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Loud music exposure: etiology for Spontaneous Pneumomediastinum

Article type Case Report

Author(s) Saravana V Karuppiah and Amanjot Dhaliwal

Journal name and URL Clinical Medicine: Case Report
Accessible at <http://la.press.com>

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Title page

Loud music exposure: etiology for Spontaneous Pneumomediastinum

Karuppiah, Saravana V (MRCS)¹; Dhaliwal, Amanjot (MRCS)¹

Running title: Loud music exposure: etiology for Spontaneous
Pneumomediastinum

Affiliation:

1. Aberdeen Royal Infirmary Hospital

Grampian University Hospital NHS Trust

Foresterhill Road

Aberdeen AB25 2ZN

Scotland, UK

Corresponding Author:

Mr Saravana V Karuppiah

128, Cornhill Terrace, Aberdeen, AB16 5EW, Scotland, UK. Tel: +44 1224 482877 Fax: +44 1224 482877

Email: saravanavail@yahoo.com

Abstract

Spontaneous pneumomediastinum (SPM) is a benign condition most commonly occurring in male patients with no predisposing factors. Although there has been reports published suggesting various causes of spontaneous pneumomediastinum, none have so far shown loud music as a possible reason. We report a case of SPM in an 18 year old previously healthy male patient after exposure to continuous loud music. With extensive investigation including chest x-ray, gastrograffin swallow and contrast chest CT, there was no other cause found for his pneumomediastinum. To our knowledge there has been no previous report on a similar case.

Keywords: spontaneous pneumomediastinum, pnemothorax, sound, loud music

Introduction

Spontaneous pneumomediastinum (SPM) is an infrequent, benign, and self-limiting condition that predominantly affects young males. The incidence of SPM is around 1 in 7000-12000 of hospital admissions (Gerazounis 2003) but emergency admissions is around 1 in 30000-45000 (Newcomb).

. The precise cause for SPM is unknown; however various precipitating factors have been reported. This is the first report of primary spontaneous pneumomediastinum occurring after exposure to very loud music, to our knowledge there has been no similar previous reports in literature.

Case Report

An 18-year-old previously healthy male presented to the Accident and Emergency Department with 12 hour history of sudden onset retrosternal chest pain and difficulty in taking deep inspiration. Prior to the symptoms he had been drinking in a bar seated in close proximity to a speaker playing heavy metal music. He was in the pub for about 2 hours after which there was sudden onset of retrosternal chest pain. There was no history of chest trauma, fever, cough, haemoptysis, violent sneezing, vomiting or rhinolalia. He was a non-smoker and there was no significant past medical history or family history suggestive of lung or hyaline membrane diseases.

On examination he was mildly tachypnoeic, respiratory rate of 20 per minute, vitals otherwise stable. There was evidence of subcutaneous emphysema at the root of his neck. On auscultation, air entry was equal bilaterally with normal vesicular breath sounds. Further examination of his cardiovascular system revealed dull heart sounds with a precordial systolic crunching sound (Hamman's sign).

Plain chest x-ray showed the medial border of the left lung very clearly demarcated from the cardiac and great vessel silhouettes, suggestive of free air in the mediastinum, and evidence of free air in the soft tissues around the root of the neck. Subcutaneous emphysema of thorax is also seen (Figure 1). There was no evidence of pneumothorax in the chest x-ray.

He was admitted in the ward and managed conservatively with high flow oxygen via a face mask for two days. During the course of his treatment he was extensively investigated to determine the cause of his spontaneous pneumomediastinum. He underwent a water soluble contrast swallow (Gastrograffin swallow) which did not reveal any evidence of esophageal tear. A contrast computerized tomography scan (CT scan) of his chest confirmed free air in the mediastinum but the lung parenchyma, bronchial tree and esophagus were otherwise normal.

Patient's chest pain had settled within 48 hours of his admission and the emphysema had completely resolved. Repeat follow up chest x-ray revealed complete resorption of free air from the mediastinum. He was discharged with advice to avoid possible barotraumas such as playing wind instruments and sea diving for at least 6 months.

Discussion:

Spontaneous pneumomediastinum (SPM) is an infrequent, benign and self-limiting condition that predominantly affects young males (Jougon 2003). The precise cause of SPM is unknown; however, there have been several reports of SPM associated with violent sneezing (Decambre 1995), blowing (Marevelli 2000), labor (Sutherland 2002), coughing (Roe 1967) and inhalation drug use. Predisposing anatomic factors such as elastic tissue fragilities seen in thin tall young individuals, smokers and asthmatics (emphysema-like changes) may increase the risk of SPM, as reported in some cases of spontaneous pneumothorax (Abolnik 1991).

Extensive investigation in this patient with Chest x-ray, chest CT scan and oral contrast studies did not show any evidence of tear in the bronchial tree or esophagus. SPM is most likely caused secondary to rupture of alveoli adjacent to major vascular structures, with the free air then tracking along peribronchial and perivascular tissue planes toward the hilum of the lung and into the mediastinum (Kaneki 2000). We propose loud music as a precipitating factor for alveolar rupture in this patient causing SPM.

Sound is a form of mechanical energy characterized by a wave front propagating through a physical medium. Propagation of sound pressure waves through the respiratory system hence results in pressure differences as the interface media is made of different densities (i.e. air, alveolar surface water, lung tissue, bronchial tissue). The

pressure difference could tear gas containing organs such as alveolar wall, bronchial tissue resulting in air leak into the mediastinum.

Primary blast injury to gas containing organs such as ears or lungs can occur if the mechanical energy of sound is very high, as in blast or explosions. Very high intensity (loud) sound, such as music in an enclosed bar, at close range could be considered as repetitive minute blasts, causing alveolar injury. The compressive and decompressive forces repeated at short intervals forcing 'trapped air' against delicate compartments such as the alveolar septa, can cause them to rupture (Zhang 1996).

Frequency band of the acoustic pressure wave spectrum have been shown as a potential mechanism for alveoli wall damage in experimental studies. Low frequency (<500) band of the acoustic pressure wave spectrum in combination with high intensity (>90dB) sound, as those used in bar and clubs, lead to structural and functional lung changes in animal models and humans too (Grande 1999). The frequency of commercial loud speakers are typically in the range of 30Hz to 20kHz and it is the lower frequency band of 30-150Hz which is usually boosted in music venues for enhanced sound effect.

The combination of these mechanisms can lead to excessive intra-alveolar pressures differences which when not equalized, secondary to mucus retention or inflammation in the bronchioles, may lead to rupture of alveoli, with subsequent leak of air into the mediastinum causing SPM or pneumothorax. Air from the mediastinum may then dissect superiorly into the visceral, retropharyngeal and subcutaneous spaces of the neck. From the neck, the subcutaneous compartment is continuous throughout the body; thus, air can diffuse widely, as seen in this patient.

The occurrence of SPM in this patient could have been coincidental. Certain professionals such as rock musicians and construction workers are more prone to continuous exposure to high intensity sound however SPM have not been reported in these individuals. A pre existing fragility of the lung tissue in combination with a precipitating factor such as loud music may have resulted in SPM in this patient.

Noppen et al (Noppen 2004) had reported a series of case reports of patients developing spontaneous pneumothorax, in previous healthy patients, after exposure to loud music. Most of the patients were smokers with some patients having radiological evidence of abnormal lung parenchyma. Loud music probably was precipitating factor acting on

an existing weaken lung tissue causing alveoli rupture leading to pneumothorax. Based on previous experimental results (Zhang 1996 and Grande 1999) and history the authors believe that loud music is the probable cause for SPM in this patient. We hope that further case reports and experiments will help shed more light into this subject in the future.

SPM is however a benign condition and the air in the mediastinum are absorbed spontaneously within two to four days. Therefore a conservative management with bed rest and analgesics is the choice of treatment in most cases. Treatment with 100percent oxygen is known to facilitate absorption of mediastinal air in a similar way as seen in spontaneous pneumothorax and is therefore advisable. Decompression of mediastinal air is recommended in cases where vitals functions are compromised; however we did not require such a treatment for our patient as his vital signs were stable.

Conclusion:

Etiology of spontaneous pneumomediastinum by and large remains uncertain. However, male patients with sudden onset of chest pain should be investigated for a possible SPM and treated accordingly. Continuous exposure to loud music should be considered as a precipitating factor in patients with SPM.

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Figure Legend:

Figure 1 – AP chest xray showing free air in the mediastinum

