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Prevalence of severe hypokalaemia in patients with traumatic brain injury

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ABSTRACT

Introduction: Patients with traumatic brain injury (TBI) are more vulnerable to develop hypokalaemia, we sought to investigate the prevalence, and the relationship between severe hypokalaemia and the mortality of traumatic brain injury patients.

Methods: Isolated traumatic brain patients who had hypokalaemia (serum potassium <3.5 mmol/L) and age ≥ 14 yrs were recruited into the study between January 2008 and March 2013. Hypokalaemia was defined as potassium level in the blood <3.5 mmol/L during the hospitalisation, which was classified by severity: mild (3.0 mmol/L ≤ K < 3.5 mmol/L), moderate (2.5 mmol/L ≤ K < 3.0 mmol/L) and severe (K < 2.5 mmol/L). Multivariable logistic regression was performed to find the impact of hypokalaemia on mortality.

Results: A total 375 cases were included in analysis. The peak incidence of severe hypokalaemia occurred in the first 24–96 h. TBI patients with severe hypokalaemia had significantly higher serum sodium and lower serum phosphorus than those patients with mild or moderate hypokalaemia ($p < 0.001$). Compare to other groups, the severe hypokalaemia group had the worst outcome. Moreover, the patients ($n = 15$) who had severe hypokalaemia, hypernatraemia (Na > 160 mmol/L), and hypophosphataemia (P < 0.3 mmol/L) all died in hospital. Multiple logistic regression analysis resulted in decrease of GCS (OR = 1.27; 95% CI = 1.15–1.41; $p < 0.001$) and potassium (OR = 4.35; 95% CI = 2.04–9.26; $p < 0.001$) being associated with significant increased risk of mortality.

Conclusions: The peak incidence of severe hypokalaemia occurred in the first 24–96 h. TBI patients with severe hypokalaemia are more vulnerable to develop hypophosphataemia and hypernatraemia than patients with mild and moderate hypokalaemia. Severe hypokalaemia are the independent risk factors for mortality in TBI patients.

Introduction

Potassium is the major intracellular cation, with relatively low extracellular levels. Hypokalaemia is associated with cardiac arrhythmias, muscle weakness, rhabdomyolysis, renal failure, and hyperglycaemia. Dyskalaemia is common in trauma patients, up to 50% of injured patients have hypokalaemia [1]. Patients with traumatic brain injury (TBI) are more vulnerable to develop hypokalaemia than patients with other types of trauma [2,3].

Hypokalaemia following head trauma is a well-known phenomenon. Although patients with mild hypokalaemia are usually asymptomatic, severe hypokalaemia ($[K^+] < 2.5$ mmol/L) is life-threatening. The mortality increases with the severity of the hypokalaemia [4]. It may pose a challenge in a neurointensive care setting. Moreover, hypokalaemia rarely occurs as an isolated

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phenomenon in clinical practice, a large number of hypokalaemia patients are accompanied by other electrolytic disorders [5]. Unfortunately, few studies have investigated the impact of severe hypokalaemia accompanied with other electrolytic disorders on the outcome in TBI patients.

In order to expand the scope of hypokalaemia in patients with TBI, we sought to investigate the prevalence of severe hypokalaemia in TBI patients, the relationship between severe hypokalaemia and other electrolytic disorders, and the impact of severe hypokalaemia on the mortality of these patients.

Patients and methods

Subjects

This was a retrospective, single-centre, observational study. The study was conducted between January 2008 and March 2013 at Shanghai Neurotrauma Center in Huashan Hospital. The research protocol was approved by Ethical Committee of Huashan Hospital affiliated to Fudan University. Written informed consent was obtained from all participants or their guardians after a full explanation of the study. The inclusion criteria were as follows: TBI with radiologic signs of intracranial brain injury [epidural or subdural haematoma, contusion or parenchymal haemorrhage, or traumatic subarachnoid haemorrhage (tSAH)] documented by CT scan; age over 14 years; admission within 24 h after TBI; and hypokalaemia occurred during the hospitalisation. The exclusion criteria were the following: (1) history of renal insufficiency; (2) long-term use of diuretics or corticosteroids; (3) diabetes mellitus, pituitary tumour; craniopharyngioma; (4) severe infection, haemodynamic unstable, hypovolemic shock; (5) frequent diarrhoea during admission. We do not exclude the patients with hyperglycaemia, who use insulin during treatment.

Measurements

The following information: age, gender, diagnosis, head CT scan, Glasgow Coma Scale (GCS), length of stay (LOS), daily potassium level in the blood (detection of blood electrolytes was done at 6:30 am and 8:00 pm, and the minimal level was used for analysis), serum sodium level, serum calcium level, serum phosphorus level, serum creatinine and serum urea nitrogen were recorded at the date when the lowest serum potassium occurred. Serum potassium and other electrolyte levels were measured by auto-analyzer (Hitachi 7600-020 Automatic Analyzer, Japan). The standard value of test indicators in our hospital: serum potassium 3.5–5.5 mmol/L, serum sodium 135–147 mmol/L, serum phosphorus 0.8–1.6 mmol/L, serum calcium 2.1–2.6 mmol/L, serum albumin 35–50 g/L, blood urea nitrogen (BUN) 2.5–7 mmol/L, creatinine 50–130 μmol/L. Hypokalaemia was defined as potassium level in the blood <3.5 mmol/L during the hospitalisation, which was classified into 3 degrees of severity: mild (3.0 mmol/L $\leq K < 3.5$ mmol/L), moderate (2.5 mmol/L $\leq K < 3.0$ mmol/L) and severe ($K < 2.5$ mmol/L). Hypernatraemia and hypophosphataemia were defined as serum sodium > 147 mmol/L and serum phosphorus <0.8 mmol/L in the blood.

Statistical analysis

Data were summarised using means and standard deviations or medians and interquartiles for continuous variables. The differences between various hypokalaemia groups were tested with the non-parametric Kruskal–Wallis test. Categorical variables are presented as count and percentage. The associations between the categorical variables and hypokalaemia groups were tested with the Chi-square test. Association between rate of measurement and the serum potassium was tested by Spearman rank correlation. Linear logistic regression and binary logistic regression were both fitted to find the impact of different variables on mortality. Box–Tidwell test was used to evaluate the assumption of linearity. The interaction terms (serum sodium, albumin and creatinine plus age) were also included in the logistic regression (using the Enter method) to evaluate the independent predictors of mortality. The results were summarised by the corresponding odds ratios (OR) and adjusted OR with 95% confidence interval (CI). For all analyses, $p < 0.05$ was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics 19.0 (IBM, New York, USA).

Results

Demographics and clinical characteristics

A total of 1158 patients were screened, with 375 patients (266 men and 109 women) who developed hypokalaemia enrolled to the study (Fig. 1). Hypokalaemia occurred in 32.4% of our population. The mean age in this study was 50.8 (SD 15.7) years. The mean GCS on admission among eligible individuals was 9.1 (SD 4.0). The intracranial pathologies in 375 patients with hypokalaemia included epidural haematoma (21.3%), subdural haematoma (40%), subarachnoid haemorrhage (56.5%) and

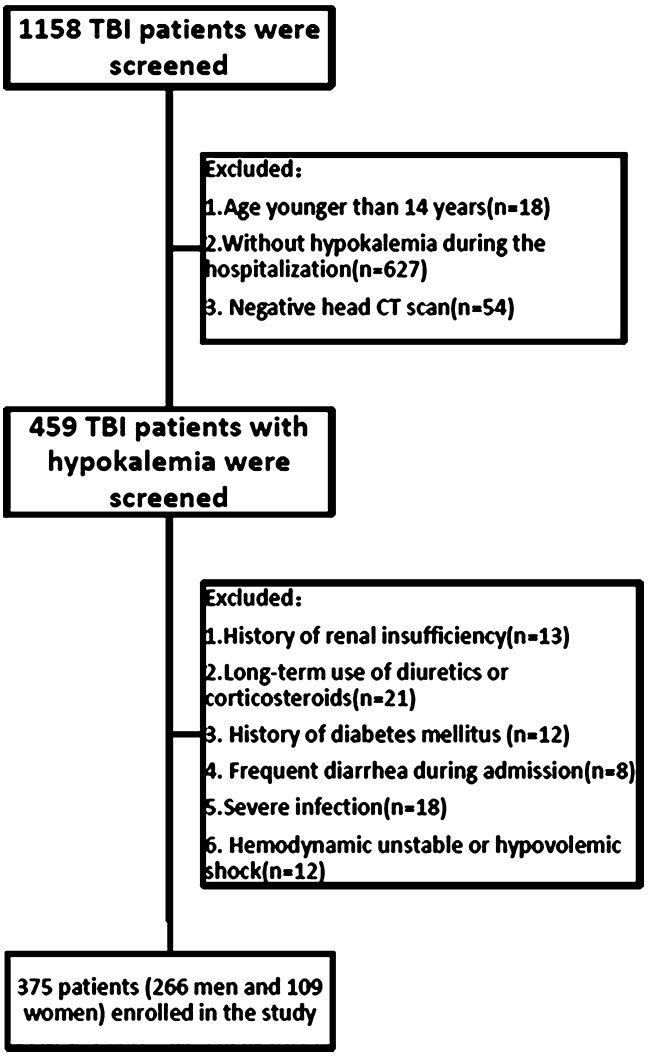

Fig. 1. A flow chart for identification process of eligible patients.