Monitoring of The Effect of Synthetic Vasopressin in Vasodilatory Shock Using Esophageal Doppler Probe

Thesis
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Abstract

Background: Septic shock is a form of vasodilatory shock characterized by arteriolar vasodilation the objective of treatment is to elevate tissue perfusion & mean arterial pressure to allow adequate organ perfusion. Noradrenaline and dopamine were the usual catecholamines used in the treatment of septic shock. Loss of response was the common problem that lead to patient loss after large continuous doses of noradrenaline which was termed as catecholamine refractory septic shock. Recently vasopressin and its analog namely terlipressin were used in the treatment of such catastrophic condition.

Methods and Results: In a prospective controlled study we included 40 patients with catecholamine resistant septic shock i.e. noradrenaline dose exceeded 0.6 μg/kg/min divided into two groups: 20 patients were treated conventionally according to surviving sepsis campaign 2008 who served as a control group and the other 20 patients were treated conventionally and when noradrenaline dose exceeded 0.6 mic/kg/min terlipresin in a dose of 1 mg I.V bolus every 12 hours for a study time of 48 hours was started. Terlipressin therapy was associated with increased MAP from 58±14 mmHg at baseline to 73±20 mmHg with P value: 0.008 after 48 hours that allowed significant reduction of noradrenaline dose from 50 mic/min on day 0 to <25±8 mic/min after 48 hours. Terlipressin therapy was associated with increased systemic vascular resistant from 546±260 dyne.sec/cm^{-5} to 986±390 dyne sec/cm^{-5} after 48 hour which represent normalized arterilor tone that is expected to allow better organ bed perfusion.

There was reduction of both stroke volume and cardiac output (from 63±16 ml/beat to 51ml/beat and from 78 liter/min to 5.3 litre/min, respectively) yet this was not associated with abnormal organ perfusion marked by improved urine output from 49 ml/hour to 133 ml/h and improved global perfusion as marked by improved base deficit which represent lactic acidosis from 9.3±3 mEq/L to 5.7±3 mEq/L P value: <0.002. Terlipressin therapy was not associated with deleterious effect on PO_2/FiO_2 ratio (from 208±74 to 211±118 after 48 hours. Yet there was significant reduction of oxygen delivery (Do_2 from 848 ml/min to 610±47 ml/min after 48 hours (P> 0.02).

There was no effect on length of ICU stay in both groups (16±6 in the terlipressin group and 12±6 days in control group, P< 0.06). This shorter length of stay in control group may be due to the rapid deterioration of hemodynamics and death without terlipressin support, shown true as mortality in control group of 70% versus 60% (12/20) in terlipressin group with absolute risk reduction of 10% and

relative risk reduction of 25%. Regarding organ function, terlipressin could improve SOFA score form 11±3.2 to 8±5 with P value: <0.02.

Conclusion: Terlipressin is a rather safe inexpensive easy to administer alternative in the treatment of septic shock. Further studies are needed to decide the ideal timing for initiation of this therapy early vs late, adjuvant or as an initial treatment.

Key words: Terlipressin, catecholamine resistant septic shock