

Anaesthesia For Removal Of Missing Guidewire. A Case Report

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Citation

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Abstract

A 41-year-old female was booked for guide wire removal under general anaesthesia. She was admitted through the renal unit with a diagnosis of acute renal failure secondary to sepsis. She was scheduled to undergo sessions of haemodialysis. However, the guide wire for cannulation was lost at attempt to gain vascular access for the procedure. An urgent fluoroscopy was done to localize the guide wire and she was then booked for surgery. When all conservative treatment modalities fail, a haemodialysis is indicated in patients with renal insufficiency.¹ For the purpose of haemodialysis, a vascular access with a flow of at least 200ml/minute is needed.¹ Like every other invasive procedure, the process of cannulation is fraught with complications which may include sepsis, kinking of guide wire or inadvertent arterial puncture.² Loss of the guide wire itself is rare in the literature. The anaesthesia for the surgical removal of the guide wire as an emergency in a uraemic patient is presented.

CASE REPORT

A 41-year-old female booked for guide wire removal under general anaesthesia. She was admitted through the renal unit with a diagnosis of acute renal failure secondary to sepsis. She was scheduled to undergo sessions of haemodialysis. However, the guide wire for cannulation was lost at attempt to gain vascular access for the procedure. Fig.1 An urgent fluoroscopy was done to localize the guide wire and she was then booked for surgery. Fig 2

Other investigations done with their results were:

Haemoglobin.....8.7g/dl

Platelets..... 4×10^9

ESR.....105/1ST hour

The liver function test was normal, but the serum potassium, urea and creatinine were deranged. She was on tablets of amlodipine 10mg daily, and hydrochlorothiazide, 50mg daily.

At preoperative assessment, she was obese, pale and obtunded with generalized body swelling. She had a Mallampati II airway. She was hypertensive with a blood pressure of 170/100mmhg. There were bilateral coarse crepitations on her lung fields. She had massive ascites with fluid thrill. An assessment of ASA III E patient was made.

Because of her obtunded state, and obesity, the choice of regional anaesthesia was not favoured and we scheduled her for an emergency general anaesthesia. She was transfused 500mls of blood under frusemide cover. Intravenous injection of ranitidine 150mg was given for acid prophylaxis.

In theater, we secured an intravenous access on her left hand with an 18-gauge cannula. An omnicon multi-channel monitor comprising ECG, SpO₂, BP and capnography were connected and baseline values noted. She was preoxygenated with 100% oxygen for 5 minutes and an SpO₂ of 100% achieved. We induced her with intravenous propofol, 2mg/kg(200mg), and cricoid pressure was applied by an assistant. Intravenous fentanyl, 100 µg was administered, followed by atracurium, 50mg and she was manually ventilated. The larynx was intubated with a size 8 cuffed endotracheal tube. Anaesthesia was subsequently maintained with isoflurane at 0.5 volume%. She was mechanically ventilated at 18 cycles per minute with a tidal volume of 600ml and PEEP of 5cm. Vital signs were continuously monitored throughout the procedure which lasted 45minutes. The surgery involved dissection of the groin to locate the missing guide wire which was found to measure 30cm along the femoral vein. Fig 3. A venous access on the contra lateral leg was also established for an immediate dialysis.

At the end of the procedure, the isoflurane was discontinued

and residual muscle paralysis in the patient antagonize by intravenous neostigmine, 2.5mg administered with 0.8mg of glycopyrrolate. The patient was extubated in theatre and received oxygen by face mask for 10 minutes before being transferred to the recovery room where we continued oxygen by nasal prongs at 2l/minute. She was subsequently transferred to the renal unit where she had a successful haemodialysis.

DISCUSSION

When all conservative treatment modalities fail, a haemodialysis is indicated in patients with renal insufficiency.¹ For the purpose of haemodialysis, a vascular access with a flow of at least 200ml/minute is needed.¹ Like every other invasive procedure, the process of cannulation is fraught with complications which may include sepsis, kinking of guide wire or inadvertent arterial puncture.² Loss of the guide wire itself is rare in the literature. The anaesthesia for the surgical removal of the guide wire is an emergency procedure in a uraemic patient.

At preoperative assessment, it was important to have a quick review of the patient's case note and ascertain, if possible, the cause of renal failure. The common causes of renal failure requiring chronic dialysis include diabetes mellitus, arterial diseases and glomerulonephritis. Our patient was a known hypertensive with sepsis. Marked chronic anaemia is common in renal patients, and patients often required peri-operative blood transfusion as in this case.^{3,4} Uraemia results in a prolonged bleeding time, due to a low platelet count and reduced platelet function. Prothrombin time and partial thromboplastin times are usually normal. Delayed gastric emptying is common, and an H₂ receptor antagonist, metoclopramide, or sodium citrate may be administered preoperatively. We administered ranitidine to this patient.

The patients are often anxious, and so it is important to establish a rapport, and explain the procedure. Anxiolytic premedication, e.g. temazepam, may be required. Our patient was obtunded and as such, sedative premedication was unnecessary. Anaesthesia may be induced slowly with propofol, thiopentone or etomidate, whilst monitoring haemodynamic parameters, and titrated to effect if a rapid sequence technique is not required. Propofol has been successfully used for total intravenous anaesthesia for kidney surgery, and is associated with a reduction in postoperative nausea and vomiting.^{5,6} Propofol was our choice for induction.

The patient's airway was protected with an endotracheal

tube, as uraemic patients are at risk of aspiration.³ Suxamethonium in intubating dose causes a rise in serum potassium averaging 0.5 mmol l⁻¹, (maximum of 0.7 mmol l⁻¹) in patients with renal failure.⁷ Cardiac arrest and death have been reported in patients with preexisting hyperkalaemia, and in those given repeated doses.⁸ It should not be administered to patients with serum potassium concentrations >5.5 mmol l⁻¹, or those with uraemic neuropathies. Under these circumstances we modify any rapid sequence induction technique to avoid its use. Atracurium besylate was used to achieve muscle relaxation. Because of the patient's hypertension, intravenous fentanyl was used to blunt the stress response to laryngoscopy and tracheal intubation.

The non-depolarising relaxants, atracurium, and cis-atracurium, are suitable as their excretion is independent of the kidney.⁹ Atracurium has theoretical advantages as it is also broken down by Hofmann degradation.⁹ Pancuronium is best avoided as its action may be prolonged, 80% being eliminated through the kidneys.

Isoflurane was our inhalational agent of choice as only 0.2% is metabolised, it produces low levels of inorganic fluoride ions, and causes few cardiac arrhythmias.¹⁰ It may also have less effects on cardiac output and renal blood flow than other agents. Enflurane fluoride levels approach 75% of nephrotoxic levels, and it is not recommended.¹¹ Halothane has been used extensively, but its arrhythmogenic potential may be enhanced in these patients.¹¹

Fentanyl may be used in normal doses, as excretion is mainly by hepatic metabolism.¹² Morphine can cause prolonged effects, e.g. sedation and respiratory depression in renal failure, because the active metabolite, morphine-6-glucuronide, accumulates. It should be titrated carefully, and prolonged effects should be anticipated for a given dose. Meperidine has no particular advantages in these patients, and normeperidine can accumulate.¹³

During routine surgery in patients with co-existing renal failure, intravenous fluids are often minimised, to prevent fluid overload and reduce the need for postoperative dialysis. The type of intravenous fluid used is less important. Normal (0.9%) saline is a logical choice, as it is high in sodium, (particularly important if mannitol is used), and contains no potassium or lactate. Albumin and colloids have also been advocated. Blood should be transfused if required. Intra-operative blood loss was less than 250 ml in this patient.

The patient was closely monitored in the immediate postoperative period, with particular attention to fluid balance. Urine output must be monitored constantly. Hypovolaemia must be avoided, and isotonic 0.9% saline at 30 ml h⁻¹ plus the previous hour's urine output is usually appropriate. It should be adjusted according to blood results. Additional fluids may be required to maintain the CVP, which may fall as a result of third-spacing of fluids, or blood loss. Hyponatraemia and hyperkalaemia may occur associated with mannitol. Blood should be transfused if necessary.

CONCLUSION

Patients with renal failure rarely receive general anaesthesia without either a prior haemodialysis or optimizing their renal status. In the occasional patient that must be anaesthetized without these measures, meticulous attention to details and avoiding factors that could worsen an already bad clinical situation should be followed in order to have a favourable outcome.

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