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Elderly Man; Going into Shock; Needs Fluids Now!

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Stem Case and Key Questions Content

A 74-year-old male presents to the emergency room with severe abdominal pain and vomiting for 24 hours. He weighs 230lbs and is 5'9" tall. Medical history includes hypertension, diabetes type 2 and a long standing history of diverticulitis. Medications are hydrochlorothiazide, amlodipine, hyosciamine, metformin. He has had repeated courses of antibiotics. Surgical history is not contributory Initial vital signs are blood pressure 95/70, heart rate 122, temperature 101.3F and SPO2 93 on room air. Free air is seen on abdominal x ray. A diagnosis of ruptured diverticulum has been made and he is scheduled for emergent exploration. While the operating room is being prepared, the patient is resuscitated with intravenous fluids, nasogastric suction, antibiotics and morphine for pain. He receives a total of 2.5l crystalloids without any increase in blood pressure. At preanesthetic assessment, his blood pressure is 77/50, heart rate 135, respiratory rate 34, oxygen saturation 86% on 3l O2 He has been oliguric for 2 hours. Blood sugar is 194mg/dl., Hb 9gm, and platelet count 51thousand. The patient is fully conscious, complaining of severe pain. The surgeon advises the anesthesiologist to give normal saline for further resuscitation.

1. Would any other tests add information that might impact anesthetic care?
2. Would echocardiography be of value?
3. How could volume status be evaluated?
4. What is the significance of his medications?
5. What are the possible causes of anuria for 2 hours?
6. What is the differential diagnosis of hypotension and tachycardia in this case?
7. What are the effects of hyperglycemia?
8. Discuss the pros and cons of tight glycemic control.
9. What might be an appropriate fluid replacement plan?

10. Are vasopressors indicated; if so, which?

11. What are the pros and cons of central vein and arterial cannulation?

12. Are intraoperative tests indicated?

13. How reliable are the vital signs in determining fluid replacement?

A 12 lead electrocardiogram shows tachycardia but no acute ischemic changes. An attempt is made to insert a central jugular line but the patient is groaning and moving in pain and placement is not possible. A second peripheral vein and the right radial artery are cannulated. Arterial blood gas analyses indicates paO2 101, (5l non rebreathing mask), paCO2 30, and pH 7.15. Electrolyte panel reveals sodium 147, potassium 3.3, chloride 95, bicarbonate 20 glucose 205, anion gap 32mEq/L, BUN 18.

1. Is central vein cannulation essential?

2. Is this patient hypoxic? If so, what is the differential diagnosis?

3. Does he have lactic acidosis?

4. What are the possible causes?

The surgeon again maintains that the fluid deficit must be at least 5 liters. He insists that replacement only be with normal saline, as he believes all colloids cause bleeding and renal failure. Fluids, 3l, are given without any improvement in vital signs or urine output. A dopamine infusion is started. Furosemide, 10mg is added.
As surgery starts, the anesthesiologist infuses Plasmalyte® solution at a rate of 75ml/hour, albumin, 2 units (25% in 100ml) are added. One unit of banked blood is given. Fresh frozen plasma and platelets were requested. Urine output remained zero. Blood pressure increases to around 100/65-95/70. Pulse rate remains high at 120 beats per minute. Laparotomy reveals fluid in the abdomen from a ruptured diverticulum. Colostomy is performed. Blood loss is estimated at 500ml. 24 hour fluid replacement includes crystalloids 8.5l, colloids 2 units, and blood 1 unit. Urine output over 7 hours is 50ml. The final Hb is 8gm. The patient's face is very swollen. The decision is made to leave the endotracheal tube in place and maintain assisted ventilation.

1. Was the perioperative fluid replacement appropriate?

2. Were more or less fluids indicated and if so, which?

3. Is the decision to leave the endotracheal tube in place appropriate?

4. Is blood pressure control optimal?
5. What orders for fluid replacement should be written now?
6. Are any tests or consultations indicated?
7. Is the patient at risk for developing ventilator-associated pneumonia?

Model Discussion Content

Perioperative fluid therapy has received considerable attention over the past decade as the effects of both fluid overload and restriction are emphasized in different settings. Many studies address these issues, often with conflicting results, in part due to lack of standardization of trials. Critical review of clinical studies reveals that current standard fluid therapy is hardly evidence based and has been challenged for years (1). Almost a century ago Cannon pointed out that the administration of fluids before operative control of an injury was ineffective (2). In trauma Bickell emphasized the benefit of surgical correction before resuscitation noting that fluids and restoration of blood pressure could dislodge a soft clot and cause more bleeding (3). However, during both the Korean and Vietnam campaigns, large fluid volume resuscitation was advised to maintain renal perfusion (and the da Nang lung was born).

Traditionally we have been taught that there are three fluid spaces. The first is readily recognizable as the intravascular space. The second generally considered to be interstitial and extravascular spaces where fluid accumulates either normally or in response to injury or edema formation, is not as well defined. Fluid shift between these 2 spaces is ongoing and can obscure hypovolemia or overload. Vascular beds can undergo dramatic capacitance changes secondary to anesthetic drugs, infusion of hyperosmolar substances and pathologic states and thus draw fluid from the interstitium. Or fluid may leak to interstitial spaces in response to failure of the cardiovascular pump system. Identification of a third space is much more difficult. It is said to be related to the surgical experience. It came into being almost 50 years ago when a surgical team studied two groups of patients in an attempt to understand the acute changes that determine the perioperative management of fluids and electrolytes (4). The control group consisted of 5 patients undergoing minor surgery with general anesthesia (cyclopropane and ether) and the second group (13 patients) had elective major surgical procedures (cholecystectomy, gastrectomy and colectomy). Plasma volume, red blood cell mass and extracellular fluid volumes were measured in all patients on 2 occasions during the operative period by using *I 131* tagged serum albumin, chromate *51* red blood cells and sulphur *35* tagged sodium sulphate. Based on finding a decrease in functional extracellular fluid in group 2, the authors concluded that there was internal redistribution of fluid associated with surgery (that is, the third space), which should be replaced by fluid administration. These findings were “confirmed” in an exsanguinated dog model which did better with immediate fluid rather than blood replacement (5). Arguing against this theory Moore postulated that a metabolic response to surgical stress

caused sodium and water retention and perioperative fluid restriction was indicated (6). The debate prompted an editorial by the two combatants both of whom urged moderation (7). Nevertheless, concept of the “third space” remained. Protocols calculated deficits based on degree of trauma and insensible losses among other “variable” fluid decreases, all of which were to be replaced with crystalloids. The 4:2:1 “rule” remains part of resident teaching today, (1st0-10kg requires 4ml/kg, next 11-20kg 2ml/kg, then >21kg is 1ml/kg.).The original description of the rule comes from a paper by Holliday and Segar who, in an attempt to simplify fluid requirements devised an arbitrary 100-50-20 scheme as a baseline for children (8). They compared their system to 3 others (9-11), which considered in turn that:

1. Surface area is a good estimate of water expenditure,
2. Caloric expenditure is based on age, weight, activity and food intake (comparing a rat and a steer) and
3. Urinary volume and insensible losses relate to age.

None of this reasoning takes into account neurologic, endocrine, pharmacologic (anesthetic) and cardiovascular status, and other pathologic conditions.
The concept of fluid deficit is based on several assumptions:

4. The preoperative fasted patient is hypovolemic,
5. Insensible perspiration increases with surgery,
6. Fluid shifts to the third space must be replaced and
7. The kidneys can regulate any fluid overload.

Fasting guidelines allow for clear fluids up to 2 hours preoperatively. Moreover after 8 hours of sleep, requirements in the non comatose individual may be little more than 1-2 cups of fluid (240-480ml) Very few patients are likely to require 1500-2000ml fluid within the first 1-2 hours of surgery and perhaps a total of 3-5 liters over 4-5h. Preoperative fasting causes a slight decrease in extracellular fluid while maintaining intravascular volume. The use of evanescent anesthetic agents ensures a rapid return to consciousness and oral intake. Insensible losses are decreased with laparoscopic incisions and by constant wound irrigation. Finally, antidiuretic hormone release during anesthesia severely curtails the ability of the kidneys to remove excess fluid.
There is increasing evidence that intraoperative fluid therapy influences postoperative outcomes (12, 13). A critique of fluid bolus therapy (20-40ml/kg) indicated weak physiological and limited experimental support and is at odds with emerging observational date in critically ill patients or those undergoing major abdominal surgery (14). Over generous fluid infusions resulting in weight gain contribute to complications such as pulmonary edema or extravascular lung water, respiratory failure, myocardial dysfunction, bacterial translocation and development of sepsis,

renal failure, wound infection and multiorgan failure (15, 16). Patients who developed postoperative blindness after lumbar surgery had a very large positive fluid balance, suggesting compartment syndrome as one of the causes of the complication. Tissue oxygenation is increased by supplemental oxygen and not by excess fluids (17). Comparison of the standard (>3L, normal saline) versus restricted (<2L, 0.45 normal saline) protocols for postoperative fluids after hemicolectomy indicated significantly more complications in the standard group and longer hospital stay. (18). Intravenous fluid therapy does not result in extracellular volume distribution expected from Starling's original model of semi-permeable capillaries subject to hydrostatic and oncotic pressure gradients within the extracellular fluid (19). Rather the contribution of the endothelial glycocalyx to vascular permeability must be considered. The glycocalyx is easily and rapidly disrupted in sepsis, by direct contact with catheters and by large fluid infusions leading to an extravasation of albumin and fluid and tissue edema (20). Other studies have emphasized the need to improve skin microcirculation in older patients, decreased due to age related changes including decline in thickness and composition by judicious use of fluids, thus improving wound healing (21).

The choice of fluid has received much attention. During short, ambulatory cases with low surgical risk it may be of little importance. Intravenous crystalloids remain in the intravascular space for short periods, redistributing quickly to soft and damaged tissue and dependent areas (gut, lungs and larynx). Edema in the gut wall increases the inflammatory response and retards forward movement. A more serious complication is abdominal compartment syndrome causing respiratory and renal dysfunction and increased epidural bleeding during prone spine surgery (22) Excessive crystalloids also increase coagulation abnormalities (dilutional or hypercoagulation), the need for more blood transfusion and delayed wound healing through increased cutaneous edema (23).
Crystalloids are divided to balanced salt solutions (BSS, such as lactated Ringer's, (LR) Plasmalyte®) and non-physiologic solutions such as normal saline. This latter substance has a pH of 7.14 (LR is 7.39), and contains 154mEq of sodium and154mEq of chloride (corresponding values for LR are 135 and 109). Hyperchloremia after infusion has been associated with metabolic and dilutional acidosis, decreased renal blood flow, coagulopathies, increased inflammatory response and mortality (24).In a study of almost 32,000 patients, comparing 0.9% saline infusion to a BSS, in hospital mortality in the saline group was 5.6% and 2.9% in the BSS group (25). One or more complications occurred in 33.7% of the saline group and 23% of the BSS group. The authors suggest the perioperative use of a calcium free solution. A Cochrane database review concluded that while administration of buffered or non buffered saline based solutions was equally safe, saline caused more hyperchloremia and metabolic derangements (26).

Colloidal expanders include albumin and hydroxyethylstarches (HES including Hespan ®, Hextend®, and Voluven®). Albumin, 5% or 25% supplied in 100ml aliquots is derived from pooled human venous plasma, which is heated to 60 degrees for 10 hours to inactivate hepatitis viruses. It contains no isoagglutinins and thus the risk of adverse reactions is very low. Preparation charges make it significantly more expensive. HES in 0.9% sodium chloride is a synthetic polymer derived from a waxy starch composed of amylopetin. It is supplied in 500ml bags. Dose related side effects include coagulopathy and greater need for renal replacement therapy (27). A Cochrane database review found no evidence from randomized trials that resuscitation with colloids reduced the risk of death compared to resuscitation with crystalloids in patients with trauma, burns or following surgery (28). In patients with sepsis, HES increased

the risk of serious adverse events, especially acute kidney injury (29). The FDA issued a black box warning for HES in June of 2013 advising that side effects are a class effect and that it should not be used in the ICU due to increased mortality and need of dialysis (30). Other studies have indicated improved outcome with colloids in some settings (31, 32).The American Society of Anesthesiologists has advocated the combination of colloids and reduced crystalloids in the prevention of postoperative visual loss (POVL) (33).

The purpose of fluid administration is to maintain vascular volume, cardiac function and tissue oxygenation. Goal directed therapy is increasingly being used to direct the amount, timing and type of fluid resuscitation. Assessment of the adequacy of intravascular volume would seem essential in determining these factors. Arterial pulse pressure variation induced by mechanical ventilation has been appreciated for decades as an indicator of hypovolemia. Computerized analyses have incorporated information from the pulse oximeter arterial wave- form to provide a continuous display of arterial pressures, stroke volume, cardiac output, pulse pressure variation and stroke volume variation. Thus fluid versus vasopressor therapy can be tailored to individual patient's needs rather than general application of formulae (34, 35).

An endotracheal (ET) cardiac output monitor incorporated in the cuff of an endotracheal tube is also available. Based on the principle that the electrical resistance of blood changes when it moves or changes in volume, flexible electrodes on the ET cuff utilize information from an arterial pressure line and thus continuously calculate stroke volume (36). The transesophageal Doppler, supplying continuous real time objective data, monitors preload conditions and helps optimize cardiac contractility and the effect of afterload impedance on left ventricular performance (37). Such a monitor would have been indicated in this case.

Quantification of non-hydrostatic pulmonary edema may be used to predict mortality and morbidity and be used to guide fluid therapy and ventilator strategies (38). Bedside assessments may be made using dilution methods and by ultrasonography, monitors that are standard in many ICU settings (38, 39).

It would appear that in most cases there is a need to restrict and reevaluate perioperative fluid management. Preoperative volume loading is rarely necessary. After a careful review of available literature and studies it appears that the classic "third space" as postulated by Shires over 50 years ago does not exist (40). Crystalloid and colloid overload have deleterious effects. Routine replacement of insensible losses is unnecessary. Demand related regimens should be followed to improve patient outcome. Restricting excessive administration of fluids that are quickly redistributed outside the vascular space minimizes perioperative shifting. Fluid balance should be maintained. Inappropriate intravenous fluid therapy is a significant cause of patient morbidity and mortality. It remains to be seen whether colloid administration should be abandoned or used in small amounts with reduced crystalloids. Intraoperative Hb determinations are far from reliable as an indicator of need to transfuse. Guidelines that note that transfusion is rarely needed if the Hb level is 7gm do not generally take the patient's age, cardiovascular state or other co morbidities into account or even the rate of blood loss although they do state that these guidelines may not be applicable to all patients (41). But traditions die hard. Evidenced based medicine is still new. Many believe (in spite of all the evidence to the contrary) that blood pressure, heart rate, urine output, blood loss can all be "optimized" by giving more fluid. So when parameters fall out of range, we tend to give more fluids, without looking further for other causes of the perturbations. Evidence against this thinking is mounting.

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