hypertonia and hyperreflexia on the right limbs, while the power, tone and reflexes were normal on the left limbs. The pulse rate was 116bpm; blood pressure was 200/150mmHg. An assessment of left hemispheric cerebrovascular disease most likely haemorrhage in the middle cerebral artery territory was made. The dialysis session was terminated and he was commenced on cerebral decompression with mannitol and furosemide.

The brain scanogram was within normal limit. Serial axial non – contrast slices demonstrate an irregular hyperdense focus in the left cerebral hemisphere, located in the region of the posterior limb of the internal capsule with extension to the external capsule. A much smaller focus is noted above it in the anterior internal capsule. Minimal vasogenic oedema surrounds it. The ipsilateral thalamus (left) is compressed. There is attenuation of the left lateral ventricle especially the body and trigone with minimal mass effect. The cerebellar hemispheres are intact. The base of skull and cranial vault are also within normal limits. A diagnosis of Acute left cerebral haemorrhage secondary to haemorrhagic stroke was made.

Discussion:

The risk of stroke in chronic kidney disease (CKD) patients is high (4). CKD patients on dialysis as compared to the general population had five times higher risk of developing stroke

(4). CKD and dialysis population also had higher death rate from stroke (4,5,6). Stroke was found to be one of the leading causes of death, accounting for 12.7% of total CKD-related deaths in a study from Japan (5).

The incidence of ischemic stroke was 5.3 times higher than that of hemorrhagic stroke in end stage kidney disease as reported by the United States Renal Data System (USRDS)(11). Some of the reasons reported for the increased incidence and recognition of brain infarcts in CKD patients were: inclusion of older patients with multiple risk factors for haemodialysis, usage of erythropoietin and advanced diagnostic facilities in the form of magnetic resonance imaging (MRI)(2). However, the age range of our patients on dialysis is in the 3rd and 4th decades of life (6) in this environment.

The high prevalence of hypertension, proteinuria, malnutrition, and hypoalbuminaemia, which directly affected erythrocyte deformability and endothelial dysfunction, may be responsible for increased incidence of cerebral haemorrhage in CKD and dialysis patients (7). The thalamic and basal ganglia region are the commonest site for cerebral haemorrhage (4), as in this case. Cerebral haemorrhage usually occurs 35.5 hour after the last dialysis whereas brain infarcts were frequently found to occur during or shortly after dialysis procedures (8). Increased risk of haemorrhagic stroke may be due to routine administration of heparin during haemodialysis combined with ureamic bleeding diathesis (9). In this patient, the blood pressure control was also poor, and this may explained why he had the stroke intra dialysis. Ureamic process is a non traditional risk factor for stroke (8), and the dialysis process on its own (10) may worsen the probability of the patient undergoing dialysis to develop stroke. The elevated serum creatinine was a strong and independent predictor of the increased risk and adverse outcome after stroke. This patient's serum creatinine was very high. This patient was under dialysed, partly because of the anaemia and finance. The anaemia management was suboptimal because of his non acceptance of blood transfusion. One of the markers of generalized vascular disease is serum creatinine (7,8).

Education and sensitization of the populace will be important for optimum outcome and to reduce this kind of complication. It is also important to have intradialysis electrocardiographic monitoring of our patients.

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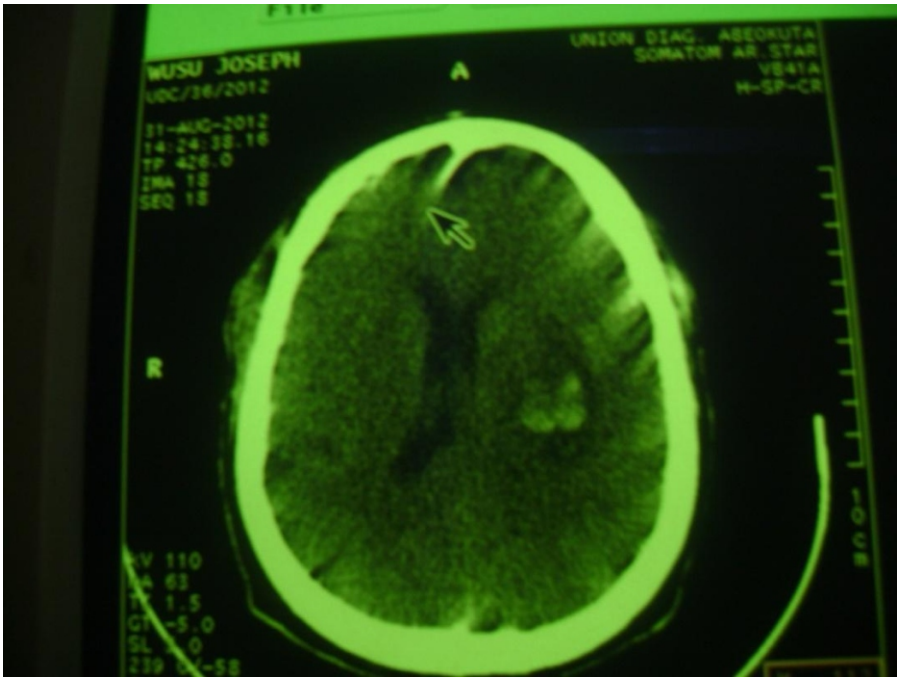

Fig I: Corona section of the brain, with displaced cerebra falx to the right

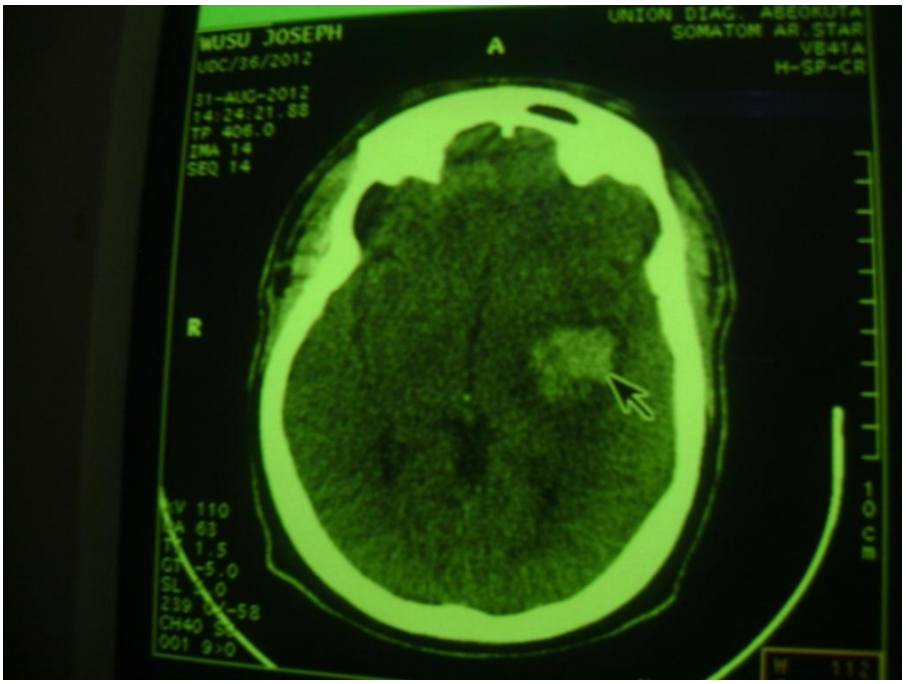

Fig 2: Corona section of the brain showing hyperdense lesion at the level of the 3rd ventricle

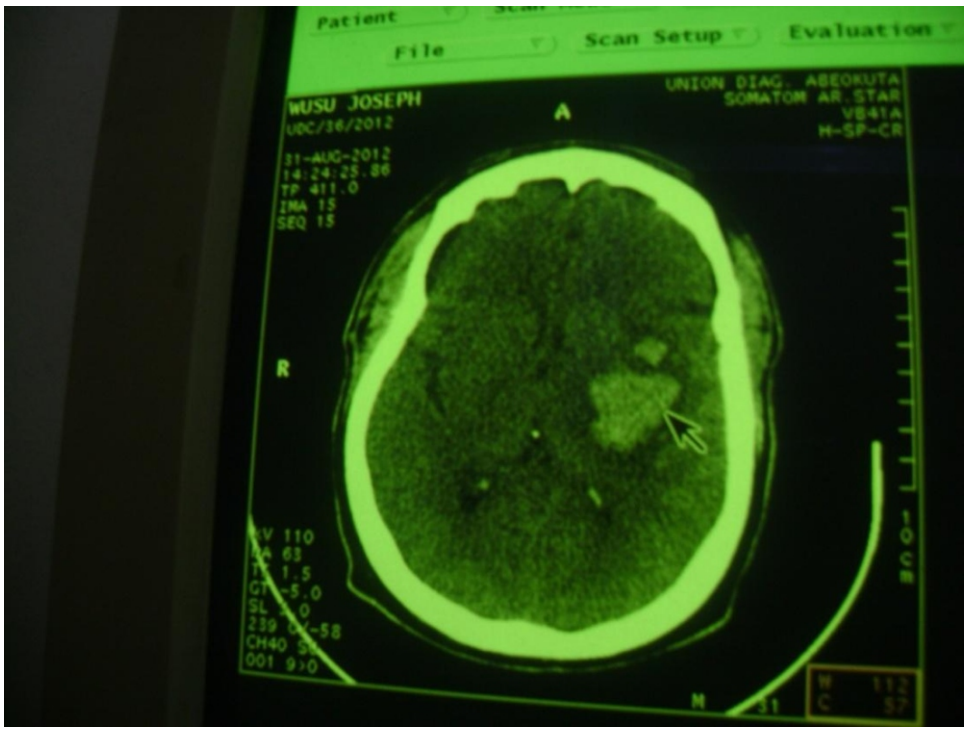

Fig 3: Corona section of the brain showing the hyperdense mass in relation to the posterior limb of the left lateral ventricle

Table 1: Results of serum electrolytes, urea and creatinine

Electrolyte	15/7/2011	17/7/2011	17/7/2011
Sodium	136mmol/L	137mmol/L	139mmol/L
Potassium	4.8mmol/L	5.6mmol/L	4.2mmol/L
$HC0_3$	18mmol/L	18mmol/L	20mmol/L
Urea	106.4mmol/L	151.4mmol/L	96mmol/L
Creatinine	1060.8µmol/L	2006.7µmol/L	795.6µmol/L