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The Unexpected Deadly Shift

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INTRODUCTION

Dialysis disequilibrium syndrome (DDS) is a syndrome characterized by wide range of neurologic symptoms attributed to cerebral oedema[(2)]. It occurs either to patients when they first start on dialysis or miss multiple consecutive dialysis. Clinical manifestation ranges from mild to severe which can be fatal as illustrated in this case[(4)].

CASE PRESENTATION

A 14-year-old lady with advanced chronic kidney disease presented to emergency department with menorrhagia and worsening shortness of breath for the past 2 weeks.

Upon arrival patient was alert, pale and tachypneic. Clinical examination revealed signs of fluid overload such as lung crepitations and bilateral lower limb edema. Her laboratory parameters showed acute kidney failure with severe metabolic acidosis and normochromic normocytic anemia (refer to Table 1).

Patient was started on Helmet Continuous Positive Airway Pressure (CPAP) and given intravenous frusemide for diuresis. Hemodialysis was performed at the emergency department with transfusion of blood. However, 2 hours into hemodialysis, she became unresponsive and was intubated for airway protection.

Urgent Computed Tomography (CT) brain showed generalized cerebral and cerebellar oedema with tonsillar herniation. In view of the severe brain pathology (Figure 1), the patient was treated conservatively with mannitol infusion. Patient succumbed to her illness the next day.

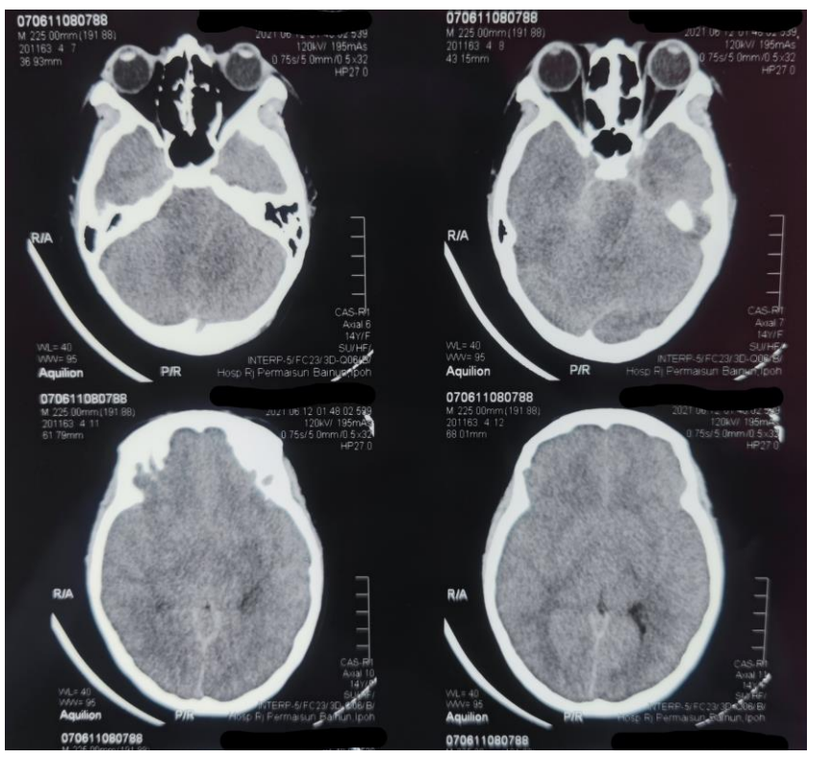

Figure 1: CT Brain cut section showing generalized cerebellar and cerebral edema

DISCUSSION

Dialysis disequilibrium syndrome is a clinical diagnosis and there is no specific test for it. The exact epidemiology is rarely reported and not established[(5)].

In this case patient developed severe DDS (stupor and coma) due to the development of generalized cerebral and cerebellar oedema.

Several pathogenesis had been proposed such as dialysis causing swift removal of urea (Table 1) which exerts osmotic force on brain cells[(1)]. In addition, a paradoxical acidemia (fall in pH) of the cerebral spinal fluid (CSF) occurs due to increased organic acid production in the brain and when systemic metabolic acidosis is rapidly corrected. This increase in brain osmole content leads to an increase in the brain water content which is the hallmark of DDS.

Measures that could have been taken to limit the effect of cerebral oedema include[(6)]:

1) reducing clearance so as to lessen the reduction of plasma osmolality,
2) increasing the time over which clearance is performed and
3) adding another osmotically active agent like sodium or mannitol as urea is removed by hemodialysis, so that plasma osmolality does not change significantly.

Several challenges that we can identify from this case are:

a) Difficulty in early recognition of sign and symptoms of DDS as patient had concurrent uremia.
b) DDS is not anticipated in this patient with severe uraemia.
c) Constant monitoring for dialysis patient in a busy emergency department setting.

Serial Blood Investigation				
Date	**11/06/2021**	**11/06/2021**	**11/06/2021**	**12/62021**
Time	**14:00H**	**01:30H**	**03.30H**	**06:50H**
Full blood count				
TWC	**10.1**	**11.3**	**15.5**	**5.2**
Hb	**4.5**	**8.6**	**8.7**	**5.2**
HCT	**12.5**	**23.8**	**24.5**	**14.4**
Platelet	**36**	**117**	**124**	**36**
Renal Profile				
Urea (mmol/L)	**114.3**	**32.8**	**44.7**	**46.8**
Creatinine (umol/L)	**3520**	**1018**	**1437**	**1642**
Sodium (mmol/L)	**141**	**134**	**144**	**144**
Potassium (mmol/L)	**6.1**	**2.7**	**3.0**	**3.2**
Chloride (mmol/L)	**110**	**95**	**106**	**107**
ABG				
pH	**7.184**	**7.278**	**7.320**	**7.376**
Pco2 (mmHg)	**14.7**	**50.90**	**36.40**	**23.30**
Po2 (mmHg)	**139**	**80.8**	**135.2**	**187.1**
HCO3- (mmol/L)	**5.4**	**23.3**	**18.3**	**13.40**
Base excess	**-22.90**	**-3.5**	**-7.8**	**-11.80**

Table 1: Serial blood investigations

CONCLUSION

In summary, prevention is the mainstay therapy to prevent DDS in a patient with severe uraemia undergoing initial hemodialysis therapy. Measures should been taken to slow down the change of plasma osmolality which can increase the risk of cerebral oedema. Constant monitoring during dialysis is important to recognize the development of this syndrome.

ACKNOWLEDGEMENT

The authors would like to acknowledge Hospital Raja Permaisuri Bainun Ipoh (HRPB) for providing us study facilities.

DECLARATION OF CONFLICT

The authors whose names are listed certify that they have NO affiliations with or involvement in any organization or entity with any financial or nonfinancial interest.

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