IATROGENIC PNEUMOTHORAX FOLLOWING PLATE FIXATION OF THE CLAVICLE

Editor,

A 37-year-old right hand dominant male sustained a comminuted, displaced midshaft fracture of his left clavicle as the result of a motorcycle accident. He did not incur any other injuries. After discussing the treatment options, a decision was taken to proceed with open reduction and plate fixation of his left clavicular fracture. The procedure was performed under general anaesthesia in the beach chair position. A direct incision was made over the left clavicle and the fracture was exposed and reduced. The fracture was stabilised using a pre-contoured titanium plate with a combination of non-locking and locking screws. No concerns were reported in the peri-operative period by the anaesthetic team. A routine check x-ray of the left clavicle was obtained the following day which demonstrated an excessively long medial screw (Figure 1a) with a left apical pneumothorax confirmed on a chest radiograph (Figure 1b). The patient returned to theatre for insertion of a left-sided chest drain and screw exchange. The pneumothorax resolved and the patient’s left clavicular fracture proceeded to complete union.

Fractures of the clavicle are common representing 2.6 to 5% of all fractures and approximately 80% of fractures affect the middle third of the clavicle. The incidence of high-energy fractures with displacement, comminution and shortening is increasing and as a result operative fixation for such injuries is being performed more commonly. Infection, implant failure, non-union, scar-related pain, prominent hardware and refracture are the most commonly reported operative complications.

Plate fixation is the most common method of operative management. The plate is most commonly placed on the superior surface of the clavicle with screws inserted in a cranial-caudal direction potentially placing the lung apex and the neurovascular structures at risk during drilling and screw insertion. The risk however of either an iatrogenic pneumothorax or neurovascular injury is regarded in the literature as a rare occurrence. Some centres have recommended obtaining a chest x-ray routinely to exclude pneumothorax following clavicle fixation. Shubert et al. concluded from their study that due to the rarity of iatrogenic pneumothorax, radiation exposure and cost, in combination with the poor sensitivity of chest radiographs to detect pneumothoraces, obtaining a routine chest x-ray without clinical indication may be unnecessary.

Pneumothorax in relation to clavicular fractures is a well-described preoperative complication existing in the literature. In our case, the patient had a preoperative chest x-ray which did not demonstrate pulmonary trauma and given the excessive difference in length between the most medial screw and the adjacent screw we conclude that the patient incurred an iatrogenic pneumothorax due to surgical error. We acknowledge that intra-operative screening would have identified the long medial screw but the pneumothorax may not have been appreciated.

We emphasise the importance of careful surgical technique when performing plate fixation of a midshaft clavicular fracture, in particular, ensuring a guard is placed under the clavicle when drilling and close attention to screw length. Furthermore, we recommend careful scrutiny of postoperative clavicle radiographs due to the rare but potential risk of iatrogenic pneumothorax.

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Figure 1a (top): left clavicle check x-ray demonstrating an apical pneumothorax and an excessively long medial screw (white arrow); Figure 1b (bottom) demonstrating a left-sided pneumothorax (white arrow pointing to edge of lung).
The first reading was thought to be erroneous. Repeat sampling as would be expected with hyperkalaemia and initially this her ECG did not demonstrate dramatically peaked T waves demonstrated elevated potassium of 8.3 mmol/L. Interestingly, an arterial gas sample was taken at this point which assumption that she would require a pacemaker. Management was deferred to the cardiology team with the cerebral hypoperfusion in the context of CHB and her blood pressure. Dysarthria was felt to be secondary to ventricular rate remained 40 bpm albeit with a satisfactory 1). A further 1.8 mg atropine did not rectify her CHB and heart block (CHB) with a ventricular rate of 29 bpm (Figure 1). A further 1.8 mg atropine did not rectify her CHB and ventricular rate remained 40 bpm albeit with a satisfactory blood pressure. Dysarthria was felt to be secondary to cerebral hypoperfusion in the context of CHB and her management was deferred to the cardiology team with the assumption that she would require a pacemaker.

An arterial gas sample was taken at this point which demonstrated elevated potassium of 8.3 mmol/L. Interestingly, her ECG did not demonstrate dramatically peaked T waves as would be expected with hyperkalaemia and initially this first reading was thought to be erroneous. Repeat sampling however confirmed hyperkalaemia. Additionally, her formal laboratory biochemistry soon confirmed she was in acute on chronic renal failure with an eGFR of 12 ml/min which had deteriorated from a baseline of 30 ml/min. The hyperkalaemia remained refractory to conventional medical treatment and haemofiltration was commenced. Upon normalisation of serum potassium, her rhythm reverted to sinus of rate 74 bpm (Figure 2). Haemofiltration was weaned over the coming days and a permanent pacemaker was not required.

It emerged she had been taking both atenolol and Ramipril for hypertension and had recently commenced a NSAID for joint pain. This likely precipitated an acute nephrogenic insult resulting in the accumulation of atenolol causing further renal hypoperfusion and hyperkalaemia which, in synergy with B-blockade, precipitated CHB and cerebral hypoperfusion.

This case illustrates the recently coined BRASH syndrome (Bradycardia, Renal failure, AV-node blockers, Shock and Hyperkalaemia). This describes a series of events in a patient with CKD taking AV nodal blockers where an initial insult (such as dehydration or nephrotoxic medication) triggers a cascade of events where AV nodal suppression impairs the normal compensatory response to renal hypoperfusion thus causing renal decompensation resulting in worsening hyperkalaemia. The synergistic effect of hyperkalaemia and B-blockade on AV nodal function causes further decompensation resulting in a pathological downward spiral of events.

This is an under-recognised cause of CHB and renal failure which may be refractory to initial conventional treatment measures. ECG changes may not be characteristic of classical hyperkalaemia, occur at lower than expected serum potassium levels and remain refractory to conventional treatment. Co-morbid elderly patients on multiple medications are at high risk of developing this syndrome therefore as physicians we must be cognisant of prescribing AV nodal blockers or indeed additional nephrotoxic agents, so as to not incite the pathological cascade of events leading to BRASH syndrome.

**BRASH SYNDROME: AN UNDER RECOGNISED CAUSE OF COMPLETE HEART BLOCK IN THE ELDERLY**

**Editor,**

An 81 year old lady with a background of chronic kidney disease (CKD), hypertension and type 2 diabetes mellitus presented to Craigavon Area Hospital via ambulance as a stroke lysis call. She was dysarthric and profusely bradycardic with an unreadable blood pressure. Following administration of 600 mcg atropine a blood pressure of 100/60 mmHg was obtained. An ECG demonstrated complete heart block (CHB) with a ventricular rate of 29 bpm (Figure 1). A further 1.8 mg atropine did not rectify her CHB and ventricular rate remained 40 bpm albeit with a satisfactory blood pressure. Dysarthria was felt to be secondary to cerebral hypoperfusion in the context of CHB and her management was deferred to the cardiology team with the assumption that she would require a pacemaker.

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This is an under-recognised cause of CHB and renal failure which may be refractory to initial conventional treatment measures. ECG changes may not be characteristic of classical hyperkalaemia, occur at lower than expected serum potassium levels and remain refractory to conventional treatment. Co-morbid elderly patients on multiple medications are at high risk of developing this syndrome therefore as physicians we must be cognisant of prescribing AV nodal blockers or indeed additional nephrotoxic agents, so as to not incite the pathological cascade of events leading to BRASH syndrome.