

Cardiac complication secondary to jugular catheter insertion in a renal failure patient

Manickam RANGASAMI ¹, Muhammad KHALIL ¹, Jayashree RANGASAMI ², Jackson Chee Seng TAN ¹

¹Department of Renal Medicine and ²Department of Internal Medicine, Suri Seri Begawan Hospital, Kuala Belait, Brunei Darussalam

ABSTRACT

Central venous catheterisation is a common procedure performed for emergency dialysis. It is usually carried out without any cardiac monitoring. Cardiac arrhythmias with associated conduction blocks are rare complications. The underlying pathogenesis is trauma to the endocardium by the guide wire or catheter. It occurs more frequently in patients with acute renal failure and azotaemia than patients with established end stage renal disease. Disturbances in acid base balance and electrolyte abnormalities are contributing factors. Fortunately, most are benign but occasionally can lead to potentially fatal arrhythmias. We report a case of a 46-year-old lady with end stage renal failure secondary to diabetes mellitus who developed runs of transient ventricular ectopics and right bundle branch block during internal jugular catheterisation. This spontaneously resolved 12 hours later.

Keywords: Arrhythmia, atrioventricular block, central venous catheterisation, dialysis, heart blocks, right bundle branch block

INTRODUCTION

Central venous catheter (CVC) insertion for dialysis is a common procedure performed in renal units. In many large renal centres, this procedure is usually done under ultrasound guidance and cardiac monitoring especially when performed by interventional radiologists. Cardiac arrhythmias and conduction blocks are not uncommon complications and

are reported to occur in up to 12% of procedures. ¹ Fortunately, most are transient. However, potentially fatal arrhythmias can occur. Patients with severe azotaemia and metabolic disturbances from acute renal failure are at higher risk for developing these complications. Other non-arrhythmia complications include pneumothorax, haemothorax, haemopericardium, perforation and cardiac tamponade. ^{2,3}

We report a case of a 46-year-old lady with end stage renal failure secondary to diabetes mellitus who developed runs of transient ventricular ectopics and right bundle branch

Correspondence author: Jackson TAN
Department of Renal Medicine,
Rimba Dialysis Unit,
Bandar Seri Begawan, Brunei Darussalam.
Tel: +673 2450483, Fax: +673 2450483
E mail: drjacksontan@yahoo.co.uk

block (RBBB) during internal jugular catheterisation. This spontaneously resolved 12 hours later.

CASE REPORT

A 46-year-old lady was admitted with a history of tiredness, feeling unwell and persistently vomiting. She was known to have stage IV chronic kidney disease from underlying diabetes mellitus. At the time of admission there was no evidence of volume overload or haemodynamic compromise. Routine examination was unremarkable.

Investigations revealed metabolic acidosis (HCO₃ 13.8 mmol/L, range 22 to 26) and elevated urea (43 mmol/L, range 3.2 to 7.4) and creatinine (1,280 µmol/L, range 53 to 110). She was however normokalaemic and normocalcaemic. As she was azotaemic, the decision was made to start dialysis. Electrocardiogram (ECG) on admission revealed sinus rhythm (Figure 1).

After informed consent, she was prepared for right internal jugular catheter insertion. The patient developed mild chest discomfort and palpitations during the insertion of the guide wire using the Seldinger technique. The cardiac monitor showed runs of intermittent wide complex ventricular ectopics. This subsided following a slight withdrawal of the guide wire. However as the catheter was inserted over the guide wire, the patient developed RBBB (Figure 2). As the patient was haemodynamically stable and was asymptomatic, it was decided to complete the procedure despite the persistence of the RBBB. Post catheter insertion chest radiography showed the catheter to be in the correct position (Figure 3). Later she completed three hours of haemodialysis without any complications. A repeat ECG showed no new changes except for the RBBB.

Subsequent ECG performed 12 hours later revealed normal sinus rhythm (Figure 4) Echocardiogram did not show any wall motion

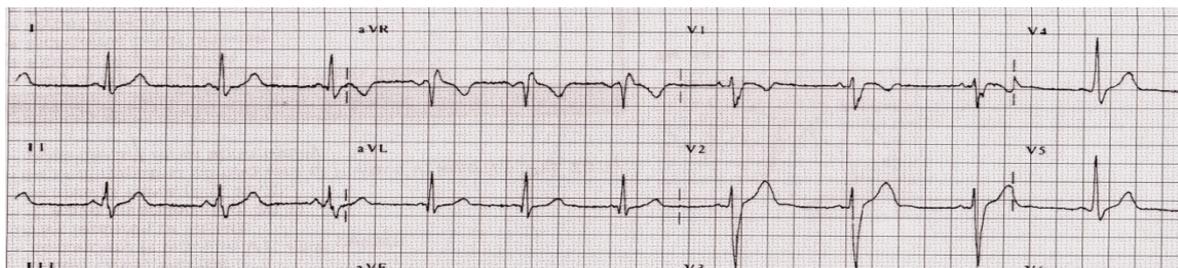


Fig 1. Normal sinus rhythm before catheter insertion.

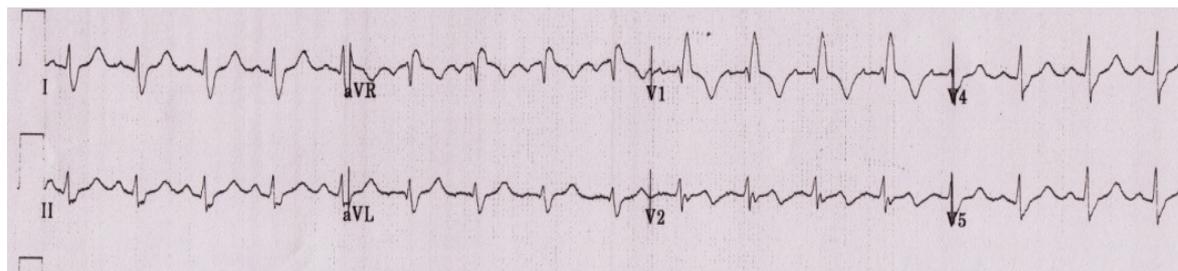


Fig 2. RBBB during catheter insertion.



Fig 3. Chest radiography showing normal position of catheter.

abnormalities or dyskinesia. Cardiac enzymes (creatine kinase and creatinine kinase MB) were also normal. Her renal function did not improve and she was initiated on long-term haemodialysis. There was no recurrence of RBBB in subsequent follow up ECGs when she was reviewed in the Renal Clinic.

DISCUSSION

CVC insertion either tunneled or non-tunneled is a routine procedure carried out in the management of renal failure patients with symptoms of azotaemia or volume overload. After CVC placement, the tip of the CVC should approximate the junction of the superior vena cava and the right atrium. However, there is no consensus on the optimal site for placement of the catheter tip.² Chest radiography

is usually done to confirm the position as there is a chance of the CVC going in to the ipsilateral subclavian, or contralateral brachiocephalic veins. Misplacement of CVCs can happen in up to 20% of procedures.⁴

The incidence of RBBB during the right jugular catheter insertion is reported to be around three to 12%.¹ Occurrence of left bundle, left anterior and posterior blocks along with RBBB have been reported.¹ One study showed that 41% of CVC insertion was associated with atrial arrhythmias, 25% with ventricular ectopy and 11% with ventricular couplets or advanced ventricular arrhythmias.⁵ Interestingly, ventricular ectopy was more common in patients with shorter stature and when CVC was inserted from the right sub-

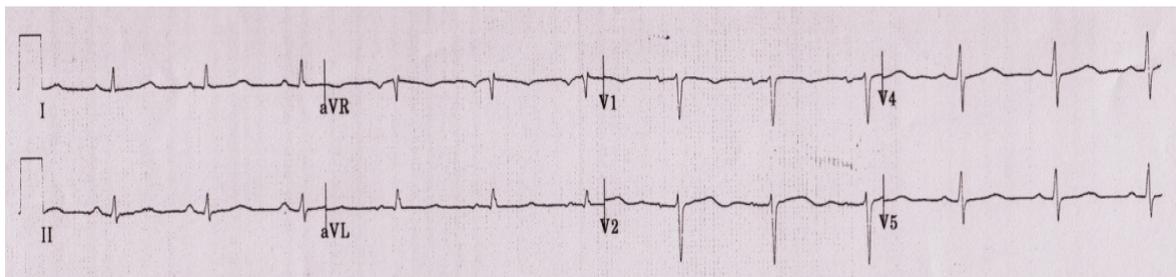


Fig 4. Normal sinus rhythm after 12 hours.

clavian route.⁵ Other variables such as age, cardiac history, serum potassium, type of procedure, and catheter brand were not significant. The conduction block is probably due to a block in the right bundle branch since it is located in the right ventricle below the tricuspid valves. The left bundle branch is less susceptible because it branches earlier in the septum.⁶ When it occurs with pre-existing left bundle branch block (LBBB), complete heart block can occur and there are reports of more dangerous ventricular arrhythmias and even asystole requiring immediate intervention.^{3, 5} Occasionally fibrillations, supraventricular and ventricular ectopics can occur with fatal complications.³

The mild trauma caused by the guide wire or catheter is probably responsible for the arrhythmias. The guide wire is inflexible and has a hard tip which makes it more likely to cause trauma.⁶ To minimise the complications, over-insertions of the both guide wires and CVCs should be avoided. Markings on the guide wires will help to reduce complications. The safe limit of guide wire insertion in an adult is 18 cm, and the tip of the guide wire can be one to three cm beyond the tip of catheter. Generally, the guide wire should not be inserted to more than 22 cm.⁶ The recommended depth of catheter is 13 to 16 cm through the right jugular or 15 to 20 cm through the left jugular vein.⁷ There are several techniques for identifying the approximate location of the catheter tip. Some of the methods used include right atrial ECG monitoring, trans-oesophageal echocardiography (TOE), use of surface land marks, use of Pare's formula where the patient's height in centimetre is divided by ten, and intra-atrial electrode to trace atrial P waves.^{4, 8} Ultra-

sound has also been found to be successful in placing CVCs with fewer complications.⁹ Interestingly, slippage of wire into the circulation has also been reported.¹⁰

The risk of arrhythmias during CVC insertion is higher in acute renal failure with azotaemia than those on regular dialysis as acid base disturbance and electrolyte abnormalities are more common in the former condition.¹ These increase the risk for complications. However, arrhythmia has been reported in patients with normal renal function.⁵ Conduction blocks during internal jugular catheterisation are uncommon as the catheter does not enter the heart fully but the guide wire can sometimes be over inserted. When the catheter insertion is done with no ECG monitoring complications may occur without being noticed.

Fortunately, in most cases, the arrhythmias are transient and asymptomatic. However if this occurs with a patient with pre existing LBBB, complete heart block and subsequent asystole can occur.² In our case, it was fortunate that the arrhythmias settled spontaneously without causing many symptoms. However, given the duration of the arrhythmia, it could have progressed to a more dangerous arrhythmia. In our case, the presence of left ventricular hypertrophy and uraemia may have also decreased her arrhythmogenic threshold and therefore triggered and prolonged her arrhythmia.

In conclusion, this case report highlights the importance of cardiac monitoring during the insertion of CVCs especially in the presence of azotaemia and pre-existing heart problems. Dangerous conduction blocks and

arrhythmias may be missed without cardiac monitoring and can be fatal.

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