

Pneumothorax secondary to septic pulmonary emboli in tricuspid endocarditis

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Abstract

Two cases of pneumothorax secondary to pulmonary septic infarctions occurred in the course of tricuspid endocarditis in intravenous drug misusers. This unusual complication must be considered in patients with right sided endocarditis who develop pleuritic chest pain, haemoptysis, or breathlessness.

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The tricuspid valve is affected in 5-10% of cases of infectious endocarditis, and in these cases intravenous drug abuse is the most important cause.^{1,2} The most frequent complication of right sided endocarditis is pulmonary infarction due to septic emboli; it was present in 60-100% of all cases of tricuspid endocarditis.¹⁻³ To our knowledge, only one case of pneumothorax secondary to septic pulmonary embolisation in the course of tricuspid endocarditis has been reported.⁴ We report two new cases of pneumothorax and tricuspid endocarditis in intravenous drug misusers.

Case reports

PATIENT 1

A 23 year old man, who had been dependent on intravenous heroin for the past two years, was admitted to the hospital because of three days of high fever, chills, arthralgia, and blood stained purulent sputum. His temperature was 39°C with a respiratory rate of 30/min, a heart rate of 140/min, and decreased breath sounds at both lung bases. Red and white blood cell counts were normal, except that there was lymphopenia ($0.6 \times 10^9/l$). The results of serological tests were positive for both human immunodeficiency virus antibodies and hepatitis B surface antigen. The chest radiograph was normal. Blood cultures yielded *Staphylococcus aureus* sensitive to methicillin. Intravenous treatment with cloxacillin (8 g/day) and gentamicin (240 mg/day) was initiated. Doppler echocardiography showed two vegetations on the tricuspid valve and moderate incompetence of this valve. After the fifth hospital day a chest radiograph showed several small bilateral patchy infiltrates. Owing to the persistence of high grade fever and progressive clinical impairment cloxacillin was replaced by vancomycin (2 g/day).

On the 15th hospital day the patient became breathless and his chest radiograph showed bilateral hydropneumothoraces (fig 1). Inter-costal drains were inserted into both pleural cavities. Purulent fluid was drained, from which *S aureus* sensitive to methicillin was cultured. The clinical condition of the patient improved slowly. Parenteral antibiotic treatment was maintained for eight weeks. Further chest radiographs showed re-expansion of both lungs with bilateral basal pleural thickening.

PATIENT 2

A 26 year old man, dependent on heroin for the past six years, came to the hospital with high fever and right pleuritic chest pain. He appeared severely ill; his temperature was 40°C with a respiratory rate of 30/min and a heart rate of 120/min. The breath sounds were decreased at the right lung base. The red blood cell count showed microcytic hypochromic anaemia (haemoglobin 10.5 g/dl, mean corpuscular volume 79 fl, mean corpuscular haemoglobin concentration 28 g/dl). The white blood cell count was normal. The results of serological tests were positive for both human immunodeficiency virus and hepatitis B virus (anti-HBc). Doppler echocardiography showed discrete lateral tricuspid valve thickening. Chest radiography showed a small infiltrate in the right lower lobe. After some hours a right hydropneumothorax developed (fig 2) and the patient's general condition deteriorated, with dyspnoea and cyanosis. An intercostal tube was inserted and purulent fluid drained. *Staphylococcus epidermidis* was isolated from this fluid and also from several blood cultures taken at the same time. The patient was given vancomycin (3 g/day) and gentamicin (240 mg/day) for four weeks. Further chest radiographs showed good re-expansion with residual right basal pleural thickening.

Discussion

Pulmonary lesions occur commonly (in 58% of cases) in intravenous drug misusers, and pleural lesions have occasionally been described (in 6% of cases).³ Pneumothorax has developed in drug misusers who inject into the jugular or subclavian vein^{5,6}; bilateral pneumothorax also has been described occasionally in this setting⁷ and is attributed to

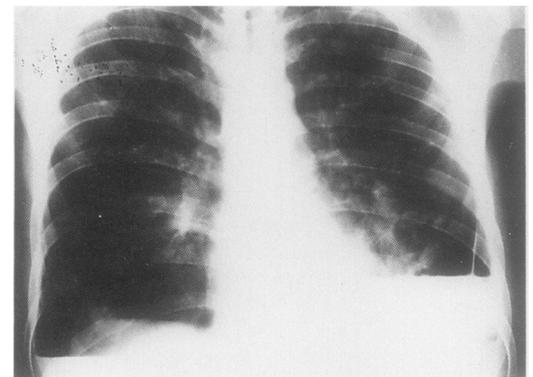


Figure 1 Patient 1: Posteroanterior radiograph of chest showing bilateral hydropneumothorax.

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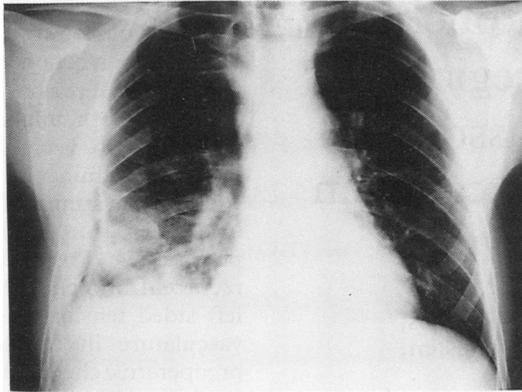
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Figure 2 Patient 2: Posteroanterior radiograph of chest showing right hydropneumothorax.



pleural trauma by needles inserted into central veins. Central venous injection was not attempted by either of our patients.

Several pulmonary diseases described in intravenous drug misusers may produce a secondary pneumothorax, notably miliary tuberculosis and pneumonia.^{8,9} In the same way a spontaneous pneumothorax without any pulmonary lesions has been reported in a patient not misusing drugs who had mitral valve endocarditis.¹⁰ Except for the case recently reported by Aguado *et al*,⁴ we know of no other reports of secondary pneumothorax from septic pulmonary emboli during the course of tricuspid endocarditis. The pneumothorax must be due to progression of septic pulmonary

infiltrates with subsequent leakage of air into the pleural cavity.

Consequently, in patients with active right sided endocarditis who present with pleuritic chest pain, dyspnoea, or haemoptysis a secondary pneumothorax must be suspected.

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BOOK NOTICES

Asthma in Practice. Mark Levy and Sean Hilton. (Pp116; £12.50.) London: Royal College of General Practitioners, 1992. ISBN 0 85084 165 8.

Asthma is an increasingly important problem for general practitioners. Yet all too often its diagnosis may be missed or made late. Furthermore, there is evidence that the quality of its management is highly variable. This excellent book by two leading general practitioners summarises very neatly the growing concerns about how general practitioners should care for their asthmatic patients. It is very well written and brief enough to be read in an evening. It covers the pathogenesis and diagnosis of asthma and reviews the evidence concerning the apparent increase in its prevalence. It discusses the management of asthma by age group and includes a special chapter on the role of the practice nurse and asthma clinics, including examples of protocols and so on. There is a separate discussion of health education in asthma and ways in which the practitioner can audit his performance and monitor the use of inhalation devices. Thus it is as much a practical manual for the doctor in his surgery as a textbook. There is also a brief glance at the future developments in asthma care, particularly how clinical performance can be assessed by means of quality of life measures. The General Practitioners in Asthma Group is discussed for those doctors who would like to make asthma a special interest. All in all this is a very good buy for any practice library. It is well referenced and indexed and its illustrations, although in monochrome, are apt and well chosen. If I have any criticisms they relate to the somewhat scant handling of the issue of allergy. Particularly I would have liked more

explanation on the importance of looking for allergens, such as pet danders, in patients who are relatively unresponsive to steroids. I would also have liked a more thorough account of occupational asthma. But any increase in the size of the book may have detracted from its readability, which is one of its major virtues.—Md'S

Manual of Clinical Problems in Pulmonary Medicine. 3rd edition. Edited by Richard A Bordwo and Kenneth M Moser. (Pp521; £16.95.) Edinburgh: Churchill Livingstone, 1991. ISBN 0 316 10272 5.

The preface to the first edition states that the book aims to provide the reader with a concise framework with which to approach a clinical problem and to direct him or her to the relevant publications comprising the basis for traditional diagnostic and therapeutic "wisdom." This third edition very successfully meets these original aims, maintaining the format and style of the previous editions. A great deal of information is packed into the 500 pages, organised into manageable and logically grouped sections. A minor criticism is the small print size, presumably selected to ensure "pocketbook size." A particularly attractive feature of the book is the key references, with a note about their findings, which accompany each topic. Inevitably there is a North American flavour, reflected in some of the pulmonary infections and in 56 annotated references on pulmonary complications of sickle cell disease. There are excellent contributions on pulmonary thromboembolism from Dr Moser, but by contrast the section on airways disease is somewhat disappointing with respect to the important issues of home nebuliser treatment and criteria for long term oxygen. Nevertheless, a third edition is a welcome replacement for well thumbed earlier editions, with the updated key references excellent value in themselves. For those unfamiliar with the book this is a clinical handbook that can be highly recommended to clinicians of all grades working in respiratory medicine.—SWB