South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 16 August 1958 Weekly 2s. 6d. Vol. 32 No. 33 Deel 32 Kaapstad, 16 Augustus 1958 Weekliks 2s. 6d.

SPONTANEOUS PNEUMOTHORAX IN ASIAN INFLUENZA

R. I. H. WELSH, M.B., B.CH. (RAND), F.R.C.S. (EDIN. AND ENG.), *Clinical Assistant, General Hospital, Pretoria*

The epidemic of Asian influenza was at its height in the Transvaal Province, South Africa, between July and August 1957. Two hundred nurses were admitted to the General Hospital, Pretoria, suffering from this condition. Two of them developed a spontaneous pneumothorax and are the cases reported below.

CASE REPORTS

Case 1

E.W.O., aged 17, was admitted to the Pretoria General Hospital on 17 August 1957 as a case of Asian influenza. She had complained of malaise, headache, feverishness and muscle pains. Her temperature was 100°F, pulse rate 104 per minute, and respiration rate 24 per minute. She was given Sulphatriad and penicillin.

On 18 August crepitations were heard at the bases of both lower lobes; temperature 101°F, pulse rate 128 per minute, respirations 32 per minute. Her temperature rose to 103°F that evening. Next day, widespread rhonchi were heard on auscultation of the lungs.

On 20 August she became critically ill. She was orthopnoeic, cyanotic, cold and clammy and surgical emphysema was present anteriorly in the neck. The sputum was frothy and stained pink. A portable X-ray plate (Fig. 1) showed bilateral patchy consolidation, atelectasis of the right lower lobe, and a pneumothorax on the same side. The presence of surgical emphysema was confirmed.

Her critical condition remained unchanged. Continuous oxygen was administered. In view of the large pneumothorax and her respiratory distress a catheter was introduced into the pleural cavity *via* the 2nd right intercostal space anteriorly and connected to an under-water drainage system. The pressure in the pleural cavity was positive. There was a slight improvement in her breathing after release of the tension; this improvement, however, was not maintained and she died on 20 August some 4 hours later.

Case 2

A.M.C.W., aged 23, was admitted to the hospital on 21 August 1957. One week before admission she had had influenza but had not taken it seriously. For 3 days before admission she had had a retrosternal pain and had developed a troublesome cough.

On examination she was obviously ill-looking and there was a tinge of cyanosis in her lips and finger-tips. The pulse rate was 120 per minute, respirations 28 per minute and the temperature 101°F. Crepitations were heard at both lung bases.

Aureomycin and a stimulant cough mixture were given.

By 23 August her general attitude was one of anxiety and fear, with a complaint of intense pleural pain. Cyanosis was more obvious and orthopnoea more pronounced, the respirations being 36-40 per minute. The temperature was 103°F and the pulse rate 140 per minute. Examination of the chest revealed dullness to percussion over both lung fields posteriorly, and on auscultation bronchial breathing was heard in these areas. A portable X-ray plate showed bilateral bronchopneumonia. Albamycin, penicillin and ACTH were given.

Next day she began to cough small quantities of thick, blood-stained, purulent sputum. Her temperature remained high over the next 3 days and pleural pain was intense. A pleural rub was detected on the left side.

On 27 August her temperature fell to 98°F, her pain lessened and her appetite returned. Sputum culture grew a coagulase-positive staphylococcus sensitive to chloromycetin, streptomycin, albamycin, sulphonamide, erythromycin and polymyxin. Albamycin and aureomycin were replaced by chloromycetin and

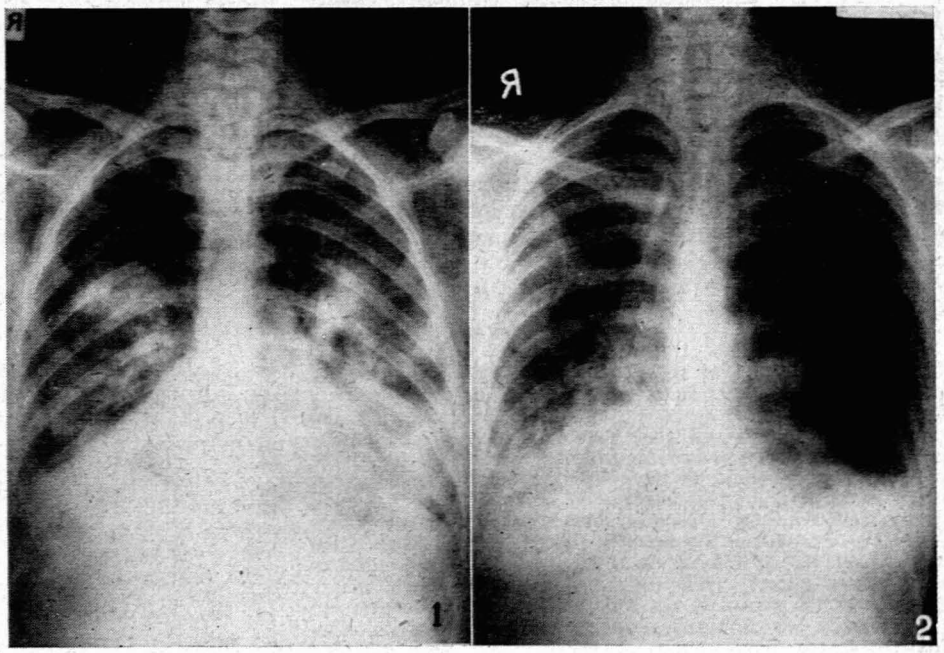

Fig. 1. Case 1. Bilateral patchy consolidation, atelectasis of the right lower lobe and right-sided pneumothorax. Surgical emphysema is present. *Fig.* 2. Case 2. Left-sided tension pneumothorax. There is an effusion visible in the left costo-phrenic sulcus. The pneumatoceles in the compressed left lung can be seen.

streptomycin. An X-ray plate showed numerous pneumatoceles in both lungs varying in size from 1 to 5 cm.

On 30 August a dramatic deterioration took place in her condition. She became even more short of breath, cyanosed again, cold, clammy, and extremely frightened. There was hyper-resonance over the left side of her chest with displacement of the trachea to the right. A portable X-ray plate confirmed the presence of a left-sided tension pneumothorax (Fig. 2). A catheter was introduced into the left pleural cavity *via* the 2nd intercostal space anteriorly and the tension was relieved by drainage of the air under water. The patient improved dramatically and a follow-up X-ray plate showed that the lung had re-expanded. The improvement, however, was short-lived, and she passed into a state of peripheral circulatory failure, dying on the following day (1 September).

DISCUSSION

Air finds its way into the perivascular tissue planes of the lung *via* ruptured alveoli, bronchioles or a bronchus and spreads towards the mediastinum. Its path may then follow the fascial plane of the great vessels into the neck and present clinically as surgical emphysema. A build-up of pressure in the mediastinum may, however, result in a rupture of the pleural covering with the development of pneumothorax.[1]

It is highly likely that this was the sequence of events in case 1. Air rarely leaks into the interstitial pulmonary tissues without predisposing causes. In this instance atelectasis of the right lower lobe, with over-inflation of adjoining areas, bronchospasm and cough, are sufficient factors to predispose the lung to this condition. A pneumatocele bursting into the interstitial lung tissue would have been a ready explanation for the sequence of events in this case. The radiological decision whether pneumatoceles had formed in the right lung was made difficult by the presence of surgical emphysema.

The spontaneous pneumothorax in case 2 was the result of rupture of a pneumatocele into the left pleural cavity. Pneumatoceles forming in areas of consolidation, and resultant spontaneous pneumothorax from their rupture, are characteristic radiographic findings of staphylococcal pneumonia.[2] Staphylococcal pneumonia is rare but seems to occur most commonly as a complication of influenza.[3]

In the 1957 epidemic of Asian influenza *Staphylococcus aureus* was the most dangerous secondary invader.[4,5] The sputum culture of a coagulase-positive staphylococcus and characteristic radiographic signs of pneumatoceles in this patient confirmed the diagnosis of secondary invasion. That the improvement after release of the tension within the pleural cavity was only temporary indicates the overwhelming nature of the combined infection.

Cases of influenza secondarily infected by the staphylococcus may die at the stage of acute tracheo-bronchitis.[5] Bronchopneumonic changes, however, may herald a train of events as here described and will help to indicate those cases of Asian influenza which are not following the expected benign course.

SUMMARY

1. Two cases of Asian influenza complicated by spontaneous pneumothorax are described.

2. The probable mechanism of the development of spontaneous pneumothorax in each case is discussed.

3. The danger in Asian influenza of the secondary invasion by coagulase-positive staphylococci is emphasized. Such invasion may alter the course of a relatively benign illness into a potentially fatal one.

OPSOMMING

1. Twee gevalle van Asiatiese griep, gekompliseer deur spontane pneumotoraks, word beskryf.

2. Die waarskynlike meganisme van die ontwikkeling van die spontane pneumotoraks in albei gevalle word bespreek.

3. Die gevaar by Asiatiese griep van die sekondêre inval deur koagulase positiewe Stafilokokke word beklemtoon. So 'n inval mag die verloop van 'n betreklik onskadelike siekte tot 'n potensiële dodelike een verander.

I should like to thank Dr. J. D. Verster, Deputy Superintendent of the Pretoria Hospital, under whose care these patients originally fell, for permission to publish, Dr. J. Hough for the use of his clinical notes in case 2, and Mr. Theo Marais, head of the Photographic Department of the University of Pretoria, for the photographs of the X-ray plates. Dr. J. C. van der Spuy, head of the Department of Thoracic Surgery, has greatly encouraged the writing of this paper and for his help I should like to express my special thanks.

REFERENCES

1. Macklin, C. C. (1939): Arch. Intern. Med., **64**, 913.
2. Beaven, D. W. and Burry, A. F. (1956): Lancet, **2**, 211.
3. Evans, A. D. and Evans, M. (1956): *Ibid.*, **1**, 773.
4. Editorial (1957): Brit. Med. J., **2**, 872.
5. Roberts, G. B. S. (1957): Lancet, **2**, 944.