

A CASE OF NEAR-DROWNING WITH PERSISTENT RADIOLOGICAL AND FUNCTIONAL CHANGES

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SYNOPSIS

Previous reports on near-drowning indicated that, in almost all cases, radiological and other abnormalities cleared up within 10 days postsubmersion. This report describes a case of near-drowning involving a sand quarry worker in which radiological and functional changes persisted for over 2½ years postsubmersion. The possible underlying mechanisms are discussed.

INTRODUCTION

Most reports on near-drowning cases emphasised on the clinico-pathological features (Rivers et al, 1970; Hasan et al, 1971; Modell et al, 1976 and Alkan et al, 1977). Few laid emphasis on the radiological sequelae (Rosenbaum et al, 1964).

In almost all cases, the radiological changes cleared up within 10 days. Only one case was reported to have persistent changes for 41 days postsubmersion.

- Our case of near-drowning has 2 interesting features:—
- Radiological and functional changes persisting for over 2½ years.
 - Its presentation as a "masquerader" of silicosis, a pneumoconiosis resulting from inhalation of free silica dust.

CASE REPORT

In November 1976, a 44 year old sand quarry worker was notified as a case of Silicosis to the Chief Inspector of Factories. (In Singapore silicosis is a notifiable industrial disease under The Factories Act 1973). The Chest X-ray picture was very suggestive of silicosis, classifiable as p 2/2 under the ILO U/C Classification (ILO, 1972). However there was no significant occupational exposure to silica hazard.

The patient had been farming for 19 years before he became a sandwasher in 1972. His work at the sand quarry involved operation of the water pump and attending to the discharge of wet sand from hoppers into lorries. (See Figures 1 — 3). Thus at the time of notification of silicosis, he had been working in the sand quarry in essentially wet processes for approximately four years. He had no history of exposure to silica dust, occupationally or otherwise. In view of the negative exposure history it was felt that the medical and other aspects of the case should be examined further for clues on the cause of the radiological findings.

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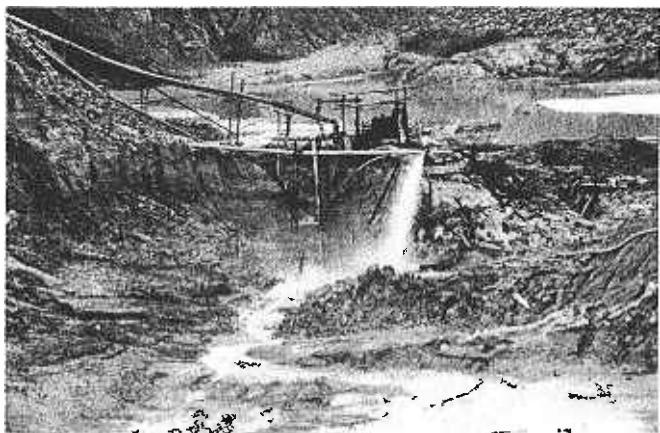


Fig. 1 View of sand quarry. Earth deposited by bulldozers are loosened by water jet and resultant sand-water mixture is pumped up to 'palongs' (sedimentation units).



Fig. 2 View of pump shed, 'palongs' (sedimentation units) and hoppers.

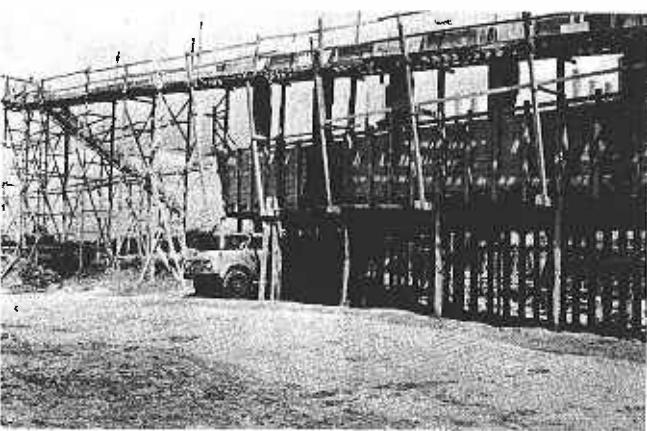


Fig. 3 Discharge of wet sand from hoppers into lorries.

It was learnt subsequently that the patient had in fact been involved in an accident at the quarry on 5 October 1976. He had slipped and fell four metres into the sand-pit. It is estimated that he was submerged unconscious in the sand-water mixture for almost a minute before he was rescued and brought to hospital. He regained consciousness on the way to the hospital (approximately 15 minutes

postsubmersion). He had a positive smoking history of over 20 years, smoking about 20 small sticks of Chinese tobacco per day. There was no other medical history of note.

On examination he was found to be conscious, rational, afebrile and not in respiratory distress (respiratory rate: 20 per minute). He had a seven cm laceration in the right parietal region. The blood pressure was 130/75, and pulse 92 per minute. There was good air entry at the time of admission but on the second day, auscultation revealed expiratory rhonchi bilaterally. Chest X-ray examination showed no rib fractures but there were bilateral fluffy nodular opacities throughout the lung fields.

Treatment comprised toilet and suture for the scalp laceration, oral bronchodilator and a course of ampicillin. He continued to cough out 'muddy' sputum for four days. He was discharged on the fourth day and referred to another hospital for further investigations regarding his lung lesions. He was found to be clinically well. He was not dyspnoeic at rest. Auscultation revealed good breath sounds with bilateral rhonchi.

The chest radiograph on the 5th day (Figure 4) showed no significant change from that taken on the 1st day. Lung function tests (13 days postsubmersion) were essentially normal except for a low resting diffusing capacity of 9.9 ml/min/mm Hg (predicted value 17.7 ml/min/mm Hg) and impairment of the maximum mid-expiratory flow rate to 2.1 L/sec (predicted value 3.64 L/sec). Arterial blood gas analysis was normal.

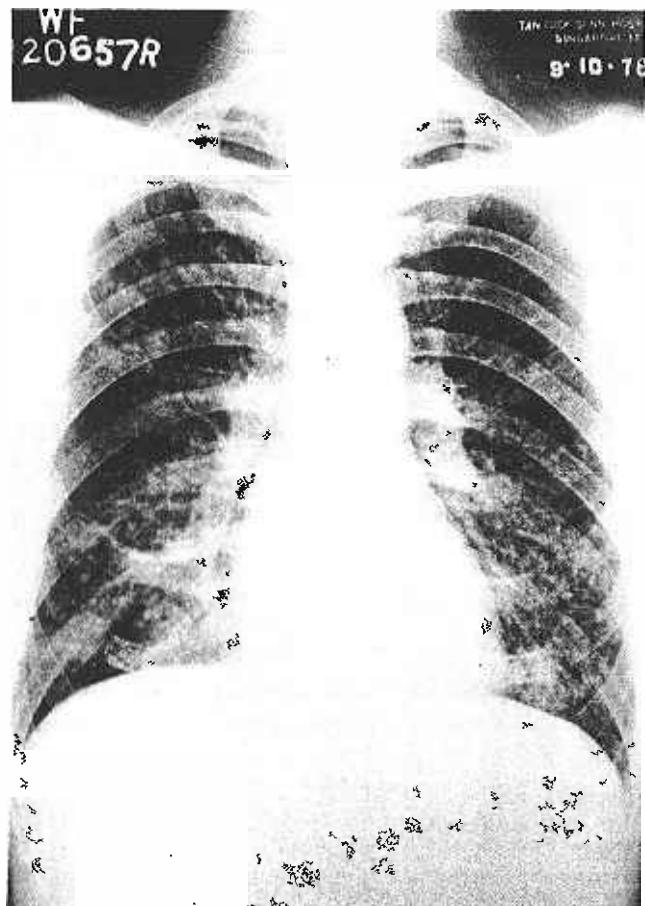


Fig. 4 Chest radiograph showing bilateral fluffy nodular opacities.

Prior to the accident, the patient had been well and free of respiratory symptoms. A routine chest radiograph taken in February 1976 was normal. (Figure 5).

He remained well on follow-up except for occasional cough productive of some whitish sputum. Sputum cultures were repeatedly negative for acid-fast bacilli. Serial chest radiographs showed the bilateral nodular opacities to be more discrete. (Figure 6). There was no significant change in lung function.

The results of analysis of the sand-water mixture taken from the site where the patient was submerged (Figure 7) are given in Table 1. Another sample taken from an area where the mixture was agitated by water jet was analysed in a similar manner. It is probable that the pH, electrolyte, silica and grease/oil contents of the sand-water mixture aspirated by the patient would have been within the range of values for these two samples, since there would have been some agitation of the mixture during the fall and submersion.

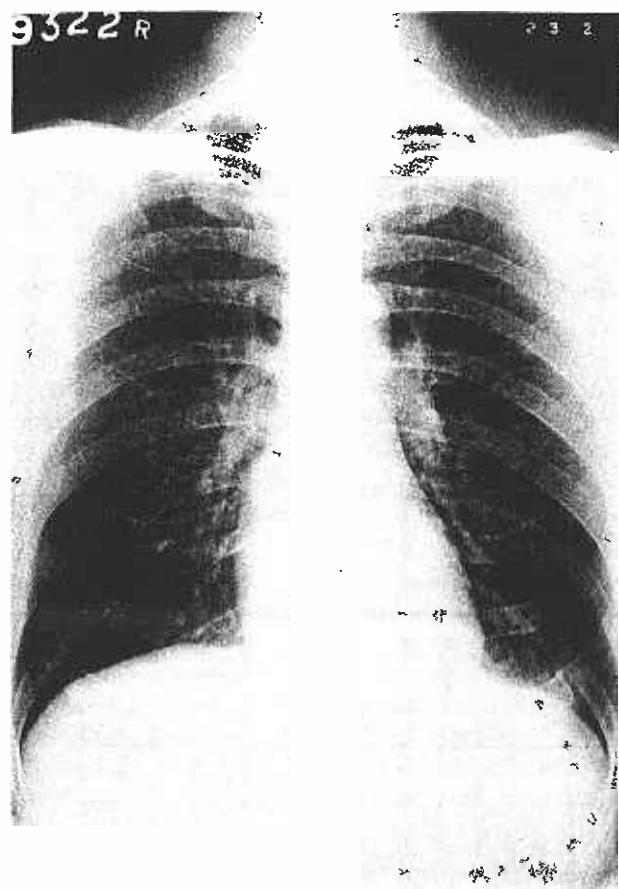


Fig. 5 Normal chest radiograph taken on 23 February 1976.

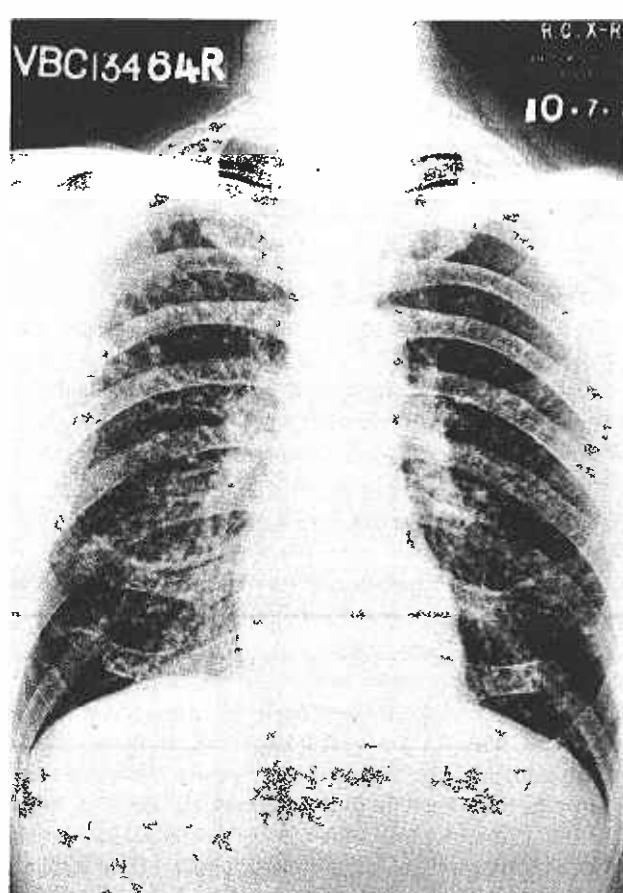


Fig. 6 Chest radiograph two years later showing persistent bilateral nodular opacities.



Fig. 7 Site of accident.

TABLE 1
RESULTS OF ANALYSIS OF SAND-WATER MIXTURES

| Sample | pH | Ca ⁺⁺ (gm/m ³) | Mg ⁺⁺ (gm/m ³) | Na ⁺ (gm/m ³) | Cl ⁻ (gm/m ³) | SO ₄ ⁻⁻ (gm/m ³) | HCO ₃ ⁻ (gm/m ³) | Free Silica (gm/m ³) | Grease/ oil content (mg/litre)* |
|-------------------------------------------|------|------------------------------------------|------------------------------------------|-----------------------------------------|-----------------------------------------|-------------------------------------------------------|-------------------------------------------------------|----------------------------------------|---------------------------------------|
| a) Accident site — Stagnant Sample: | 6.28 | 1.8 | 1.1 | 4.6 | 15 | 7 | <30 | ND | 10 |
| b) Agitated Sample: | 4.05 | 12.1 | 4.5 | 6.9 | 17 | 10 | ND | 2000 | 19 |

ND: Not Detected

*: (a) Corrected values using distilled water as blank
(b) Compare with Singapore Trade Effluent Standards:

| Type of Effluent | Std (mg/litre) |
|---------------------------|----------------|
| Severe | 60 |
| Uncontrolled water course | 10 |
| Controlled water course | 5 |

DISCUSSION

As far as we know, there have been no previous publications documenting cases of near-drowning with persistent lung lesions. Rosenbaum et al (1964), in their review of the radiological features in 10 cases of near-drowning, found that all cases but one cleared up by 8-10 days postsubmersion. The exception was a case of near-drowning in swamp water whose Chest X-ray apparently was "completely normal" 41 days postsubmersion.

Autopsy findings in cases of delayed death from drowning have been reported to range from various degrees of pneumonitis to bronchopneumonia and lung abscess. (Rosenbaum et al, 1964). Aspirated foreign particles, including silt, sand and diatoms, have been found in many cases. Microscopic evidence of inflammatory polymorphonuclear reaction is present quite early on, becoming more dominant as survival time increases.

The pathological changes are thought to be the result of hypoxia secondary to drowning, leading to desquamation of alveolar epithelium, increased capillary permeability and consequent exudation of plasma into the alveoli. Gaseous exchange across the alveolar membrane is thus interfered with. Hypoxaemia and metabolic acidosis are notably the most significant pathophysiological abnormalities in near-drowning victims (Hasan, 1971). Irritative bronchospasm may also be present (Rivers, 1970).

The abovementioned mechanisms could possibly account for the clinical and radiological findings in the initial acute phase in our patient. The chronic features (ie

persistent radiological picture and low diffusing capacity) could perhaps be attributed to some chronic inflammatory reaction to the presence of silica or silicates in the alveolar walls. While it is possible that the grease/oil in the aspirate may have contributed to the changes, we feel that the content is not significant enough to make it a major factor.

Unfortunately, as the patient was subjectively quite well, except for slight exertional dyspnoea, he was not agreeable to an open lung biopsy. Nevertheless, the two year follow-up post-submersion has revealed no significant changes in the clinical and radiological picture. It would certainly be interesting to study the natural history of this case to the end.

COMMENTS

The Chest X-ray is often the main tool in the diagnosis of silicosis. However a detailed occupational history is vital to exclude masqueraders (Van Ordstrand, 1977). This case well illustrates the point.

ACKNOWLEDGEMENT

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