CASE REPORT

ACUTE ABDOMINAL DISTENSION SECONDARY TO URINARY RETENTION IN A PATIENT AFTER ALCOHOL WITHDRAWAL

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Abstract: Several cases of alcohol-induced bladder dysfunction have been reported previously, but the mechanism of its development is varied and unclear. We report a case of symptomatic abdominal distension due to urinary retention after alcohol withdrawal. The timing of the onset suggests that it was induced by alcohol withdrawal.

Mrs A was a 48-year-old married Japanese woman with three children. She gave an informed consent to this case report, some biographical details of which have been changed to preserve anonymity. After passing out of university, she worked in an office for 6 years. She began to drink occasionally at this time. She suffered from panic disorder at the age of 38 and came to our department for the first time. The amount of alcohol ingestion increased gradually from the age of 42, which accelerated rapidly and markedly at the age of 48. After severe abuse of spirits (1 l/day) and poor nutrition for 2 months, she was admitted to the psychiatry department. On admission, she had regular meals and was prescribed oral vitamins including thiamine. Two days later, she developed symptoms of alcohol withdrawal (e.g. tremor, sweating, insomnia and visual hallucinations), which improved after about a week. She was aware of a progressive abdominal distension ~10 days after admission. Her consciousness was alert and she showed no memory disturbance or neurological deficit except for slight tremor and sweating. The abdomen was extensively distended but soft and non tender; there was no shifting dullness. Laboratory data revealed normal electrolytes, blood cell counts and slight elevation of serum aspartate amino transferase, serum alanine transferase and gamma-glutamyl transferase. She was suspected to have ascites and referred to an internal physician.

The results of an abdominal ultrasound suggested severe urinary retention and no ascites. An abdominal computed tomography scan performed after emptying the bladder (Fig. 1) showed a marked distension of the bladder at the S5 level. No pelvic mass or ascites was identified. A urodynamic study confirmed a decrease in bladder sensation, hypoactivity of detrusor muscle and over 600 ml of residual urine. She was given a diagnosis of neurogenic bladder with autonomic dysfunction, secondary to alcoholic neuropathy. She learned to insert a urethral catheter by herself 3–4 times a day.

The neuropathy showed a moderate improvement in the next 6 months but she still needs to insert a urethral catheter once a day.

COMMENT

The abdominal distension of this patient was caused by urinary retention due to alcohol-induced neuropathy, which was precipitated by alcohol withdrawal. Three cases of alcohol-induced bladder dysfunction have been reported previously (Sheremata et al., 1972; Tjandra et al., 1997; Ruiyong et al., 2002). Ruiyong et al. (2002) has reported a case of severe alcohol-induced urinary bladder distention with bilateral hydrourereter due to alcoholic neuropathy. However, the timing of the onset of the symptom was unclear. Similar to the case of Ruiyong et al. (2002), our patient showed neither physical nor imaging evidence of obstruction, and she had not taken any medications that would predispose her to urinary retention. In contrast to the other two cases (Sheremata et al., 1972; Tjandra et al., 1997), she showed symptoms and signs of neither thiamine deficiency nor alcoholic myelopathy.

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Fig. 1. Abdominal computed tomography scan after emptying the bladder shows a marked distension of the bladder at the S5 level.
Thus, we believe that her urinary retention was due to bladder dysfunction by alcoholic neuropathy. Interestingly, her urinary retention started only after alcohol withdrawal. Alcohol withdrawal is associated with the onset of other alcoholic neuropathies such as peripheral polyneuropathy and Korsakoff’s syndrome. Recent experimental researches suggest that ethanol withdrawal causes neurotoxicity and inhibits neuronal recovery processes in rat organotypic hippocampal cultures, and that alcoholic neurodegeneration occurs through multiple mechanisms during intoxication and withdrawal (Fulton et al., 2004). The findings in our case suggest that neurogenic bladder due to alcoholic neuropathy could be precipitated by alcohol withdrawal.

REFERENCES


