LOCALIZED OBSTRUCTIVE PULMONARY EMPHYSEMA DURING DIVING TRAINING (A Case Report)

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ABSTRACT

A case of localized obstructive emphysema in a 23-year-old Diving trainee is reported. Clinical presentation and radiological features were the key to the diagnosis. The mechanism of development of this condition during diving due to pressure changes, along with its implications are discussed.

Introduction

Localized obstructive emphysema occurs secondary to partial airway obstruction. This partial obstruction acts as a one-way valve leading to localized air entrapment. When ascending from a depth during diving, there is an expansion of gases in the body cavities. Expansion of gases trapped distal to the airway obstruction can cause overdistension of lung tissue leading to rupture and escape of air which can create serious complications.

A case of localized obstructive emphysema in a diving trainee is reported. This case presented with features of localized emphysema. X-ray chest helped in establishing the diagnosis and timely conservative management ensured uneventful recovery.

CASE REPORT

A 23-year-old sailor was diving at a depth of 2-3 mtrs. On coming to the surface of water he had a dry cough which subsided after a few minutes and he resumed diving at the same depth. However he was unable to continue and on surfacing the cough was markedly exaggerated and was associated with hyperpnoea.

During examination the sailor was found to have a severe, uncontrollable dry cough with hyperpnoea. There was however no history suggestive of haemoptysis, hoarseness or change in voice, retrosternal discomfort or severe pleuritic pain.

Clinical examination revealed pulse rate of 100/min regular, BP-140/90 Torr and Resp Rate-28/min. There was no cyanosis. Examination of the respiratory system revealed no mediastinal shift. There were reduced respiratory excursions in the left inframammary and infrascapular regions. Vocal fremitus and vocal resonance was reduced and there was reduced air entry with occasional rhonchi in the left inframammary and infrascapular regions. No crepitations were discernible. Other areas in the chest were normal. X-ray chest (Fig.1) revealed reduced vascular marking in the left lower zone with hyp-

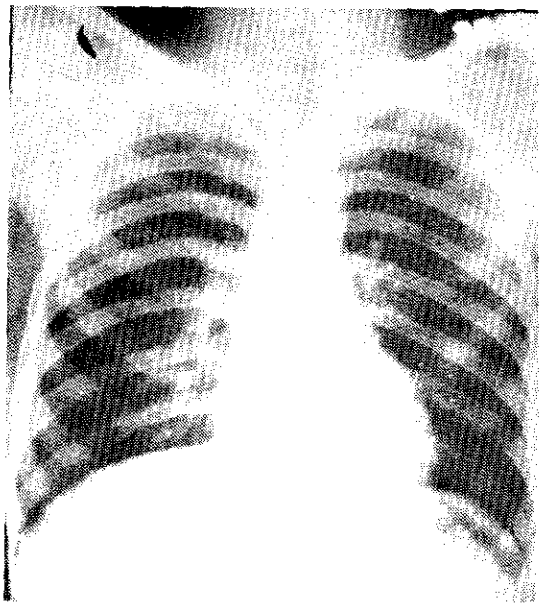

Fig. 1. X-ray chest showing reduced vascular marking and hyper translucency in the left lower zone.

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er-translucency. There was no evidence of air in the pleural or mediastinal cavity. X-ray of the neck confirmed absence of air in the neck tissue. Routine haematological investigations were within normal limits.

Patient made good recovery with antibiotics, antihistaminics ad mucolytic agents. Serial x-ray showed regression of the emphysematous areas and normalcy was restored in 10 days.

Discussion

Localized obstructive pulmonary emphysema is encountered in medical practice, where it may be secondary to a foreign body aspiration or to primary pulmonary tuberculosis[1].

Two features are significant when this occurs during diving. Firstly, during diving breathing is carried out with the help of a diving set. Inspiration requires a positive act of suction through a mouth piece. This suction causes a negative pressure in the breathing mask and a demand valve is opened which lets in air into the mask and the lungs. During expiration this demand valve remains closed and expired gas escapes through a separate expiration valve[2]. In addition to resistance to breathing, there is also an increased dead space and increased density of gases[3]. All these lead to an increased work of breathing and a reduced maximum breathing capacity[3,4].

Secondly, as the diver descends he continues to breathe equal volumes of air which are supplied at higher pressure so as to equalise the pressure within the thoracic cavity to that of outside water pressure[3,3]. When he ascends to the surface this air at high pressure expands in volume with the reduction in pressure in accordance with Boyles law for gases[6]. These expanding gases normally escape from an open glottis, which divers are taught to keep open[2,3]. However, if there is entrapment of air locally it can lead to alveolar overdistension (emphysema) and rupture (pulmonary Barotrauma)[7,8]. The pressure change necessary to cause pulmonary barotrauma is approx. 70 to 100 mm of Hg[2,3], i.e. an ascent from a depth of just over 1 metre to the surface. Alveolar air which has escaped into lung tissue secondary to pulmonary barotrauma can follow several pathways with serious implications.

(a) It can enter the pulmonary veins and thence into the systemic circulation as air embolism causing vascular obstruction and infarction.
(b) It can enter the pleural cavity causing pneumothorax.
(c) It can track along the loose tissue planes into the mediastinum and neck causing mediastinal emphysema, cervical emphysema, pneumopericardium and even pneumoperitoneum[3,5,7]. Treatment for pulmonary barotrauma by recompression may be indicated[3]. However, there is no role of recompression in pulmonary emphysema[9].

Airway obstruction can be caused in many ways[1]. These include

(a) mucus plug in the bronchial lumen,
(b) thickening of bronchial mucus membrane,
(c) increase in muscle tone,
(d) distortion of peripheral airways due to lung destruction, fibrosis and emphysema,
(e) invagination of the soft posterior wall of large bronchi and trachea on expiration due pressure gradient between extrabronchial and intra-luminal to pressure. Questioning in this case brought out a history of incompletely treated respiratory tract infection. It is postulated that (a) and (e) caused airway obstruction in this case.

In the first instance when the diver ascended to the surface, coughing reduced the intra-luminal pressure and there was relief. When he continued diving again, there was further air entrapment due to the suction during inspiration, valve-like action of the mucus plug and a recurrence of symptoms. On coming to the surface there was further expansion of the entraped air thereby causing localized emphysema and worsening of his condition

when he reported for medical assistance (Fig. 2).

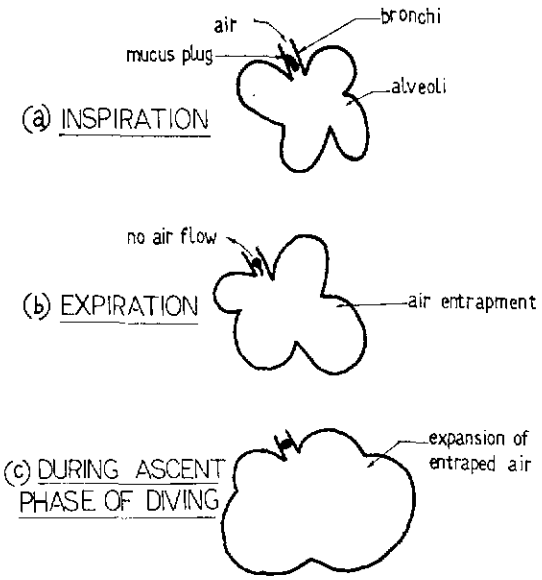

Fig. 2. (a) and (b) Entrapment of air during expiration due to one way valve action of mucus plug. (c) Expansion of entraped air during ascent phase of diving.

Shallow depths of 2-3 metres are commonly encountered in swimming pools. This case exemplifies the fact that such an accident can also occur in non-divers during swimming or diving, if they have a predisposing condition.

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