

# DIFFICULT INTUBATION STYLOHYOID LIGAMENT CALCIFICATION

A M Aris, E O Elegbe, R Krishna

## ABSTRACT

*Intubation was difficult and traumatic in a 40-year-old patient presented for emergency oesophagoscopy because the diagnosis of stylohyoid ligament calcification was not suspected. High probability of stylohyoid ligament calcification should be suspected when there is difficulty in lifting the epiglottis and fibre-optic laryngoscopy is suggested as the best way to tackle this problem to prevent trauma and possible risk of regurgitation and aspiration especially in emergency situation.*

*Keywords : Intubation, Trachea, Complications, Calcified stylohyoid ligament*

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## INTRODUCTION

Many reasons have been put forward for difficulty in intubation<sup>(1)</sup>. Difficulty in intubation can often be anticipated prior to anaesthesia but occasionally unexpected difficulty may be encountered in particular in emergency situation. The case report describes a difficult intubation due to stylohyoid ligament calcification, a known cause of difficult intubation which was not recognised at the time of intubation.

## CASE HISTORY

A 40-year-old male African was seen at the emergency room with a history of a fish bone stuck to his throat. X-ray of the neck showed the position of the fish bone (Fig 1). Removal under local anaesthesia failed and he was therefore scheduled for removal under general anaesthesia. There was no previous history of anaesthesia or surgery. Physical examination did not reveal any abnormality. Haemoglobin, ECG and Chest X-ray were normal.

Anaesthesia was induced with thiopentone 250mg followed by 100mg of suxamethonium chloride intravenously. Several attempts at laryngoscopy and intubation failed. The epiglottis was resting against the posterior pharyngeal wall and the laryngoscope blade could not lift it to expose the glottis. Blind nasal intubation was attempted to no avail. Since the fish bone must be removed, the procedure could not be abandoned. The patient was however allowed to wake up and a fresh attempt at intubation with the fibre-optic laryngoscope was planned.

The patient was again induced but this time with Midazolam 7.5mg intravenously after spraying the nostrils and the pharynx with 10% lignocaine. Apart from the ECG monitor and Dinamap for recording blood pressure, oxygen saturation with pulse oximeter was also monitored. A fibre-optic laryngoscope was passed through the nose but attempts at the intubation

Fig 1 - X-ray of neck with arrows showing calcified stylohyoid ligaments and fish bone, within prevertebral soft tissue at C4,5



failed because there was already much bleeding in the area due to trauma. This was again abandoned and the patient was given additional Nitrous oxide/oxygen and halothane to facilitate another attempt at a blind nasal intubation. This was successful and the surgeon, proceeded with the oesophagoscopy.

## DISCUSSION

Sharwood-Smith<sup>(2)</sup> described calcified stylohyoid ligament as an unexpected cause of difficult intubation and also described the presence of a recognisable skin crease over the hyoid bone as the warning sign. Akinyemi and Elegbe<sup>(3)</sup> however could not find any recognisable skin crease in the two cases they described. In the case described no skin crease was observed on the neck over the hyoid bone. The use of stylet and the straight blade laryngoscope were not of much help in this case. Cormack and Lehane<sup>(4)</sup> classified causes of difficult intubation according to views obtained at laryngoscopy from grade 1 to

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Department of Anaesthesiology  
Faculty of Medicine  
University Kebangsaan Malaysia  
Jln Raja Muda Abdul Aziz  
50300, Kuala Lumpur  
Malaysia

A M Aris, MD(UKM), M Med (Anaesth)(UKM)  
Specialist

E O Elegbe, MBBS, DA, FFARCSI  
Consultant

R Krishna, MBBS, FFARCSI, FFARACS  
Consultant

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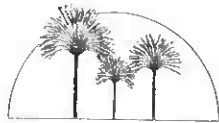
4. In their classification this case was grade 3, as no part of the glottis could be seen. This grade of difficulty should bring to the minds of anaesthetists a high probability of calcified stylohyoid ligament which is a common incidental finding<sup>(5)</sup>. Without causing much trauma the anaesthetist should embark on the use of the fibre-optic laryngoscope<sup>(6)</sup> to intubate the patient. A neck X-ray should be done to confirm the cause postoperatively or if a neck X-ray is available as it was in this case, calcification of stylohyoid ligament should be looked for immediately as the possible cause of the difficult intubation.

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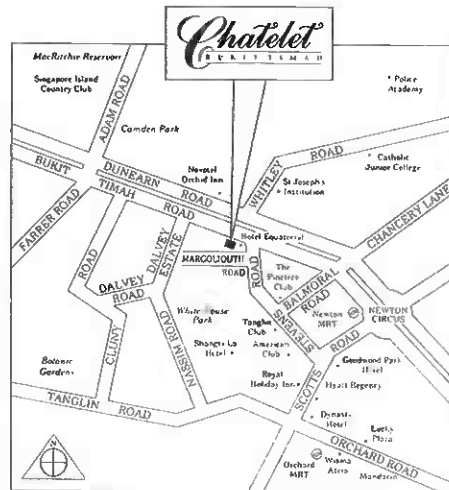
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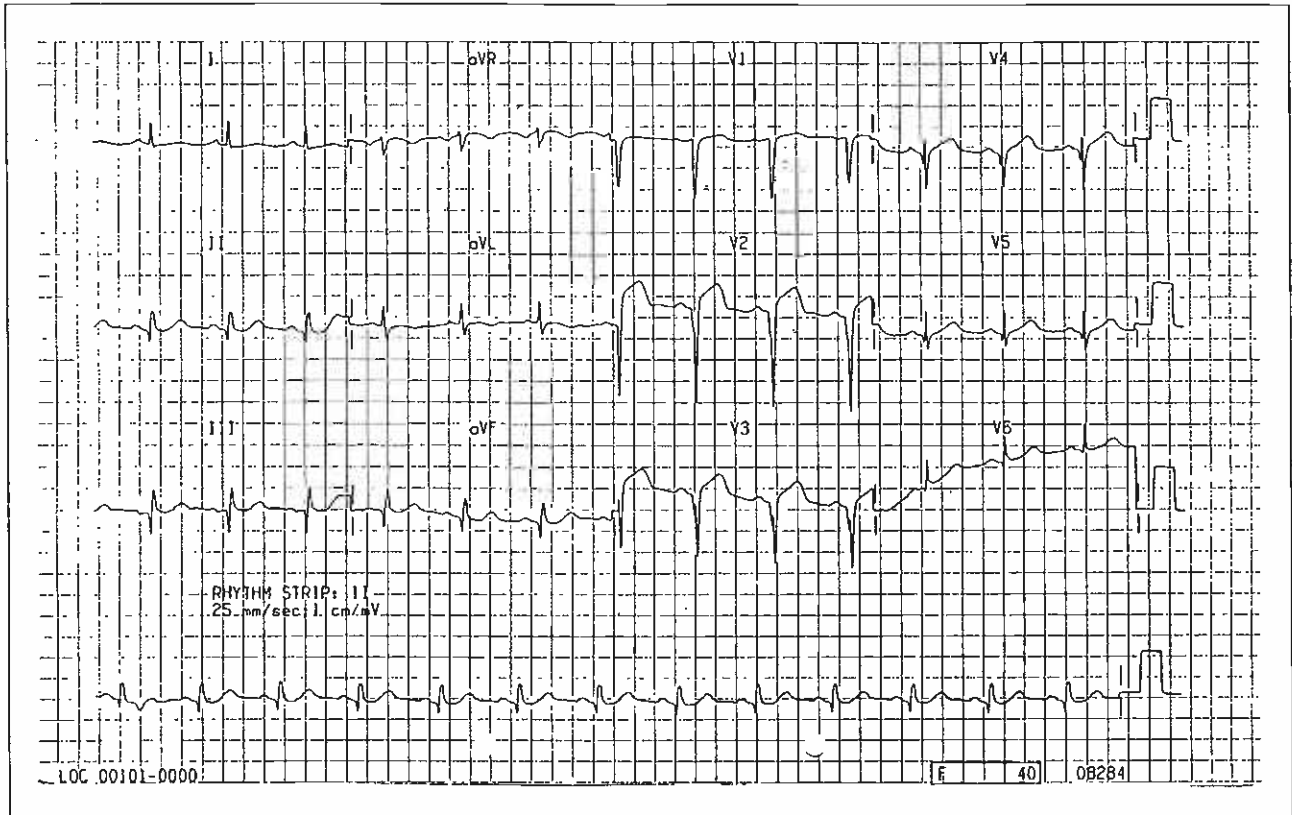
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Fig 2-12 electrocardiogram repeated 5 hours later when the patient had recurrence of chest pain



**Diagnosis:** Hyperacute phase of acute myocardial infarction.

**DISCUSSION:**

The 12 lead ECG (Fig 1) recorded initially was interpreted as being normal. Five hours later when the patient complained of chest pain again, an ECG was immediately repeated. This showed QS complexes, loss of R waves and ST elevation over the anteroseptal leads (Fig 2). The diagnosis of acute anteroseptal myocardial infarct was then made and the patient admitted to the Cardiac Care Unit.

In retrospect, the patient's initial complaints of central gripping chest pain associated with breathlessness, sweating, nausea and vomiting were very suggestive of an acute myocardial infarction. The diagnosis was dismissed because the ECG recorded at admission was interpreted as being normal (Fig 1). However on careful inspection of the ECG, one can notice that the T waves in the leads V2, V3, V4 are tall and asymmetrical. The ST segments in these leads cannot be identified with accuracy but one can see that the ST segment is taken up by the T wave, with a sharp angle between the beginning of the T wave and the end of the ST segment. These are features which can be seen in the hyperacute phase of a myocardial infarction<sup>(1)</sup>. The ECG repeated 5 hours later because of renewal of chest pain showed features of a more evolved acute anteroseptal infarct with the development of QS complexes and loss of R waves in V2, V3, and V4. Further on the T waves in those leads became less tall. The delay in the diagnosis resulted in the patient exceeding the time window for which thrombolytic therapy with intravenous streptokinase would be ideal.

The first few hours of a myocardial infarction is called the hyperacute phase. In this period the myocardium is acutely injured, but not yet necrosed. Therefore it is still able to conduct the electrical activation front, but at a slower rate. The electrocardiographic features of the hyperacute phase are as follows: increased ventricular activation time, increased amplitude of the R wave, slope elevation of the S-T segment and tall and widened T waves. The ventricular activation time, the time from the beginning of the QRS complex to the apex of

the R wave is delayed beyond 0.45 sec. Tall and widened T waves in the precordial leads, like in the patient above, may be the earliest manifestation of acute anteroseptal myocardial infarction and may appear before ST changes. It may occasionally be the dominant feature of the hyperacute phase of myocardial infarction. Tall widened T waves in the anteroseptal leads are however not specific for the hyperacute phase of an anteroseptal myocardial infarction. They can also be found in the following conditions: Prinzmetal's angina, hyperkalaemia, left ventricular diastolic overload, in healthy vagotonic individuals (the early repolarisation pattern), acute pericarditis, left bundle branch block, acute posterior wall subepicardial ischaemia and acute head injury<sup>(2)</sup>. It is beyond the scope of this discussion to specify the differential diagnosis of the above conditions on clinical and electrocardiographic grounds. However, if the history is suggestive and the ECG shows tall T waves, one must assume that the patient has an acute myocardial infarction until proven otherwise. The classical pattern of symmetrical or asymmetrical tall T waves in the hyperacute phase of a myocardial infarction usually lasts for only a few hours. It rarely persists for long.

In patients presenting with chest pain and later proven to have myocardial infarction the initial ECG is "diagnostic" in slightly more than half the patients. Serial ECG's can increase the sensitivity of this diagnostic tool up to 90%<sup>(3,4)</sup>. It is therefore important to repeat the ECG in patients suspected of having a myocardial infarction.

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