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Pediatric vascular filling solutions: Fluid replacement solution in paediatric

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ABSTRACT

Vascular filling is one of the first therapeutics used for acute circulatory failure, whether in the emergency department or in intensive care units. Its effect involves an increase in cardiac preload leading to an increase in systolic ejection volume and cardiac output. This vascular filling uses solutes whose first expected effect is volume expansion. However, all filling solutes have associated or metabolic effects. These may be favorable effects such as the normalization of lactic acidosis or renal insufficiency induced by hypovolemia. These include adverse effects such as hyponatremia induced by hypotonic solutes, disorders of acid-base balance such as hyperchloremic acidosis, and effects related to buffer solutions associated with solutes (lactate, acetate).

Keywords: Thyroid disorders, inflammatory bowel diseases, thyroid nodules, female gender.

INTRODUCTION

The use of pediatric vascular filling is a common occurrence. It is one of the pillars of therapeutic management of resuscitation patients, and is an aggressive therapy with a number of risks. The pitfalls and errors come from the ignorance of the indications, the non-respect of the therapeutic objectives and especially the side effects of the solutes used.

Thus, the choice of solutes has been debated for several years in pediatrics, where literature is much less abundant than in adults. This article provides an update on the different filling products.

MATERIAL AND METHODS

The different filling solutes:

Three types of products are available: crystalloids, synthetic colloids and albumin.

These products are equivalent in terms of efficiency when their distribution volume is taken into account. The choice of solute is determined by its cost, expected plasma expansion and possible side effects.

Isotonic crystalloids:

The isotonic crystalloids available are the isotonic saline (SSI) and the Ringer lactate (RL). If their power of expansion is comparable, their chemical composition is very different (Table 1).

Advantages:

The low potency of crystalloid expansion is in contrast to their widespread use in daily practice.When RL is administered to a normovolemic conscious subject, the leakage rate to the interstitium ("elimination constant") is 133 ml / min. When the subjects are rendered hypovolemic after subtraction of 450 and 900 ml of blood, this constant decreases respectively to 100 then to 34 ml / min, ie a division by 4 of the leakage constant.

The isotonic saline serum (0.9% NaCl) allows a satisfactory but short-lived volume expansion with rapid diffusion to the interstitial medium, requiring large volumes to maintain the filling effect as all the crystalloids (perfused volume / volemic expansion ratio : 4/1).

The volumic efficacy of a crystalloid is greater than the conventional 20% value in the acute phase of hypovolemia and may explain the confidence that practitioners have in these situations during moderate hypovolemia. This is no longer true when the patient has a capillary leak syndrome in a systemic inflammatory response regardless of its origin. Early work suggests that the effectiveness of crystalloids decreases with volume perfused during haemorrhagic shock, probably as an increase in capillary leak [1,3].

Disadvantages:

Tissue (especially digestive) edema inherent in the administration of high volumes of crystalloids could alter tissue oxygenation and participate in the development of multivisceral syndromes (MDS).

In pathology, a positive hydrosodal balance is associated with excess mortality during acute respiratory distress syndrome and in patients with acute renal failure.

In traumatology, a "supranormal" filling by Ringer lactate in polytraumatised patients is the cause of a mesenteric edema with abdominal compartment syndrome and excess mortality by SDMV.

As a corollary, the volume of crystalloids administered in prehospital and emergency departments in patients admitted for complex fractures of the lower limbs is an independent factor in the occurrence of abdominal compartment syndrome even in the absence of associated abdominal trauma.

High doses (> 30 ml / kg) of SSI induce hyperchloremic acidosis due to the supra-physiological concentration (154 mmol / l) of Chlorine (strong anions).

These acidosis are not observed with RL at the same doses due to a chlorine concentration close to that of plasma and the presence of lactate ion. Insofar as lactate ion is a base, the use of large volumes of RL exposes the risk of metabolic alkalosis. The RL containing potassium, this solute is conventionally contraindicated during hyperkalaemias. Finally, RL exposes the risk of hyponatremia (sodium concentration of 6 g / l) and hypoosmolarity (osmolarity of 277 mmol / l) potentially deleterious in acute cerebral diseases [1,2].

Table 1: Plasma, SSI and RL composition [1].

Concentrations (mmol/l)	***plasma***	***Isotonic saline serum***	***Ringer lactate***
Na+	***142***	***154***	***130***
K+	***5***	***0***	***4***
CL-	***103***	***154***	***108***
Ca++	***2,5***	***0***	***0,91***

Mg++	***1***	***0***	***0***
HCO3-	***27***	***0***	***0***
Lactate	***5***	***0***	***027,6***
Osmolarité	***295***	***308***	***277***

Hypertonic crystalloids:

Hypertonic saline (SSH) salts (7.5% NaCl) with a dosage of 2 to 3 ml / kg have a high volume expansion capacity.

Due to hypertonicity, infusion volumes greater than 250 ml expose to serum levels greater than 160 mmol / l causing serious complications.

Experimentally, SSH has multiple favorable effects on microcirculation and immune functions. At the macrocirculatory level, SSH exerts a volume expansion of 2 to 3 times the perfused volume with increased filling pressures and cardiac output during haemorrhagic shock and septic shock. It also has direct positive inotropic effects. Too rapid administration is associated with hypotension and arrhythmias. The hemodynamic effects of SSH are transient because the duration of SSH action is two to three hours during septic shock, even if a macromolecule is associated with it, suggesting a modest effect of the addition of a macromolecule.

The resulting hypernatremia and hyperosmolarity are beneficial for cerebral perfusion pressure and intracranial pressure in severe cranial trauma [4].

Synthetic Colloids:

Several types of synthetic colloids are marketed.

The dextrans:

They lead to a decrease in the platelet aggregability, and may therefore increase or cause haemorrhage. They have been implicated in the occurrence of renal insufficiency with tubular lesions. They are not currently used [5].

The gelatines:

Gelatins come from degradation of bovine bone collagen. These products have a short duration of action (2-3 h) and are essentially eliminated by glomerular filtration. Their filling power is modest because about 20% of the administered amount passes rapidly through the interstitium. The effects on coagulation are more modest than those of dextrans but the aggregability tests are modified in vitro by gelatin. Gelatins can even be considered procoagulants. In France, they cause 95% of anaphylactic accidents involving a colloid [5].

The hydroxyethyl starches:

Hydroxyethyl starches (HEA) may cause primary haemostasis disorders. It is mainly the high molecular weight (MW) HEA which are the source of these anomalies, similar to acquired Willebrand. In Europe, it is mainly medium and low-dose PMUs (Elohes®, Hesteril®, Lomol®, Pentastarch®) which are rarely responsible for major haemorrhages. However, the maximum volume to be infused is limited to 35 ml / kg per day.

HEAs interfere with all coagulation colloids, more importantly than albumin and gelatin and less than dextrans. This hypocoagulability is related to a disorder of platelet aggregability by interference of HEA with factor VIII.

HEAs are the least allergenic colloids. Most synthetic colloids provide reactions or even anaphylactic shocks. This risk is lower in children than in adults. The risk varies, with in decreasing order: dextrans> gelatins> HEA.

Renal tolerance of HEA remains subject to debate. The renal toxicity of HEA is related to the hyperoncotic nature of the solutions responsible for a drop in glomerular filtration and to a tubular deposit of HEA materialized by the evidence of vacuoles in the proximal tubules. An increase in urinary viscosity at the tubular level would be a third mechanism of toxicity, the importance of which is however less clear. All the studies carried out over the last 15 years show that the renal complications of HEA are related to the prolonged administration of high doses of HEA of high concentration (> 6%) and / or of high PM (200 kD) and high molar substitution time (TSM) (≥ 0.5).

In total, the latest generation HEA should be considered safe if the recommended doses (<50 ml / kg) are met [1,5].

Albumin: natural colloid

A small endogenous protein, the total amount of albumin is 4 to 5 g / kg, of which one third is in the vascular area and 2/3 in the interstitium. Despite its small size, albumin does not cross the vascular membrane in the healthy subject. This fragile balance seems easily broken in pathology. During hypovolemic shock or heavy surgery, leakage of albumin is frequently observed. These findings are also valid during sepsis.

The perfusion of 4% albumin is indeed responsible for a modest or no increase in oncotic pressure in resuscitation patients. The administration of albumin in resuscitation patients seems logical insofar as patients with hypoalbuminemia have an unfavorable prognosis. A new meta-analysis published in 2004 concluded that albumin is the colloid with the least side effect. A recent literature review of 71 randomized controlled trials (3782 patients) comparing albumin with other filling solutes suggests that the administration of albumin decreases the morbidity of patients hospitalized in intensive care. This effect is more pronounced in the groups where the albuminemia is the lowest (burns, cirrhosis ...) than in patients of surgery or traumatology. The authors emphasize the "obvious protective effect" of maintaining normal oncotic pressure by the administration of albumin in terms of water balance management. The issue of albumin is at the boundary between filling and correction of hypoalbuminemia ("albumin drug"). In a randomized study published in 2006, correction of hypoalbuminaemia in resuscitation patients decreased organ failure.

It is still widely used as a filling solution, especially in newborns. The main criticism attributable to albumin is the higher cost of this filling solution compared to HEA. Finally, the risk of transmission of atypical infectious agents (prion, for example) can not be considered as null.

In summary, the tolerance of isotonic albumin is excellent, but its power of expansion falls rapidly in patients with capillary leak syndrome. There is no mortality attributable to the use of this molecule. The high price of this product, the risk of transmission of prion diseases and its moderate expansion capacity mean that there is almost no indication of albumin for filling in emergency situations. The use of albumin to correct the hypoalbuminaemia of resuscitation patients is probably more permissible than for vascular filling [6,7].

CONCLUSION

The vascular filling of the child is therefore not a simple prescription. Many complications can aggravate an already precarious situation. It is therefore important to choose the modalities and the type of filling solution and to remain vigilant throughout the duration of the infusion in order to adapt the procedure in case of incidents

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