Selected Summaries

Furosemide: Much More Than a Diuretic

[Yetman AT, Singh NC, Parbtani A, et al. Acute hemodynamic and neurohormonal effects of furosemide in critically ill pediatric patients. Crit Care Med 1996, 24: 398-402].

The acute hemodynamic effects of furosemide, the temporal relationship between hemodynamic changes and changes in neuroendocrine axis and between hemodynamic changes and urine output were studied in 14 critically ill children who clinically required diuretic therapy and were admitted to Pediatric Intensive Care Unit of a tertiary care university center. Hemodynamic and neurohormonal measurements were taken before and after furosemide administration (1 mg/kg/dose). Cardiac index deteriorated by 10 min after drug administration (-9.4 ± 3.9%; $p < 0.05$) and was associated with an increase in systemic vascular resistance (17.1 ± 4.8%; $p < 0.05$). There was a subsequent increase in cardiac index (20 ± 4.9%; $p < 0.05$) at 30 min, with a decrease in systemic vascular resistance (-11.5 ± 5.2%; $p < 0.05$). These hemodynamic changes were associated with marked increases in renin and norepinephrine concentrations and an increase in urinary prostaglandin release. The hemodynamic and neurohormonal effects had their onset before maximum diuresis. The authors concluded that intravenous furosemide administration in acutely ill pediatric patients results in an acute but transient deterioration in cardiac function that appear to parallel the neuroendocrine changes rather than the acute diuresis.

Comments

Furosemide, a loop diuretic is frequently administered to critically ill pediatric patients to augment urine output and to relieve pulmonary edema. It is believed that it improves pulmonary edema by promoting a rapid diuresis that subsequently leads to a decrease in circulating blood volume and a concomitant reduction of the increased pulmonary artery occlusion pressure. However, in the adult patients the drug has been shown to have important hemodynamic effects that often precede the onset of diuresis. In addition, clinically important changes in hemodynamic variables have been found to occur, even in the presence of a marked impairment in renal function. Occurring parallel to these hemodynamic changes are increases in plasma renin and norepinephrine concentrations which may be mediated, atleast in part, by an increase in prostaglandin biosynthesis in the kidneys.

All these data have been derived from studies on adult patients suffering from the medical problems that are uncommon entities in the pediatric age group. There exists a paucity of studies demonstrating the drug's effects on hemodynamic variables and the mechanism of these changes in critically ill children. The authors for the first the time demonstrate that furosemide administration (l mg/kg IV) causes a transient deterioration in cardiac function followed by an improvement that appears to be hormonally mediated, resulting in alteration of the systemic vascular resistance index. In contrast to the adult population, the increase in systemic

vascular resistance index in children occurred earlier (peak 10 min vs 1 h) and was of shorter duration (10 min vs 20 min). There was on abrupt decrease in cardiac index in association with the increase in systemic vascular resistance index at 10 min, followed by an increase in cardiac index at 20 min with a decrease in systemic vascular resistance index. Urine output began to increase immediately after drug administration, despite a decrease in cardiac output. Renin and norepinephrine concentrations increased acutely and, unlike the findings in several studies on adults, remained above baseline values. This acute and prolonged increase in renin concentration may reflect the difference between the disease processes between adults and children. Furosemide induced increased prostaglandins may have played an integral role in the causation of the ensuing hemodynamic, diuretic, and neurohormonal changes.

All of us who use fursemide in acutely ill children should consider such hemodynamic alterations, especially the deterioration in cardiac output, even in the absence of a clinically important diuretic response. The furosemide induced decrease in cardiac output and increase in systemic vascular resistance index may increase the potential for paradoxical pulmonary edema. To ensure more controlled diuresis with less hemodynamic alterations the clinician may consider a continuous infusion of furosemide in the hemodynamically unstable patient(l).

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REFERENCE

1. Steven JM, Danziger LH. Continuous infusion of loop diuretics in the critically ill: A review of the literature. Crit Care Med 1994, 22:1323-1329.