Fluid therapy – conceptual changes

Clinicians have been using intravenous fluids from the 19th century for treatment of cholera[1]. Peri operative fluid therapy dates back to the late 19th century. Since then there have been many changes in the type of fluids administered, amount and rate of administration, monitoring of fluid therapy and fluid balance and treatment of fluid overload.

The goal in fluid administration is to optimize tissue perfusion. Improving the systemic haemodynamics is the first aim followed by optimization of microvascular perfusion and tissue oxygenation. The main clinical indicator of lack of fluid is hypotension with evidence of compromised organ perfusion. The hypothesis for fluid therapy in this scenario is that the fluid volume given would increase cardiac output, thereby increasing the arterial blood pressure and tissue perfusion. Practically there is a wide variation in patient response as the cardiac output produced is not only due to cardiac pump performance but also due to preload and afterload. There is also an absence of a sustained increase in blood pressure with increased cardiac output due to peripheral vasodilatation. Further there are different mechanisms for control of the microcirculation other than the systemic haemodynamics[2]. In this context the parameters to be monitored to show fluid adequacy becomes a problem. Should we be monitoring systemic haemodynamics or should it be the microcirculation?

Once the decision to give fluid is made the next hurdle is to decide the type of fluid to be used. The separation of fluids as crystalloids and colloids is adequate when the vascular barrier is intact but not when it is breached. It seems that none of the existing fluids are perfect for fluid resuscitation and need further evaluation.

National Institute of Clinical Excellence (NICE) issued a guideline in December 2013 for intravenous fluid therapy in adults in hospital.[3] It identifies key priorities for implementation. They are principals and protocols for IV fluid therapy, assessment and monitoring, resuscitation, routine maintenance and training and education. In the principals and priorities it reminds of 5 Rs, Resuscitation, Routine maintenance, Replacement, Redistribution and Reassessment and also advocates following of protocols for these. It details assessment and monitoring, recommends volumes and fluid type for resuscitation and maintenance, and suggests the need for an IV fluid lead in a department in order to train and educate. These recommendations were criticized[4] and defended[5] in Anaesthesia May 2014.

It is in light of all these controversies that the 12th Consensus Conference of the Acute Dialysis Quality Initiative (ADQI XII) addressed the issues of fluid administration and removal in perioperative and critical care setting.[6] Their report was published in the British Journal of Anaesthesia of November 2014. It is identified that correct fluid therapy is life saving but use of certain types and volumes of fluid can increase risk of harm or even death in certain patient groups. The consensus group proposes a conceptual model of fluid therapy considering fluid as a 'drug' with dose response relationships and side effects. It is recognized that there are four distinct phases of resuscitation. They are **R**escue, **O**ptimization, **S**tabilisation and **D**e-escalation(ROS-D). Rescue phase is relevant in septic shock and major trauma. It is life saving to correct the shock and requires fluid boluses within minutes. Optimization occurs intraoperatively, in burns and diabetic ketoacidosis. It is for organ rescue and fluid therapy is titrated by use of fluid challenges over hours. Stabilisation phase is as in the postoperative 'nil by mouth' patient. Fluid therapy here is for organ support, aiming for a zero or negative fluid balance using a minimal maintenance infusion if oral intake is inadequate. De-escalation occurs as in recovery from a critical illness or acute tubular necrosis. The principal is organ recovery with the aim to mobilize the fluid accumulated and to avoid unnecessary i.v. fluids. Monitoring during the rescue phase should be bedside clinical observations. Additional monitoring of fluid responsiveness by more invasive means can identify the transition from rescue to optimization phase. In stabilization and de-escalation phases observations become less

frequent and are for prescription of fluids or diuretics on basis of physical examination, blood chemistry and known clinical course.

The ADQI XII group further states that there is a conceptual approach proposed to incorporate knowledge on vascular biology. This is to develop a more practical means to manage a patient with hypoperfusion for better outcomes. The identified vascular components (VC) are vascular content (vC), vascular tone (vT), vascular barrier (vB) and blood flow (BF).[7] These components would differ in the different forms of shock.eg. in haemorrhagic shock BF and vC are decreased, vB is impaired and vT is normal as opposed to cardiogenic shock where vC, vB, and vT are intact with BF decreased. This approach results in more understanding of 'volume responsiveness' and is able to assess systemic, regional and microvasculature.

Rapid volume resuscitation in hypotensive critically ill patients with poor tissue perfusion often develops or is at risk of developing a positive fluid balance leading to a poor outcome. Fluid overload is managed by pharmacological means or by extracorporeal fluid removal. The ADQI XII group address when pharmacological fluid management should be initiated, the optimal mechanisms to monitor and what the ideal endpoints are to discontinue pharmacological fluid removal.[8] They also propose the need for prospective outcome studies to identify whether biomarkers would predict the need and failure of diuretic therapy, whether continuous or intermittent diuretic therapy is better, and whether addition of thiazide diuretics to loop diuretics is beneficial. A further group provides guidelines for mechanical management of fluid overlod.[7]

The ADQI XII also explored the choice of fluid in acute illness. It discusses the role of the endothelial glycocalyx, the equal efficacy of crystalloids to colloids when the vascular barrier is compromised and what is known about the choice of fluids. They conclude that there is little evidence of superiority for any i.v. fluid. They further add that hydroxyl ethyl starch appear to increase the need for renal replacement therapy though there is no increase in mortality. Certain fluids might be superior in certain settings such as saline in head injury and balanced fluids when there is a risk of renal injury. They further conclude that chloride content of fluids may have an important bearing on outcomes and that presently balanced salt solutions may be a reasonable default choice.[9]

The traditional thinking on fluid therapy has undergone many changes. We need to keep track of these and alter our practice and contribute towards finding new evidence to support appropriate fluid therapy for individuals in peri operative and critical care practice.

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