Int J Emerg Med (2009) 2:57–58
DOI 10.1007/s12245-008-0079-2

LETTER TO THE EDITOR

Comment on Grmec et al.: A treatment protocol including vasopressin and hydroxyethyl starch solution is associated with increased rate of return of spontaneous circulation in blunt trauma patients with pulseless electrical activity

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Received: 16 November 2008 / Accepted: 30 November 2008 / Published online: 23 January 2009

Dear Editor,

The study by Grmec et al. is encouraging given the very high mortality of trauma victims who experience out-of-hospital pulseless electrical activity (PEA) arrest [1, 2]. In the treatment group there were three interventions: vasopressin, hypertonic saline and hydroxyethyl starch (hypertonic hydroxyethyl starch in the study). However, caution is to be advocated in the use of hydroxyethyl starch (HES) as the preferred colloid in this clinical setting. The recently published VISEPT study (10% 200 kDa/0.5 HES) and others (10% 200 kDa/0.62 HES) have shown an association of HES with increased frequency of renal impairment in sepsis and also in renal transplantation [3–5]. A systematic review concluded that HES increases the risk of acute renal failure among patients with sepsis and may also reduce the probability of survival [6]. With the overwhelming renal insult from a haemorrhagic PEA arrest, the addition of the vasoconstrictive effects of vasopressin and an absence of solid evidence for benefit of HES, it would be better to use a colloid not associated with renal dysfunction. The newer starches are smaller, less substituted (130 kDa/0.4) and may be safer than the ones used in previous studies but the uncertainty remains.

Fluid replacement is established in controlled haemorrhagic shock, but its use in uncontrolled haemorrhagic shock is still controversial. In traumatic PEA the primary goal is to restore blood pressure and cardiac output to a level that can sustain life and prevent secondary cerebral injury. Given that haemorrhage is the most likely cause of the PEA, then judicious administration of fluid in combination with vasopressin is physiologically sound as the shift in blood away from subdiagphragmatic structures towards the heart and brain, mediated by the administration of vasopressin, may not be sufficient on its own to restore spontaneous circulation. In such an acute extreme pathophysiological situation it is more likely that the provision of intravascular volume is more important than the type of fluid. The debate over crystalloid vs colloid and colloid vs colloid continues and is now confounded by the increasing interest in the use of hypertonic saline in trauma.

An international, multicentre, randomised controlled trial to assess the effects of vasopressin vs saline placebo in pre-hospital traumatic haemorrhagic shock patients not responding to standard shock treatment will commence in January 2009 [Vasopressin in traumatic haemorrhagic shock (VITRIS.at) study] [7, 8]. The purpose of the trial is to assess the effect of vasopressin on hospital admission rate (primary study end point), as well as on haemodynamic variables, fluid resuscitation requirements and hospital discharge rate (secondary study end points), in pre-hospital, presumed traumatic haemorrhage shock patients with a systolic arterial blood pressure below 90 mmHg after more than 10 min but less than 30 min of standard shock treatment by the emergency medical physician [intubation, crystalloid, colloid and hypertonic saline (up to 4 ml/kg) fluid resuscitation, and catecholamines (ephedrine, phenylephrine, norepinephrine)]. The trial is scheduled to complete in April 2011 and the results may obviate the need for further studies of vasopressin in traumatic PEA.

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