Symptomatic hypoglycaemia during haemodialysis in a non-diabetic patient with acute hepatitis

Sir,

Hypoglycaemia occurring during haemodialysis is a recognized but uncommon complication associated with glucose-free haemodialysis. Predisposing factors for haemodialysis-induced hypoglycaemia include insulin infusion [1], β-adrenergic blockade [2], chronic liver dysfunction and shock [3]. We would like to draw attention to the fact that acute hepatitis could precipitate symptomatic hypoglycaemia during haemodialysis in the absence of other known predisposing factors.

Case. A 50-year-old Chinese man, who had been on maintenance haemodialysis for 10 years, developed confusion and twitching of his upper limbs 2 h after the commencement of a routine haemodialysis treatment. He was found to be hypoglycaemic with a serum glucose level of 1 mmol/l. His serum electrolytes were generally normal. His symptoms resolved promptly after an intravenous injection of 40 ml of 50% dextrose solution. Blood tests performed at the start of the haemodialysis session (before the hypoglycaemic attack) showed an acute hepatitic picture: AST, 4790 U/l; ALT, 2250 U/l; bilirubin, 18 μmol/l; and γ-GTP, 72 U/l. The patient subsequently volunteered a history of feeling malaise, with a loss of appetite for a few days prior to the hypoglycaemic attack. The patient was not a hepatitis B or C carrier and did not suffer from chronic liver disease. Serological test results for recent hepatitis infection were as follows: HAV Ab IgM, negative; HBsAg, negative; HBsAb, positive; HBeAb IgM, negative; HCV Ab, negative; and cytomegalovirus antigen, negative. Hepatitis C RNA in the serum had not been tested. Autoimmune marker screening was negative. The patient was not taking aspirin or β-blockers and there was no recent change of drug prescription. The patient’s appetite remained poor for the following 2 weeks and he required regular infusions of 20% dextrose during haemodialysis to maintain normoglycaemia. The patient’s liver function gradually improved and became normalized 3 weeks after the hypoglycaemic episode (AST, 28 U/l; ALT, 16 U/l; bilirubin, 14 μmol/l; and γ-GTP, 48 U/l). Resolution of the acute hepatitis was accompanied by return of appetite and normalization of blood glucose levels during haemodialysis.

Comment. Patients with chronic renal failure are prone to developing hypoglycaemia because of impaired hepatic glycogenolysis and gluconeogenesis [4]. However, symptomatic hypoglycaemia during haemodialysis is not commonly seen. Our patient had been stable on haemodialysis for a long time and he did not possess any of the known predisposing factors for haemodialysis-induced hypoglycaemia, such as insulin therapy or the use of β-blocking drugs. We believe that the transient liver dysfunction and the loss of appetite caused by the acute hepatitis in our patient precipitated the symptomatic hypoglycaemic attack. The exact cause of the acute hepatitis in our patient has not been established. The self-limiting nature of the hepatitis, the associated constitutional symptoms and the lack of other obvious causative factors suggest that it might have been caused by some form of viral infection (other than hepatitis A, B, C or CMV) or toxic aetiological agents. Obviously, the possibility of a recent HCV infection could not be entirely ruled out in our patient since we had not tested for the presence of HCV RNA in his serum.

Our case highlights the possibility that inter-current illness such as acute hepatitis might precipitate hypoglycaemic attack during haemodialysis in otherwise stable maintenance haemodialysis patients. Careful monitoring of blood glucose level and provision of systemic dextrose supplementation during haemodialysis in such patients are necessary so as to prevent hypoglycaemia, which could have serious consequences.

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