Eur Respir J, 1994, 7, 1906–1908
DOI: 10.1183/09031936.94.07101906
Printed in UK - all rights reserved

European Respiratory Journal
ISSN 0903 - 1936

CASE REPORT

Trichomonas in pleural effusion

G. Radosavljevic-Asic, D. Jovanovic, D. Radovanovic, M. Tucakovic

Trichomonas in pleural effusion. G. Radosavljevic-Asic, D. Jovanovic, D. Radovanovic, M. Tucakovic. ©ERS Journals Ltd 1994.

ABSTRACT: Numerous living trichomonads were seen in a parapneumonic pleural effusion, in a patient who was at risk of aspiration pneumonia because of acute alcoholism. Of all the drugs administered, metronidazole had the most favourable therapeutic effect. However, decortication was necessary for the successful outcome.

Eur Respir J., 1994, 7, 1906–1908.

Institute for Lung Diseases and TB, University Clinical Center, Belgrade, Yugoslavia.

Correspondence: G. Radosavljevic-Asic Institute for Lung Diseases and TB, Bul. Revolucije 245, YU-11000 Belgrade, Yugoslavia

Keywords: Infection, respiratory tract, Trichomonas

Received: December 3 1993
Accepted after revision April 12 1994

The case of a patient with Trichomonas infection of the respiratory tract which was diagnosed in wet preparations of pleural fluid, is presented. As far as we know, this is the first such case reported in Serbia. Judging from the few reports in the literature, the finding of trichomonads in the human pulmonary tract is rare. According to HERSH [1], 65 cases were described up to 1985, mostly in patients with underlying pulmonary disease. It is a very unusual and unexpected opportunistic respiratory infection of unknown incidence and uncertain pathogenetic significance [1].

Case report

During divorce, accompanied by depression and heavy alcohol consumption, a 42 year old man developed coughing, expectoration of thick green sputum, haemoptysis and fever, in late September 1990. Fever persisted for several weeks. An initial chest X-ray, obtained in October 1990, was normal and the patient was treated with antibiotics, the presumptive diagnosis being recurrent bronchial infection. Sputum was bacteriologically sterile; Gram-stain was not performed. Therapy with penicillins, cephalosporins, aminoglycosides and sulphonamides caused symptoms to improve. However, complete cure was not achieved. In December 1990, chest X-ray revealed an opacity in the right lower lobe next to the spine. Bronchoscopic examination revealed inflammatory changes and foamy secretion in the bronchi of the superior segment of the right lower lobe. In January 1991, the patient had prolonged laryngitis. Chest X-ray findings deteriorated and he had daily fever of 37.5–40°C. Administration of quinolones did not improve symptoms.

In April 1991, the patient was hospitalized. On admission, he was pale and adynamic, showing poor oral hygiene, with carious teeth and gingivitis. He expectorated a foamy sputum. Vital signs were: temperature 38.5°C; pulse 100 beats·min^{-1}; respiration 18 breaths·min^{-1}; blood pressure 130/90 mmHg. Chest examination revealed decreased breath sounds on the right side. Laboratory values included: erythrocyte sedimentation rate of 125 mm·h^{-1}, haemoglobin of 13.2 g·dl^{-1}; white blood count of 21,600 cells·mm^{-3} (21.6×10^{9} cells·l^{-1}) (85% segmented); alkaline phosphatase of 249 U·l^{-1} (normal <240 U·l^{-1}). Blood urea nitrogen, glucose, bilirubin and total protein, were found to be within normal limits. Urine analysis showed a 1 + value for albumin. Chest X-ray revealed a shadow in the lower part of the right lung (fig. 1).

On the first pleural puncture, 50 ml of sterile serohaemorrhagic exudate was obtained containing 4,500

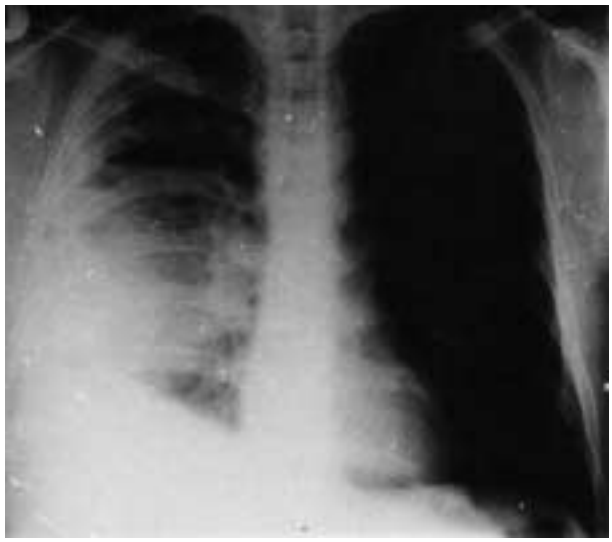

Fig. 1. – Posteroanterior chest radiography. The middle and lower parts of the right lung are obscured with two air-fluid levels near the upper border.

red blood cells·mm^{-3} and 2,000 white blood cells·mm^{-3}; protein of 53.9 g·dl^{-1}; glucose of 1.6 mmol·dl^{-1}; and lactic dehydrogenase of 3,228 U·dl^{-1}. Pleural biopsy revealed a nonspecific inflammation. A second pleural puncture was performed 6 days later and 200 ml of opaque foul-smelling exudate was obtained (12,000 white blood cells·mm^{-3}, glucose level zero), revealing a large number of actively mobile flagellates by direct microscopy of wet mounts. The organisms were identified as trichomonads based on their pyriform appearances, flagellatious and characteristic movements. Due to the unusual finding, the puncture was repeated on the following day, and in 400 ml of fetid, grossly purulent fluid the finding of numerous motile flagellates was confirmed. Bacteriological cultures of the empyema fluid yielded *Streptococcus* (*α-haemolysis*). Bronchoscopy was repeated on the same day and trichomonads were found in pleural empyema. Pus and mucus were present in the right bronchial tree. Bacterial cultures of the bronchial aspirate yielded *Klebsiella* and *Enterobacteriacae*. Urine and blood cultures revealed no pathogens. Urine was free of Trichomonas. To exclude a subphrenic process, liver scan and abdominal sonography were performed. The results of both examinations were negative. Metronidazole was administered at a dosage of 1,500 mg·day^{-1} for 10 days. General condition improved and the patient became afebrile. Pleural puncture was repeated and Trichomonas could no longer be identified. Species identification was not performed. Empyema drainage was performed, accompanied by intrapleural instillation of cephalosporins.

The patient seemed to be cured and was discharged in June 1991. However, he was readmitted two weeks later because of recurrent empyema. In the empyema fluid anaerobic bacilli (*Bacteroides distasonicus* 2) were isolated. Surgical decortication of the pleura was subsequently performed. Postoperatively, lung re-expansion was slow, fever recurred and metronidazole was again administered for 20 days (1,500 mg daily for 10 days intravenously, and 800 mg a day orally for 10 days), until the patient's body temperature had returned to normal values and all symptoms had disappeared. On follow-up examinations in 1991 and 1992, the patient was in good health.

Discussion

In patients with pulmonary diseases, trichomonads are most frequently identified in sputum [2], then bronchial aspirate [2–4], necrotic pulmonary tissue [2] and pleural effusion [5–8], by microscopic examination of fresh specimens and rarely by cultures [1, 2]. Authors of the published reports believe that wet mounts and direct microscopic examination are the best way to identify the organism by their rapid, wobbly and rolling motion [1, 7]. In fixated and stained material, organisms are not clearly recognized. Papanicolaou stain reveals the trichomonads as amorphous, pale, grey blobs, without structure. [6, 7].

Identification of flagellate species needs more accurate techniques, such as cultures and perhaps serological methods [1]. The problems encountered in identification of trichomonads in pulmonary specimens are similar in many reported cases. The discovery of flagellates takes the clinician by surprise, because the occurrence of these organisms is unexpected and has been reported so infrequently. Mobile trichomonads are usually only seen over a short period in one of two specimens per patient, and then never again. It is generally accepted that pulmonary trichomoniasis is caused by aspirated *Trichomonas tenax,* which is a harmless commensal of the human mouth, found particularly in patients with poor oral hygiene. Trichomonas survives in carious teeth and necrotic mucosal cells, where it feeds on micro-organisms in its environment. It has been detected in other structures and diseases connected with the oral cavity, including sinusitis, tonsilitis, jaw abscess, and cancer of the tongue and oesophagus [1].

According to Memik [5], the incidence of *T. tenax* being 0–25%, depends on oral hygiene and exposure. Its prevalence ranges 4–53%, [1]. Exceptionally, pulmonary trichomoniasis may be caused by an intra-abdominal *T. hominis,* or genitourinary *T. vaginalis* infection [9, 10]. *Trichomonas hominis* lives in the small intestine and in the colon and is spread by contamin-ated food or liquids. It was found in the case of enteropleural fistula, subphrenic abscess and empyema in the postgastrectomy patient [1]. *Trichomonas vaginalis,* the best known of these three species of Trichomonas parasites in humans, was isolated from the respiratory tract of infants and the tracheal aspirate of newborn babies with neonatal pneumonia by McLaren *et al.* [3] and Hiemestra *et al.* [4]. *T. vaginalis* was implicated as a respiratory pathogen in a report by Rebhum, whose patient gave a history of orogenital sexual practice [7].

The significance of Trichomonas in the respiratory tract of humans, remains unclear. Trichomonas cannot persist without associated bacterial infection. As illustrated in our case, pulmonary trichomoniasis was part of the mixed microbial flora of the empyema fluid in a patient with suspected aspiration pneumonia. We believe, as do many other authors, that the trichomonads were unable to cause the pulmonary disease on their own, but these numerous flagellates might have aggravated the poor general condition of the patient and might have prolonged the duration of the illness [2, 3, 5, 7, 8]. The large number of flagellates could not be considered as a benign finding. However, the aetiological aspects in the present case remain uncertain, however. Until the pathogenetic role of trichomonads in the human respiratory tract is resolved, metronidazole should be given. The true incidence of this parasite in the pulmonary tract might never be known. A careful look for the presence of Trichomonas in the respiratory tract would probably reveal a larger number of cases.

References

1. Hersh SM. Pulmonary trichomoniasis and *Trichomonas tenax. J Med Microbiol* 1985; 20: 1–10.

2. Walton B, Major M, Bacharacil T. Occurrence of trichomonads in the respiratory tract. Report of three cases. *J Parasitol* 1963; 49: 3–38.
3. McLaren LC, Daves LE, Haely GR. Isolation of *Trichomonas vaginalis* from the respiratory tract of infants with respiratory diseases. *Pediatrics* 1983; 71: 888–890.
4. Hiemestra I, Van Bel F, Berger HM. Can *Trichomonas vaginalis* cause pneumonia in newborn babies? *Br Med J* 1984; 289: 355–356.
5. Memik F. Trichomonads in pleural effusion. *J Am Med Assoc* 1968; 204: 211–212.
6. Walzer P, Rutherford I, East R. Empyema with Trichomonas species. *Am Rev Respir Dis* 1978; 118: 415–418.
7. Osborne TP, Giltman LI, Uthman EO. Trichomonads in the respiratory tract. A case report and literature review. *Acta Cytologica* 1983; 28: 136–138.
8. Ohkura T, Suzuki N, Hashiguchi Y. Invasion of the human respiratory tract by trichomonads. *Am J Trop Hyg* 1985; 34: 823.
9. Fardy PW, March S. Trichomonads in resected lung tissue. *Am Rev Respir Dis* 1969; 100: 893–894.
10. Polivoda NG, Demcuk ND, Krivonos ZP. A case of lung disease caused by *Trichomonas intestinalis*. *Vrach-Delo* 1987; 2: 33.