Psychiatric Manifestation in a Child with Uraemia: A Case Report

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INTRODUCTION

Adequate renal function is required for normal mental function. Hence deterioration in renal function could result in neuropsychiatric effects ranging from subtle, non-specific changes to gross distinct abnormalities. [1] When there is a severe reduction in glomerular filtration rate (GFR) either acutely or chronically, uraemic encephalopathy occurs [2]. The rate at which uraemic encephalopathy develops is proportional to the rate at which GFR falls. Acute renal failure precipitate a rapid and florid failure of mentation, whereas in chronic renal failure, it slowly evolves. [3] All aspects of psychic can be affected including cognition, psychomotor activity and personality. [4] However, the overt personality disorder manifesting in neuropsychiatric disorder are not often seen. The few that have been reported have been in adult. We therefore report a 15 year old child who presented with features suggestive of nephrotic syndrome which later progressed to acute renal failure and subsequently developed neuropsychiatric symptoms in the course of the uraemia.

Keywords: *Uraemia, mania, African child*

CASE REPORT

A 15 year old Nigerian boy was referred from a secondary health facility with 3 month history of recurrent body swelling. There was initial diurnal variation but became constant throughout the day two weeks to presentation. There was no reduction in urine output and no haematuria. There was preceding history of scorpion sting twice three days before onset of the most recent swelling. No history of using mercury containing soap, preceding sore throat or skin rash. He was taken to the referring hospital for the first two episodes only to recur again for the third time.

Examination revealed anasarca, he was not pale or jaundiced. The blood pressure was 130/ 84mmHg. There was massive ascites with no renal angle tenderness. Urinalysis revealed proteinuria of 4+ and blood was negative. Serum cholesterol was 9.5 mol/l, serum protein was 49g/l, serum albumin 13g/l, urea 8.2mmol/l, creatinine 110µmol/l potassium 3.1 mmol/l, sodium 133mmol/l, calcium 1.5 mmol/l, phosphate 2.1mmol/l, PCV -23%, 24hour urinary protein 2.29g/day. Abdominal ultrasound revealed that both kidneys were normal in size and outline with slight increase in echo and moderate calyceal fullness suggestive of grade II parenchyma disease.

An assessment of nephrotic syndrome was made and he was commenced on parenteral frusemide, hydrochlorothiazide and spironolactone. The edema resolved only to recur again with similar fluctuation in urine output between 0.6-1.9ml/kg/hr during the first 40 days of admission.

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Oral prednisolone (60mg/m^2) was commenced on the 27th day on admission when oedema had subsided significantly. It was stopped on the 30th day on admission when a resurgence of oedema was noticed again accompanied with further reduction in urine output.

On the 35th day, the blood pressure was noticed to be rising and blood pressure of 120/100 mmHg was recorded. On the same day, he started to talk to self, talk excessively and sing loudly on the ward. His sleep became grossly inadequate, was aggressive and hyperactive and had to be restrained physically. There was no episode of convulsion or loss of consciousness. He had no past episode of mental illness and there was no family history of mental illness.

Mental state examination revealed a boy who was conscious and alert, restless and uncooperative. He had irritable mood and affect, was fully oriented to time, place and person but attention and concentration was poor. His uncooperative attitude made it difficult to ascertain presence of perceptual abnormality but his behaviour was suggestive of someone having visual hallucination. There was marked pressure of speech and it was difficult to interrupt his conversation. He occasionally had flight of ideas which made it difficult to know what he was saying. Low dose haloperidol (antipsychotic drug) was added to his medication, to which he initially responded positively.

Serum creatinine rose to 247µmol/l and urea was 11.9 mmol/l, potassium 2.3 mmol/l and sodium 131mmol/l. There was also recurrence of oedema requiring plasma transfusion. Blood pressure became 140/100mmHg. Hypertensive/uraemic encephalopathy were suspected. He was therefore commenced on captopril. Subsequently talkativeness, aggressive behavior and insomnia continued to worsen in spite of haloperidol, benzhexol and diazepam. The urine output declined to as low as 0.3ml/kg/hr on the 46th day (about 10 days after commencement of irrational talk). Meanwhile, proteinuria remained between 3 and 4+ since admission. In view of the declining urinary output and refractory oedema despite the administration of diuretics and plasma transfusion, renal replacement therapy was commenced on the 50th day. There was subsequent improvement in urine output to as high as 4.1 ml/kg /hr on the 60th day after 4 sessions of dialysis. However, some oedema still remained and the aggressive behaviour persisted. Proteinuria reduced to 2+ and creatinine reduced to 121µmol/l. The patient was discharged home on the 68th day, when they could no longer afford further dialysis on captopril, nifedipine, diuretic and antipsychotic drugs. On follow up about three months after discharge, he was withdrawn and sluggish in responding to questions. Oedema had reduced but with some residual leg oedema up to the distal third of both legs. Blood pressure was 130/90mmHg. At the 5th month post discharge all the psychotic symptoms had disappeared and he was no longer withdrawn, responded normally to questions and oedema had fully subsided. Proteinuria was 2+ and BP was 120/70mmHg. All the diuretics were stopped and he was placed on only lisinopril 10mg daily. Serum creatinine was 42 µmol/l, urea 4.2 mmol/l, potassium was 4.2 mmol/l, and sodium was 137 mmol/l. Serum protein was 57g/l, albumin was 28 g/l. He remains stable on anti-psychotic medication and still being followed up in the psychiatric outpatient clinic.

DISCUSSIONS

The clinical feature of this case did satisfy the criteria for mania due to a general medical condition (uremia) or secondary mania. [6] Uraemic encephalopathy was suspected, but the absence of confusion and disorientation made it unlikely. [7] Furthermore, the psychiatric symptoms predated the commencement of dialysis thereby ruling out dialysis disequilibrium syndrome. For similar reasons, dialysis dementia which is a known cause of secondary mania was also out of consideration. [8] The possibility that the commencement of steroid could have triggered the psychiatric symptom was also not likely because it had been discontinued 5days before the manifestation of those symptoms and it was administered at the standard dose for just three days after which it was discontinued due to resurgence of oedema. A familial link could also not be established because of absent family and previous history of psychosis in this child. There was no family separation as the father and mother were together providing support for him. The extended family also provided ready financial support for the dialysis sessions received.

Manic manifestations found in this patient was in keeping with the report of other workers even though those reports were in adults who had all commenced dialysis. [8 - 11] Our patient had not

commnenced dialysis before the symptoms were manifested and he is the first child known to us with that manifestation.

There are several aetiological factors that should be considered in the pathogenesis of this organic manic syndrome. Since the disorder was precipitated by renal failure due to a glomerulopathy, it has been suggested that the metabolic derangement of uraemia include a factor present within the serum that reduces the activity of the sodium-potassium pump within neuronal membranes leading indirectly to increased neurotransmitter release and the production of mania [9]

The aetiology has also been linked to the reduced clearance of neurotoxins such as ammonia, middle molecular weight toxins (which are not effectively removed by haemodialysis (HD), but better removed by peritoneal dialysis) and porphyrins. Psychiatric disturbances are also strongly correlated with hyperparathyroidism which is a common occurrence in renal failure. It is mediated in part by hyperphosphataemia that attends renal failure and decreased degradation of parathormone [12- 13] The incidence of psychiatric disturbance in patients with primary hyperparathyroidism has been estimated to range from 3-67% depending on the type of findings which can range from mild apathy to personality changes to florid psychosis[14]In secondary hyperparathyroidism, the psychiatric manifestation is qualitatively similar. [15]

As found by other workers neuropsychiatric manifestations of uraemia are possible and plausible in view of the uraemic toxin which could be an irritant to the brain, hence the uraemia is likely to be the trigger for the manic illness in our patient. What was however surprising to us was the failure of the neuropsychiatric manifestation to resolve after the correction of the uraemia with the series of dialysis session. It may be possible that the dialysis compounded the problem. It may also be possible that dialysis does not completely clear all the neurotoxin since many molecules are involved [16] and some of these molecules are susceptible to differential clearance depending on the mode of dialysis with peritoneal dialysis (PD) generally been regarded as more effective. Indeed the type of dialysis used affects the neuropsychiatric presentation. Given the apparent susceptibility of the middle molecules to clearance by PD, it remains an important potential cause of psychiatric illness. Another possibility is the fact that it will take time for the after effect of the toxin to wear out like it occurs in tetanus. In fact the psychiatric symptoms in our patient continued unabated after the few sessions of dialysis he had and needed continuous antipsychotic therapy until it fully subsided about 5 months after discharge. He is still been managed by the Psychiatrist and on antipsychotic medication.

The serum level of creatinine in our patient though elevated did not seem significantly high enough to produce severe problem such as a neuropsychiatric manifestation. This raises the question of at what level of uraemia should one anticipate neuropsychiatric manifestation.

Finally, uraemia is a stressor, needing multiplicity of interventions some of which have attendant side effects and could run a chronic course which is enough to cause a psychological disturbance. The totality of the effect of uraemia is therefore capable of producing neuropsychologic imbalance which could cause neuropsychiatric manifestation which may have been the case in our patient. We recommend an early neuropsychologic evaluation for all children with uraemia.

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