



Chapter 12

Management of the REBOA patient in the Intensive care unit

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So your REBOA patient has made it off the table, and is being taken to the intensive care unit. With a bit of luck, things have gone well in the operating room, and the balloon has already been deflated. On the other hand, things may not be so rosy, and the balloon is still “up”. What now? Those who like to base their practice on evidence will be disappointed. Current experience of REBOA is still limited, and reports of this experience are even fewer. In particular, there is no specific guidance on how to manage the REBOA patient in the intensive care unit (ICU). However, much can be inferred from the post-operative management of trauma patients in general, and that of patients who have undergone surgery for vascular disease. There is now even limited experience with REBOA placed in the ICU due to circulatory collapse (not only in trauma).

What are the issues?

On arrival in the ICU, your patient will probably be cold, acidotic, coagulopathic, on vasopressors, anuric, and have associated injuries, which have not been fully evaluated or solved! Anything better than this, and you have lucked

out. A good handover between the anaesthetic/surgical and ICU team should, in addition to the usual details, include information on when the balloon was inflated and, if applicable, deflated, ischemia time or whether there had been trial deflations, or partial occlusion (pREBOA, iREBOA). Obviously, total time of occlusion is important as well as localisation (Zone I? Zone III?).

Resuscitation

Unless the anaesthetic and surgical teams have done an outstanding job, your patient will require further resuscitation, and repayment of the “ischaemic debt” (see text elsewhere in this manual). The quality of your resuscitation will be a key determinant of outcome. This applies particularly if the balloon is still inflated. Balloon deflation will result in redistribution of the blood volume, and unless the patient has been well resuscitated, will result in catastrophic haemodynamic deterioration. Balloon deflation should therefore be treated with the same respect as removal of the aortic cross-clamp, following abdominal aortic aneurysm repair.

Even if the balloon has been deflated successfully, it is likely that the patient will have ongoing resuscitation needs. In other words, having hemodynamic stability does not mean that the patient is well! The aim now should be to reverse hypothermia, acidosis, and coagulopathy, and to discontinue vasoactive drugs, as quickly as possible. The endpoints of resuscitation are a normal temperature, normal base deficit, normal lactate level, normal coagulation tests (whatever type you are using), the absence of vasopressor infusions, and – ideally – a good urine output. In addition, lower extremity circulation should be “normal”. Remember that you have just accessed the patients femoral vessels...

Usually, the situation will require the infusion of further blood products. Haemoglobin concentration is not an endpoint of resuscitation – haemoglobin levels can be normal, despite profound blood loss. In the face of persistent metabolic derangement, haemostatic resuscitation, with packed red cells, plasma, platelets, and cryoprecipitate, should be continued, and continued quickly, as prolonged shock will exacerbate the systemic inflammatory response. A “1:1” ratio of units of packed red cell concentrate and thawed plasma is a reasonable strategy. Unless already in place, additional venous access may be required. The easiest way to achieve this is usually by means of a “Cordis”,



or pulmonary artery catheter introducer sheath, in the subclavian or internal jugular vein(s). You might want to use a femoral vein sheath as well (depends also on your local routines), and replace it by central-line later on.

The use of synthetic crystalloids should be minimised. These fluids contribute to hyperchloraemia, and therefore metabolic acidosis, and haemodilution. There is no place for synthetic colloids in trauma resuscitation either. Buffering fluids (bicarbonates) should be considered, even though there is no clear evidence for their use.

Attention should be paid to electrolyte abnormalities: Hypocalcaemia is common, and can have profound effects on myocardial contractility and vascular responsiveness, and should be corrected by giving Calcium chloride or gluconate. Similarly, hyperkalaemia may result, as a consequence of administering blood products and acute kidney injury or due to release of intracellular potassium. Acidosis may also cause hyperkalemia. Serum potassium concentration should therefore be checked regularly and, if necessary, treated with insulin and renal replacement therapy. Hypothermia should be prevented or, if it has already occurred, be proactively treated. Under-body devices, such as heated mattresses, are particularly useful. On-the-body, forced-air devices, are less effective, but can be used in addition to under-body devices.

Monitoring

Unless there are comorbidities – in particular, cardiorespiratory disease – sophisticated cardiovascular monitoring is not required. The patient should have an arterial line, to allow the continuous monitoring of blood pressure, and to facilitate blood sampling. Some arterial access sheaths will permit pressure transduction. The patient should also have dependable venous access, which will often mean one or more central lines. However, these are used primarily for infusion, rather than monitoring. There is no value in measuring central venous pressure in trauma patients (or, indeed, any patient) – central venous pressure is not an endpoint of resuscitation. Advanced haemodynamic monitoring devices – such as PiCCO™, LiDCO™, CardioQ™, or Flotrac/Vigileo™ – also add relatively little during this phase. The diagnosis of haemorrhagic shock is usually obvious from other, more easily obtained parameters, and the resuscitation endpoints described above are more useful than calculated cardiovascular performance parameters. When there is genuine concern

about cardiac dysfunction, whether chronic or as a result of injury, or indeed over-resuscitation, a bedside echocardiogram (if necessary repeated) is usually best at quantifying preload as well as contractility. Abdominal hypertension and abdominal compartment syndrome (ACS) may develop, and intravesical pressure should therefore be monitored. For this Foley manometer can be used with ease. Elevated pressure of more than 12mmHg is by definition abdominal hypertension (but might be effected by position, pain and other factors). A patient with elevated pressure more than 20-24mmHg, or/and with signs of multiple organ failure (MOF) as decreasing urine output, is by definition ACS. These patients need aggressive treatment with different methods to decrease the pressure. If you don't take a pro-active approach, these patients will die. More on ACS and treatment can be found in other books or on the internet (e.g., world society of ACS).

As we know on rAAA patients, even if your patient does well at on day 1, don't get too happy. Keep the patient in the ICU, see a bit more, see that all metabolic issues have been resolved and monitor REBOA patients carefully. You might meet some surprises on day 2, which leads us to the next issue...



Figure 1: 12Fr sheath for REBOA used for blood pressure monitoring. Was used also during REBOA for pREBOA monitoring.



Post-reperfusion syndrome

REBOA, by design, gives rise to regional or indeed hemicorporeal ischaemia. (see the REBOA chapter) The magnitude of the insult is proportional to the completeness and duration of balloon inflation. Ischemia results in depletion of intracellular energy stores. When perfusion is restored, a post-reperfusion syndrome ensues, which involves a cascade of subsequent injuries, including activation and adhesion of leukocytes and platelets, generation of inflammatory mediators, calcium influx into cells, disruption of cellular membrane ion pumps, generation of free radicals, and cell death. Clinically, the syndrome is characterized by edema, further exacerbating intravascular volume depletion, and washout of myoglobin, potassium, lactate, and microthrombi into the systemic circulation, exacerbating hyperkalemia and hypocalcemia, and causing rhabdomyolysis and renal failure, ACS, and cardiac arrhythmias. All of these complications have to be anticipated and actively treated. Recognition can, however, be difficult when the sequelae of post-reperfusion syndrome are superimposed on the manifestations of hemorrhagic shock and hypoperfusion. In particular, when acute kidney injury has ensued, then using the resolution of metabolic acidosis as an endpoint of resuscitation is no longer possible. The situation requires experience and mature judgement.

Systematic inflammation

Injury, REBOA, operation, and post-reperfusion syndrome will all contribute to systemic inflammation. The systemic inflammatory response syndrome (SIRS) was first defined in 1991 at the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference. SIRS is a physiological state, rather than a diagnosis. SIRS is commonly seen in trauma patients, as well as in patients undergoing abdominal aortic aneurysm repair. The treatment of SIRS is that of the underlying causes. Nevertheless, it is a useful concept, and both the duration and resolution of SIRS have been shown to be useful prognostic markers.

Anticipating complications

The use of REBOA is associated with a number of potential, and serious, complications. These may **relate to the access site** – bleeding is the most obvious, and relatively easily treated. The development of thrombosis, and subsequent acute limb ischaemia, may be more insidious, but can have devastating consequences. Repeated examination, and if necessary imaging, of the limbs is therefore essential.

The possibility of developing compartment syndromes has already been alluded to. These may occur in the limbs, necessitating early consideration of the need for fasciotomies, or in the abdomen. ACS which we mentioned before, may result from a combination of pelvic and retroperitoneal haematoma formation, as well as reperfusion-related edema, particularly of the gastrointestinal tract, even when not frankly ischemic. Frequent intravesical pressure measurements (the Foley manometer) may help in identifying the development of the syndrome.

Mesenteric and renal ischaemia and, if prolonged, thrombosis, are more likely to occur with intentional Zone I placement, or inadvertent Zone II positioning. Mesenteric vascular thrombosis would be a devastating complication which is, however, difficult to detect – particularly in the context of an established systemic inflammatory response. Deviation from the anticipated clinical course, whether characterised by an “excessive” inflammatory response, or lack of resolution, should prompt a search for possible causes. However, recognising such deviations from the expected course is exceedingly difficult and, once again, requires experience, judgement, and a high degree of suspicion.

Acute tubular necrosis, whether caused by Zone I balloon placement, or haemorrhage-related hypotension, perhaps exacerbated by the administration of contrast material, will occur in many patients who have been treated with REBOA. The metabolic consequences of acute kidney injury will complicate the assessment of shock, adequacy of resuscitation, and fluid management. Supervening renal failure will result in a non-lactic metabolic acidosis, by contrast with the lactic acidosis caused by hypoperfusion, and the measurement of lactate levels may help to distinguish between these two conditions. Anuria and hyperkalaemia will necessitate renal replacement therapy. As in other conditions, the choice of modality – haemofiltration, haemodiafiltration, or haemodialysis – is probably not important.



Associated injuries

Patients who required REBOA typically did not complete even the “primary survey” of assessment. Based on the ATLS “ABCDE” (Airway, Breathing, Circulation, Disability, Exposure) paradigm, these patients only advanced to the assessment and treatment of “C”. If the more modern “<C>ABCDE” concept is considered, where “<C>” signifies the treatment of catastrophic haemorrhage, then assessment is even less complete. It is therefore imperative that patients undergo a thorough secondary survey as soon as they arrive in the ICU, and subsequently a tertiary survey. These assessments may involve additional imaging, which may have to be deferred until the patient’s haemodynamic and metabolic status permits. Nevertheless, patients should be thoroughly examined, at the earliest possible opportunity, to identify other injuries requiring urgent treatment.

There are many other issues that you have to think about. For example, the need of antibiotics, the presence of synthetic materials used (grafts or endografts), any bowel perforation, and the manifestations of systemic inflammation and multiple organ dysfunction. What is the circulation to the target organs like? To the extremities? If this a healthy young patient or someone with multiple co-morbidities that complicate the situation? All of these factors and more should be considered when approaching patients who have been treated with REBOA or other EVTm treatments, as maybe is the case for all trauma and bleeding patients.

Comment:

- » The role of ECHMO in trauma is not clear, but we might speculate that we will soon hear more on that issue.

Take-home points

Experience of managing REBOA patients in ICUs is limited. However, as with most ICU patients, outcomes are optimised by attention to detail, frequent re-evaluation, “doing simple things well”, a high index of suspicion, and a healthy dose of good judgement. Endovascular tools can save time and patients, but there is a need for re-thinking and frequent re-evaluation in the ICU. **“It’s not over until it’s over!”**