Citrate Update – June 2019

It has been 1 year since we introduced citrate and we wanted to briefly share some **valuable learning points** as well as highlighting important changes to the protocol.

Citrate Accumulation (toxicity) vs Excessive Citrate Metabolism

The above 2 terms are incorrectly used inter-changeably by staff. These are two separate conditions which require different treatments to prevent harm.

1. Citrate accumulation (toxicity) - ACIDOSIS

The more serious of the 2 conditions. Accumulation of unmetabolised citrate within the body resulting in potentially life threatening metabolic acidosis (citrate is an acid until metabolised to bicarbonate). Patients at risk are those unable to metabolise citrate including patients with severe lactic acidosis or acute liver failure. See protocol on how to identify and manage these patients. Signs citrate accumulation (toxicity) is occurring include (more than one of):

1. Marked drop in systemic ionised calcium (increasing need for calcium replacement)
2. Unexplained metabolic acidosis
3. Calcium T:I ratio > 2.5
4. Elevated total calcium > 3mmol/L (do not correct for albumin)

2. Excessive Citrate Metabolism – ALKALOSIS

Cause likely multifactorial, some patients are more susceptible. But generally as filter becomes less efficient/clogs more citrate ends up in the patients and less in the waste product of dialysis. In normal working conditions 50% of citrate ends up in the patient and 50% in dialysate. The excess citrate is metabolised to bicarbonate causing a metabolic alkalosis.

On ward rounds check patients bicarbonate and BE when on CVVHD. A BE of + 3 should trigger a review. **Several patients have ended up with a bicarbonate of > 40 due to failure to recognise.** See protocol on how to treat but generally change the filter in first instance, if not successful changes to the dialysate flow to blood flow ratio may be made to deliver less citrate to the

patient. This will mean moving away from the 20:1 ratio which is allowed in this circumstance. Changes to the blood flow and dialysate flow are likely to take several hours to have an effect. In rare circumstances citrate may need to be stopped if above measures do not work.

Hyponatraemia (Na <125mmol/L)

Rapid correction of hyponatraemia of more than 48 hours duration is not recommended. Hyponatraemia should not be corrected by **>8-10mmols**/day due to the real risk of **central pontine demylination.**
CVVHD may result in rapid correction of sodium in hyponatraemic patients due to 2 mechanisms

1. Citrate is presented as sodium citrate 4% this contains 408mmol/L of sodium. Therefore hyponatraemic patients on citrate therapy may experience a more rapid correction in sodium than desired.
2. When not using citrate the Multibic filter bags contain 140mmols/L of sodium. As the patient is dialysed against these bags there may be a more rapid correction in sodium than desired.

Management – ways to prevent rapid correction of sodium in hyponatraemic patients requiring CVVHD:

1. When choosing to filter a hyponatraemic patient choose a much lower dose of dialysis than you would normally i.e for a 90kg patient choose a dose of 25mls/kg but instead set the patients weight as <60kg – see table 2.
2. Monitor the patient's sodium hourly and if the rate of rise of sodium is too rapid you may need to temporarily stop the filter for several hours to prevent complications.
3. Infuse 5% dextrose at a set rate while the patient is on the filter to try and prevent rapid correction of sodium.

Hypernatraemia – maintaining therapeutic Hypernatraemia while on CVVHD

Patients with the following conditions may require targeted therapeutic hypernatremia 145-150mmol/L while on CVVHD.

- Fulminant liver failure with concerns regarding cerebral oedema
- Traumatic brain injury patients at risk of raised ICP

It can be difficult to maintain sodium at the desired level as the patient is being continuously filtered against a fixed concentration of sodium in the dialysate bag

Management
Commence continuous infusion of 5% hypertonic saline to maintain sodium within the desired range

- Commence infusion rate at 50mls/hour increasing as required up 100mls/hour

Acute liver failure patients

Patients admitted to ICU with acute liver failure (fulminant hepatic failure) should undergo CVVHD with **no anticoagulation including no citrate in the first instance**

Acute liver failure patients are at risk of cerebral oedema, risk factors include:

- Age < 40, paracetamol overdose, multi-organ failure, high vasopressor requirement

To reduce the risk of life threatening cerebral oedema in intubated patients with any of the above risk factors aggressive CVVHD should be commenced (even in the absence of significant renal failure) without anticoagulation.

- The blood flow should be set to 350mls/min
- The dialysate flow should be set to the maximum allowed of 4.8L/hour

Starting IV heparin – for alternative reason i.e PE

Keep citrate going if no contra-indications.
No pre-dilution as before so best to continue citrate along with other anticoagulation if no contraindications

Premature filter failure/clotting of filter on citrate anticoagulation

1. Check Vascath
    - Easy aspiration/flush? Is it kinking with patient movement?
    - Anticoagulation **CANNOT** compensate for poor vascular access
    - Consider replacing vascath
    - Beware reversing the lines makes the vascath much less efficient by causing recirculation – it may be better to insert a new vascath
2. Consider pro-coagulant illness
    - **HIT (this is one of the most common reasons for filter clotting)**
    - DIC, Antithrombin III deficiency or other haematological problem
    - Hyperlipidaemia or propofol infusion syndrome

- **Discuss with haematology if concerned** - further urgent investigations maybe warranted

3. Increase flow rates
    - Consider increasing blood flow rates. Note the blood flow to dialysate flow rate must remain at a ratio of 20:1. See table 3 this has higher blood flows as the dialysate rate is set at 35mls/kg/hour.
4. Ensure target post filter calcium results within specified range of 0.25-0.34
    - Consider reducing target filter post filter calcium to 0.2 – 0.24 by increasing citrate rate (if not suitable for IV heparin in addition to citrate)
5. Start IV heparin (if no contraindications) or other anticoagulant in addition to regional citrate anticoagulation

Actual body weight

Use actual body weight when selecting patient's dose of renal replacement therapy

What if the patient does not have an Arterial Line

Systemic calcium results can be taken from a peripheral vein if there is no arterial line. However, this sample **must not** be taken from a vein that is close to the vascath as you are likely to pick up an incorrect calcium level in the patient. I.e. you can't take the venous sample from a LIJ central line even though the vascath in the RIJ as they both terminate in the same place.

Low Pressure Return Alarm on filter

We have not changed to the recommended Fresenius vascath (shotgun catheter for a variety of reasons). As the new Fresenius filter machines use lower blood flow than previous filters and our current Vascaths are wider than the recommended Fresenius vascaths this can occasionally result in the machine detecting low pressure on the RETURN limb of the circuit. This may result in circuit loss. See protocol on how to solve this problem

Extending filter life in future

May extend filter life from 72 hours in the future to 96 or 108hours. Will likely lead to more excessive citrate metabolism as the filters will be more prone to clogging. **Need to improve our management of excessive citrate metabolism before we do this.**

Oliver Robinson and Jen Service (please contact us if you have any questions)