

A Case of Cryptococcoma Producing Tracheal Obstruction during Anaesthesia.

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SUMMARY

A case of cryptococcoma of the right upper lobe producing tracheal obstruction during anaesthesia is reported. Whilst conscious the patient breathed spontaneously with little difficulty. The reasons underlying complete tracheal obstruction during anaesthesia are discussed.

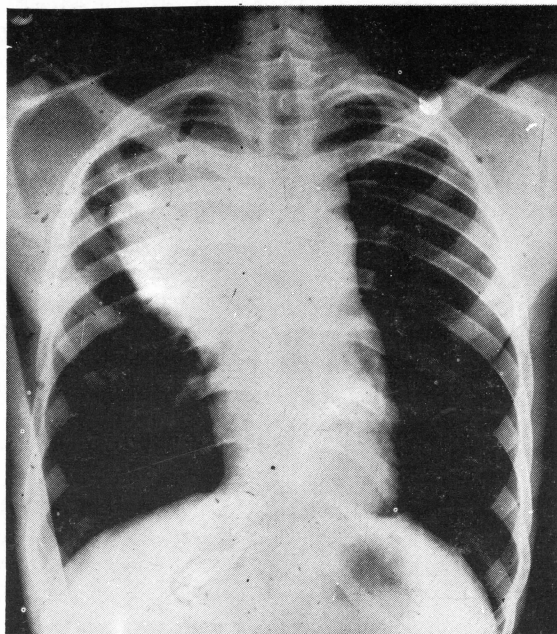
Case History

A 25 years old Zambian male was transferred to the University Teaching Hospital, Lusaka for further investigations and management of a rapidly growing mass in the right upper lung from another hospital. He first presented at another hospital in April 1976, complaining of an irritating cough, right sided chest pain and progressive weight loss. He also mentioned a similar episode in the past which was diagnosed as pulmonary tuberculosis and his symptoms responded then to the anti-tuberculosis treatment. He presented again some months later with a history of haemoptysis. A chest X-ray showed some threefold increase in the size of the opacity. Bronchoscopy was attempted at that time, but the bronchoscope could not be negotiated beyond the carina as the patient developed "severe bronchospasm" and the procedure was abandoned. The patient was transferred to the University Teaching Hospital, Lusaka for investigations and treatment.

We found him to be a young man of average build with signs of weight loss. He was mildly dyspnoeic on lying flat. He was not anaemic and was not cyanosed. There was diminished air entry in the right upper zone. Chest X-ray showed a 9cm. round homogeneous opacity in the right upper zone occupying the Anterior Mediastinum (Figure). Tomography confirmed the presence of a solid mass in the antero-superior mediastinum with some tracheal displacement, but the tracheal lumen did not appear greatly diminished. It was thought that the mass was probably a benign tumour but in view of the rapid increase in the size of the mass, development of dyspnea on lying flat, exploratory thoracotomy was thought to be the quickest method of establishing the diagnosis and instituting treatment.

treatment.

FIGURE



The chest X-ray shows a well defined opacity involving the right upper lobe and encroaching the anterior mediastinum. The trachea is outlined and the lumen does not appear to be diminished.

Anaesthetic Induction

The patient was given Atropine 0.6mg followed by Thiopentone 400mg intravenously 50mg Suxamethonium was given for muscle relaxation to facilitate intubation. On attempting to inflate the lungs prior to intubation it was found that there was no air entry in either of the lungs. Intubation was performed with 9.0 Magill's endotracheal tube. Even with the tube in situ it was found extremely difficult to inflate the lungs. The position of the tube was checked and the cuff deflated. This failed to improve the situation and another tube was substituted, but even this failed to improve the situation. On auscultation there were

scattered rhonchial over the lung fields. The pulse was feeble and slow. It was thought that the patient had developed severe bronchospasm and Aminophylline 250mg. and Hydrocortisone 300mg. was given intravenously. There was no improvement in the condition of the patient and bronchoscopy was performed. There was complete flattening of mid-trachea. The bronchoscope was passed beyond this point with some difficulty. Once the bronchoscope was passed into the left bronchus it was easy to inflate the left lung. During this period the patient had become severely hypoxic, and the patient had a hypoxic cardiac arrest. All attempts to resuscitate the heart failed.

On post mortem examination, the trachea was ribbon shaped and completely flattened anteriorly, from about the region of 2.5 to 3.5cm. above the carina. A large mass arising from the lung tissue of the right apical lobe was compressing the trachea at carina, and also compressing the right and left bronchus. The trachea was fragile and the tracheal rings easily fell apart. The pericardium was thickened. The histology of the tumour showed it to be a cryptococcoma (described in detail elsewhere).

DISCUSSION

That the patient died of hypoxia is beyond question. What is intriguing is that the patient was breathing spontaneously with little difficulty when awake yet it was impossible to inflate the lungs after he was anaesthetised and paralysed. The endotracheal tube has been found to be responsible for obstruction in some cases but it was clearly not the case in this patient. The post-mortem showed the obstruction to be at the level of the carina, the main bronchi and at the 2.5 to 3.5cm. portion of the trachea above the carina.

The explanation of this may be found in the mechanics of respiration. During spontaneous respiration the diameter of the small airways as well as the bronchii is increased because of the pull of the expanding lung tissue. During inspiration the tumour mass may have been pulled off the carina and displaced laterally. The resistance to the flow of air through any tube is inversely proportional to the fourth power of the radius of the tube. Thus any small increase in the radius of the tube can decrease the resistance significantly. This may have facilitated the inspiration in our patient. However we would expect the expiration to be forced and prolonged.

The mechanism of breathing in a paralysed patient is different. The inspiration takes place under positive pressure. The weight of the tumour and the flattened trachea may require very high pressures to overcome the resistance. The flattened trachea in this case is analogous to the flattened neck of a balloon. It becomes very difficult to overcome the resistance and

inflate the balloon with a flat neck; the surface tension being very high in this case. Once the walls are pulled apart it becomes very easy to inflate it. Thus, in our patient the weight of the tumour and the flattened trachea may explain the difficulty in inflating the lungs. That the obstruction was at the tracheal and the main bronchial level and not at the small airway level is obvious as the lungs could easily be inflated once the obstruction was by-passed with a bronchoscope.

The use of Aminophyllin and Calcium Chloride in presence of severe hypoxia may have contributed to the cardiac arrest. The dangers of intravenous Aminophylline have been pointed out in the Editorial of this Journal (October-November 1976). Clarke (1970) states that in variable extrathoracic airway obstruction, maximal inspiratory flow rates are more reduced than expiratory flow rates. Flow volume curves or the measurement of the Forced Expiratory Volume in 1 second to Forced Inspiratory Volume in 1 second ratio is useful in diagnosis of the extent of the airway obstruction and also helps differentiate between the intrathoracic and extrathoracic airway obstruction. Whenever localised obstruction is suspected full investigation is needed including pulmonary function tests, and flow volume curves to assess the extent of the upper respiratory airways obstruction.

REFERENCES

1. Clarke T.J.H. (1970) *Inspiratory Obstruction. British Medical Journal Vol. 3 Pages 682-684.*
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