Catatonia as a manifestation of uremia has rarely been mentioned in the literature (2) and never before described in detail. The two patients in this report had prominent catatonic stupor as the major neurologic finding associated with uremia. Catatonia resolved rapidly and completely in both patients with dialysis.

A 49-year-old man with a history of proteinuria and hypertension slowly developed anorexia, nausea, vomiting, disorientation, and confusion. Two days before hospital admission obtundation progressed to semistupor. No seizure activity was observed. The patient had no known history of head trauma or previous psychiatric disorder. He was minimally responsive to all stimuli. Blood pressure was 125/80 mm Hg, pulse 80 per minute and regular, and respirations 20 per minute. Tissue turgor was decreased. Except for a loud S4 gallop, his abnormalities were confined to neurologic findings. The patient was mute, lay motionless in bed, and had decreased spontaneous movements. Waxy flexibility was seen: the patient's body could be "molded" into bizarre fixed forms by manipulating his arms and legs. Withdrawal to painful stimuli was noted. Deep tendon reflexes were equal and normal. Grasp, suck, rooting, and snout reflexes were all present.

His hematocrit was 26%; serum creatinine 9.6 mg/dl; blood urea nitrogen (BUN) 153 mg/dl; sodium 120 meq/litre; total CO₂ 21 meq/litre; calcium 5.2 mg/dl; phosphorous 6.7 mg/dl; and magnesium 1.4 mg/dl. Arterial blood gas values were pH 7.35; Pco₂ 38 mm Hg; and Po₂ 120 mm Hg. Skull X rays, brain scan, echoencephalogram, lumbar puncture, and blood cultures were normal. The electroencephalogram had diffuse slow waves with a single burst of triphasic waves occurring synchronously at the rate of one per second, consistent with a metabolic encephalopathy.

Peritoneal dialysis for 48 h resulted in a BUN level 48 mg/dl and creatinine of 7.9 mg/dl. For the first 24 h postdialysis the patient exhibited psychotic behavior. Confusion, incoherent speech, and alternating inappropriate crying and laughing was noted. With no additional therapy, his psychotic behavior remitted completely within another 24 h. For the remainder of hospitalization, his behavior was normal.

A search for reversible causes of renal failure was unsuccessful, and chronic hemodialysis was started. During the next 18 months, the patient showed no evidence of abnormal behavior and was fully rehabilitated to his premorbid mental and physical state.

A 61-year-old woman with a history of lupus nephritis and renal failure was admitted to hospital with generalized pruritis, weakness, lethargy, disorientation, and incontinence. Two months earlier an arteriovenous fistula had been created because of progressive deterioration in renal function. Her blood pressure was 180/100 mm Hg, pulse 118 per minute, and respirations 16 per minute. Evidence of volume contraction was noted. Neurologic examination revealed that she was disoriented, abulic, and unable to perform simple commands. Spontaneous movements were absent, and waxy flexibility was seen. Diffuse hyperreflexia was noted, and she had no pathologic reflexes.

Lumbar puncture, brain scan, and echoencephalogram were all normal. The electroencephalogram revealed diffuse slowing without focal features, consistent with a metabolic encephalopathy. Her BUN concentration was 168 mg/dl; creatinine 22.8 mg/dl; glucose 295 mg/dl; and total CO₂ 14 meq/litre. Despite volume repletion with normal saline the patient became less responsive, and hemodialysis was started. Her abnormal motor behavior and mental status returned to normal within 24 h. after the second hemodialysis procedure.

For the past 2 years, the patient has been undergoing chronic hemodialysis and is fully rehabilitated without neurologic or psychiatric abnormalities.

The essential feature of catatonic behavior is a marked abnormality of motor function. This abnormality may
take the form of stupor or excitement. In stuporous states, patients are mute and immobile or have markedly decreased spontaneous movements. They are negativistic and frequently will not eat or drink. No attention is given to bodily needs, personal cleanliness, or neatness of dress. Grimacing and bizarre gestures and postures are common features. Waxy flexibility is a characteristic feature of this state; however, it can be induced in normal subjects by hypnosis.

Although catatonia is most commonly seen as a feature of schizophrenia, organic causes, including primary hyperparathyroidism (3), urinary retention (4), and frontal lobe lesions of the brain, have been described (5). Abnormalities of frontal lobe function may be seen in uremia (1, 6). Signs such as the rooting, grasp, snout, and suck reflex seen in our first patient probably signify depression of frontal lobe inhibitory mechanisms. It therefore seems plausible that the catatonic stupor seen in our patients may have been mediated through an effect of uremia on the frontal lobe.

In our first patient, serum sodium level was moderately depressed when catatonia developed. Hyponatremia may have contributed to a depressed state of consciousness but has not been reported to cause catatonic stupor. Our second patient had lupus nephritis (with no extrarenal manifestations); there was no evidence of lupus cerebritis before or after the episode of catatonic stupor.

These cases indicate that catatonic stupor should be added to the list of neurologic manifestations of renal failure. Rapid and complete clearing can be expected with dialysis.

REFERENCES

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