EMERGENCY CASEBOOK

Case of the month: A case of airway obstruction following tenecteplase administration

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The value of thrombolysis in the treatment of acute myocardial infarction is well established. Haemorrhage into subcutaneous tissues is fortunately a rare complication of fibrinolytic administration. However, if as a result of trauma a haematoma develops within the neck following thrombolysis, it can lead to rapid airway compromise. This is the first reported case of tenecteplase administration leading to subcutaneous haemorrhage and consequent airway compromise. It is also the first reported case where the antecedent trauma was a jaw thrust.

A 70 year old man awoke at 0230 with chest pain. An ambulance was called, and paramedics administered intravenous morphine, high flow oxygen, aspirin 300 mg, and sublingual glyceryl trinitrate at his home. During the ambulance transfer he had a monitored ventricular fibrillation arrest. His obtunded airway was supported with a jaw thrust, and one direct current shock of 200 J was administered, returning the patient to sinus rhythm.

Upon arrival at the accident and emergency department, the patient had a Glasgow Coma Score of 15, was still experiencing chest pain, and a 12 lead electrocardiogram (ECG) was indicative of an acute anterior myocardial infarction (MI). His medical history included a thrombotic cerebrovascular accident 20 years previously, hypertension, hypercholesterolaemia, and previous smoking. His medication consisted of aspirin 75 mg once daily, atenolol, doxazocin, and simvastatin. Tenecteplase 10 000 U (50 mg) was given as a bolus, followed by a loading intravenous bolus of heparin 5000 U and a heparin infusion at 1300 U/hour. His ECG 90 min post-thrombolysis showed persistent ST segment elevation. He was therefore referred for emergency angiography and possible angioplasty.

He received 300 mg of clopidogrel prior to angiography. Successful percutaneous angioplasty was performed on his left anterior descending coronary artery and a further 5000 U of heparin was administered, as the activated clotting time was only 153 seconds. A unilateral, submandibular haematoma had been noted before the angiogram, and as the activated partial thromboplastin time ratio was found to be >7.27, heparin was discontinued. The patient went on to have 200 ml of haematemesis and was therefore given protamine. Anaesthetic review was requested because the patient became increasingly short of breath, had mild stridor, and a subjective sensation of choking. The supramandibular/submandibular haematoma was still developing, and therefore a Doppler image of the carotid arteries was carried out by the surgical team. A triphasic signal was found bilaterally, indicating that the carotids were patent. The evolving haematoma posed a potential threat to the patient's airway, and he was transferred to theatre for elective intubation. Fresh frozen plasma was administered, and the maxillofacial

surgical team were available in theatre. Airway assessment revealed mouth opening to be 2–3 fingers' width, and he had obvious supramandibular and submandibular swelling (fig 1A). Rapid sequence or gaseous induction was undesirable because of the potential loss of the airway and the recent history of MI.

The preferred method of securing the airway would have been fiberoptic nasal intubation with the patient awake. We opted for oral intubation as the patient was coagulopathic. He received 10 ml of nebulised 4% lignocaine over 45 minutes. A low dose propofol infusion was started along with a remifentanyl target controlled infusion. An fiberoptic oral intubation with the patient awake was performed via a Burman airway by the anaesthetic consultant. The fiberoptic view revealed minor supraglottic swelling, a normal

Figure 1 (A) Pre-intubation: airway assessment revealing mouth opening of two to three fingers' breadth. (B) Postintubation: lightly sedated with the endotracheal tube cuff uninflated on CPAP of 100 mm H₂O. Consent was obtained from the patient for publication of these images.

Abbreviations: ECG, electrocardiogram; ETT, endotracheal tube; MI, myocardial infarction

epiglottitis, and primarily sublingual and submandibular swelling. A size 6 cuffed oral endotracheal tube (ETT) was passed over the scope but the cuff was not inflated (fig 1B). The patient experienced no chest pain or ECG changes during intubation, and systolic blood pressure remained between 120 and 135 mmHg.

Over night on in the intensive care unit, the patient received dexamethasone and minimal sedation. His ETT cuff remained deflated and he breathed spontaneously with continuous positive air pressure at 10 cm H₂O. Upon review in the morning, the haematoma was stable and the patient was able to talk around his ETT. He was therefore extubated uneventfully.

DISCUSSION

Of the few reported cases of haemorrhage into the subcutaneous neck tissues post-thrombolysis, none were linked with tenecteplase. The ASSENT-2 study¹ compared the efficacy and safety of tenecteplase to the more commonly used alteplase. The findings were that the 30 day mortality rates were almost identical (6.18% for tenecteplase and 6.15% for alteplase) but that there were fewer non-cerebral bleeding complications following tenecteplase (26.43% v 28.95%, $p=0.0003$). As with any thrombolytic agent, caution is advised with its administration following trauma. Overenthusiastic jaw thrust can lead to soft tissue trauma and even result in jaw dislocation.

Of the five other documented cases, three required endotracheal intubation.²⁻⁴ Two of these cases were not associated with any trauma,^{2,3} but the other cases were associated with a preceding localised trauma. The trauma was very specific in two cases; tongue biting resulting in lingual haematoma⁵ and laceration with Magill's forceps leading to uvular haematoma.⁶ The tenuous history of trauma in our case and its cautionary significance for thrombolysis would only be picked up upon if actively sought. If a history of this nature is elicited, while not contraindicating thrombolysis, it should lead to subsequent close observation of the patient (on the high dependency/intensive care unit).

How and when do you manage the airway in these patients? Causing stress, pain, and tachycardia on the background of a recent MI is obviously undesirable. Further bleeding due to a traumatic intubation secondary to distorted anatomy could lead to total airway obstruction. In our case, a nasal intubation would have been preferable, but the risk of bleeding and possible laryngospasm favoured the oral route. We decided to intervene and protect the airway pre-emptively rather than risk further compromise and have to carry out a physiologically stressful and dangerous intubation later. In such cases, an experienced surgeon who is able to perform a surgical tracheostomy should be readily available in the event of a failed intubation. The intubation itself should be carried out by an anaesthetist who is experienced in difficult airway techniques.

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IMAGES IN EMERGENCY MEDICINE

Cocaine induced non-ST-elevation myocardial infarction: an uncommon electrocardiographic presentation

The incidence of myocardial infarction in cocaine associated chest pain is approximately 6%.¹ The most common cocaine induced electrocardiogram changes are non-specific ST segment changes, ST segment elevation, T wave inversion, and QT interval prolongation.

A young man was brought to the emergency department after having taken intranasal cocaine. He was agitated, tachycardic, hypertensive, and complaining of substernal, pressure-like chest pain. The electrocardiogram showed sinus tachycardia and a large magnitude ST segment depression in all leads except aVL and V2, and ST segment elevation in leads aVR and V1 (fig 1). Treatment with nitroglycerin infusion and aspirin was initiated, the chest pain resolved, and the electrocardiogram normalised. Cardiac enzymes showed a typical rise and fall curve. A two-dimensional echocardiogram, performed after the chest pain had subsided, showed normal left ventricular systolic function

with no segmental wall motion abnormalities. The patient refused cardiac catheterisation and left the hospital against medical advice.

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