OCCASIONAL REVIEW

PULMONARY TRICHOMONIASIS AND *TRICHOMONAS TENAX*

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SUMMARY. Pulmonary trichomoniasis is usually caused by aspirated *Trichomonas tenax*. Adult men with chronic purulent or necrotic pulmonary disease are usually affected. Sixty-eight patients were previously described. A Russian study demonstrated pulmonary trichomoniasis in 19 of 112 patients (17%), mostly in patients with lung cancer, lung abscess, or bronchiectasis. Rarely, pulmonary trichomoniasis may be caused by an intra-abdominal (*T. hominis*) or genitourinary (*T. vaginalis*) infection. *T. tenax* is usually regarded as a harmless commensal of the human mouth. Its prevalence ranges from 4% to 53% and may exceed that of vaginal infection with *T. vaginalis* in adult females. It is frequently found in patients with poor oral hygiene. Cultural identification is superior to microscopic examination of wet-smear, gram-stained and Papanicolaou-stained preparations. Aspirated pulmonary trichomoniasis is an opportunistic infection. Until the question of possible pathogenicity is resolved, metronidazole should be given. The underlying pulmonary disease should be vigorously treated.

Introduction

Man is host to three distinct trichomonad species. The genitourinary *Trichomonas vaginalis*, the intestinal *T. hominis* and the oral *T. tenax* can be morphologically, serologically, epidemiologically and culturally distinguished from one another. Since 1867, sporadic case reports have appeared describing trichomonads in the respiratory tract. Before undertaking this review my attention was drawn to the condition by the case which is described in detail below.

Case report

A 59-year-old male alcoholic was admitted to Riverside General Hospital, Riverside, CA. He had been found comatose in a city park with fresh bruises about the face. He was partially edentulous with poor oral hygiene. He had congestive heart failure with uncontrolled atrial fibrillation. Skull X-rays were normal. Blood alcohol level was 400 mg%.

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Shortly after admission, he developed fever, *status epilepticus* and signs of impending cerebral tentorial herniation. A cerebral angiogram demonstrated a large subdural hematoma which was successfully evacuated. He was maintained thereafter on a mechanical respirator with periodic tracheostomy cuff deflation. Dexamethasone, methicillin, gentamicin, clindamycin, diphenylhydantoin, phenobarbital, digoxin and furosemide were continued. He remained comatose and intermittently febrile. Blood cultures and tracheal aspirates revealed no pathogens. On the thirteenth day in hospital, he developed respiratory distress and a left-sided pleural friction rub. Thoracentesis revealed foul-smelling, grossly purulent fluid. In addition to a mixed bacterial flora seen on a gram-stained smear, numerous motile flagellate trichomonads were seen in a fresh, wet, unstained preparation. Hypotension and bradycardia developed and the patient died.

At autopsy the left pleural cavity was coated with a yellow-grey exudate. In the left lower lobe there was an 8-mm thin-walled subpleural abscess which had apparently ruptured into the pleural cavity. In the litre of left pleural empyema fluid, numerous actively motile flagellate trichomonads were again easily seen in a fresh, wet, unstained preparation. No transdiaphragmatic abdominal communication was demonstrated. Trichomonads were not identified in histological sections. Trichomonads were identified on Papanicolaou-stained smears, but with much loss of detail (fig. 1). Attempts to culture trichomonads were unsuccessful. Bacteriological

FIG. 1.—Composite drawing of *T. tenax* from Papanicolaou-stained smear of pleural empyema fluid. Four anterior flagella, undulating membrane, nucleus and posterior protruding axostyle are seen. (Courtesy of Dr R. M. Stabler.)

TABLE I
Pulmonary trichomoniasis since 1942

Authors	Year	Country	Number of cases	Specimen in which <i>T. tenax</i> was demonstrated	Associated illness
Glaubach and Guller	1942	USA	1	Sputum	Pneumonia, pyorrhoea
Lehmann and Prendiville	1946	India	1	Sputum	Chronic bronchiectasis, malaria
Barbosa and Amaral	1950	Brazil	1	Sputum, gingiva, bronchial washings	Lung abscess, bronchiectasis, gingivitis
Kruscheva and Kryazheva	1951	Russia	1	Sputum, bronchial washings	Lung abscess, chronic bronchiectasis, meningitis, trichomonaemia
Tumka*	1956	Russia	19	Bronchial washings, resected lung tissue, sputum	Lung cancer, lung abscess, bronchiectasis, pneumonia, chronic bronchitis
Walton and Bacharach	1963	USA	3	Bronchial washings, sputum Pap-smear	Bronchogenic carcinoma, pulmonary fibrosis
Rebhun	1964	USA	1	Sputum	Chronic bronchitis, inactive tuberculosis, allergic rhinitis
Abed <i>et al.</i>	1966	France	1	Empyema fluid	Empyema, bronchopleural fistula
Skipina	1968	Russia	1	Sputum	Emphysema, chronic productive cough
Memik	1968	USA	1	Empyema fluid	Lung abscess, hydropneumothorax
Fardy and March	1969	Canada	2	Resected lung tissue	Cavitating tuberculosis on therapy
Turgel and Balode	1973	Russia	1	Bronchial washings	Endobronchitis
Walzer <i>et al.</i>	1978	USA	1	Empyema fluid	Empyema, aspiration pneumonia
Miller <i>et al.</i>	1982	USA	1	Empyema fluid	Gastric carcinoma, gastrectomy, oesophageal-pleural fistula

* *Prospective study.*

culture of the empyema fluid yielded enterococcus, *Proteus morgani*, *Staphylococcus aureus* and an unspecified non-spore-forming gram-positive bacillus.

Pulmonary trichomoniasis

Walton and Bacharach (1963) described three patients with pulmonary trichomoniasis. They also presented 16 previously published reports describing 30 other cases between 1867 and 1942. Since 1942, 17 reports have appeared describing 39 patients. Table I lists 35 patients with pulmonary trichomoniasis without evidence of an intra-abdominal or genitourinary source. There were 31 males and four females; 19 of these patients were reported in a prospective Russian study by Tumka (1956). The remaining 16 cases of pulmonary trichomoniasis in table I were usually accidental findings in patients with chronic purulent pulmonary disease.

The reports of these cases indicate that large numbers of trichomonads were seen (Glaubach and Guller, 1942; Memik, 1968; Turgel and Balode, 1973). Trichomoniasis resolved spontaneously (Walton and Bacharach, 1963) or after the underlying pulmonary disease was treated with antibiotics, sulphonamides, metronidazole, surgical drainage or resection (Glaubach and Guller, 1942; Lehmann and Prendiville, 1946; Tumka, 1956; Miller *et al.*, 1982). Descriptions of the patients' oral cavities ranged from "good" (Khrushcheva and Kryazheva, 1951; Turgel and Balode, 1973) to "gingivitis" (Barbosa and Amaral, 1950) and "far-advanced pyorrhoea" (Glaubach and Guller, 1942). The repeated descriptions of copious, foul-smelling, purulent sputum and empyema fluid with "sterile" bacteriological cultures (despite bacteria having been seen on gram-stained smears) probably represents infection by anaerobic organisms from the mouth aspirated along with the trichomonads.

In the prospective Russian study, Tumka (1956) examined specimens of sputum, secretions obtained by bronchoscopy and surgically resected tissue to demonstrate trichomonads in the lungs of 19 of 112 patients (17%) with chronic pulmonary disease. Trichomonads were present in nine of 35 patients (26%) with lung cancer, five of 25 patients (20%) with lung abscesses, three of 18 patients (17%) with bronchiectasis, one of two patients with chronic bronchitis and one patient with pneumonia. Pulmonary flagellates were not detected in 25 patients with pulmonary tuberculosis. Flagellates were visible in wet smears in only three patients. In the remaining 16 patients, the diagnosis was established by culture. On the basis of morphological and cultural data, Tumka identified this organism as the oral *Trichomonas*, *T. tenax* (*sic T. elongata*).

TABLE II

Pulmonary trichomoniasis since 1942 with evidence of intra-abdominal or genitourinary source

Author	Year	Country	Number of cases	Specimen in which <i>T. tenax</i> was demonstrated	Associated illness
Houin <i>et al.</i>	1973	France	1	Empyema fluid, subphrenic abscess	Stomach cancer, gastrectomy, subphrenic abscess, empyema, bronchopleural fistula
Hoffman <i>et al.</i>	1968	Poland	1	Lung, liver, spleen, vagina	Metastatic carcinomatosis, <i>T. vaginalis</i> vaginitis
McLaren <i>et al.</i>	1983	USA	2	Infant tracheal aspirate, urine, nasopharynx	Pneumonitis; mothers had <i>T. vaginalis</i> vaginitis

TABLE III
Trichomonads in human blood

Author	Year	Country	Number of cases	Associated illness
Pentimalli	1923	Italy	1	Febrile intestinal and pulmonary disease
Kruscheva and Kryazheva	1951	Russia	1	Lung abscess, bronchiectasis, pulmonary trichomoniasis, meningitis
Trichereau <i>et al.</i>	1965	France	1	"Weakness"

Tumka believed that the trichomonads were unable to cause disease on their own but simply multiplied freely after "travelling" from the oral cavity into a favourable, already purulent and necrotic pulmonary environment. He speculated that these numerous flagellates might be responsible for adding to the general patient debilitation and prolonging the duration of illness.

Table II lists patients with pulmonary trichomoniasis reported since 1942 with an intra-abdominal or genitourinary source for their infection. *T. hominis* was found in the subphrenic abscess and empyema in the post-gastrectomy patient reported by Houin *et al.* (1973). Intrathoracic extension from pyogenic (Navarro and De Alzaga, 1933) and amoebic (Kessel, 1925; Gray and Andrews, 1932) liver abscesses containing *T. hominis* had been reported before 1942. *T. vaginalis* was isolated from metastatic cancer foci by Hoffman *et al.* (1966) and from the respiratory tract of infants by McLaren *et al.* (1983). Trichomonads have been found in other diseased organs near the oral cavity, including sinusitis (Teisanu and Popescu-Tomus, 1971), tonsillitis (Tumka, 1957), jaw abscess (Macaskill, 1916), cancer of the tongue and oesophagus (Cohnheim, 1902), and in the hypochlorhydric stomach (Jirovec and Petru, 1968). (*T. tenax* cannot live in an acid environment with a pH less than 5.0 (Probst, 1967).) Trichomonads have been found in human blood (table III) and cerebrospinal fluid (Masur *et al.*, 1976).

Trichomonas tenax

T. tenax, referred to in older works as *T. buccalis* and *T. elongata*, is a pear-shaped, flagellate protozoon with an undulating membrane. It is of world-wide distribution in man and monkeys, its natural hosts (Levine, 1973). *T. tenax* is regarded as a harmless commensal of the human mouth. It feeds on microorganisms in its environment. It is found in and around diseased and necrotic teeth and gums. The active participation of the flagellate in oral inflammatory disease has been suggested by improvement of diseases after metronidazole therapy (Rousset and Lauvergeat, 1971). Infection is spread through saliva, droplet spray and kissing, or on contaminated dishes, glasses and hands (Belding, 1965; Faust *et al.*, 1970). It may survive in tap water for periods from hours to several days (Beatman, 1933).

Wantland *et al.* (1963) described the laboratory techniques for the proper collection of oral material for study, for the proper preparation of wet-smears, and for the identification of the organism in wet-smears and cultures. Descriptions of the light

microscopic structure and mode of division, as well as methods for fixing, staining and culturing this organism are available (Wenrich, 1944; Honigberg and Lee, 1959; Diamond, 1962; Wantland *et al.*, 1963; Belding, 1965; Probst, 1967; Faust *et al.*, 1970; Levine 1973). Because of their morphological resemblance to other trichomonads, their marked protoplasmic plasticity (Wenrich, 1944) and the environmental and genetic variation in organism size and shape (Honigberg and Lee, 1959), identification of flagellate species using only the wet-preparation is unreliable. More accurate methods, such as culture and perhaps serology, should be used for accurate species identification.

Kott and Adler (1961) demonstrated two specific types of antibodies to *T. tenax*. The varied microbial flora with which *T. tenax* is usually grown may be influential in enabling the organism to grow in either aerobic or anaerobic media, and in diverse conditions (Ohara, 1967). Studies on the physiology and nutrition of *T. tenax* (Probst, 1967), as well as fluorescence microscopy (Sato, 1957), cytochemical studies (Wantland *et al.*, 1962; Ohashi, 1971 and 1972), electronmicroscopic studies (Angelopoulos and Angelopoulos, 1972) are available. *T. tenax* can be preserved by freezing (Diamond, 1964). Culture is the superior technique for the detection of *T. tenax*. From 4% to 24% of patients tested had negative wet-smear examinations but positive cultures (Beatman, 1933; Bland and Rakoff, 1937; Wantland *et al.*, 1958; Wantland and Wantland, 1960). Trichomonads can be detected in gram-stained smears where they appear gram-negative (Cree, 1968). Walton and Bacharach (1963) appropriately described trichomonads seen in Papanicolaon-smears as "pale gray blobs of poorly

FIG. 2.—Age distribution of oral colonisation with *T. tenax* in three series.

staining protoplasm rarely showing any morphologic detail." Trichomonads are difficult to identify in histological sections.

The prevalence in the mouth of *T. tenax* in several series ranged from 4% to 53% (Bland and Rakoff, 1937; De Carneri, 1957; Wantland *et al.*, 1958; Wantland and Wantland, 1960; Jaskoski, 1963; De Carneri and Giannone, 1964; Wantland and Lauer, 1970) and varies with age (fig. 2). It may exceed the prevalence in the vagina of *T. vaginalis* in adult females (Trussell, 1947; De Carneri and Giannone, 1964). The greatest incidence in male patients over 50 years of age may be related to the generally "dirtier" mouths and poorer personal oral hygiene seen in this group. The small quantity of fluoride in the drinking water of patients studied did not alter the prevalence of this organism (Wantland and Lauer, 1970). The flagellate is killed *in vitro* by desiccation, hydrogen peroxide (Takada *et al.*, 1960), alcoholic beverages and fruit juices (Ota, 1935). Normal gastric juice, small bowel contents and bile acids may be toxic to this trichomonad (Shigeura, 1932). *T. tenax* and *T. vaginalis* were both present at their respective normal sites in 4.5% (Bland and Rakoff, 1937) and 11% (De Carneri and Giannone, 1964) of female patients tested. All three trichomonads were found in 0.5% of female patients (Bland and Rakoff, 1937). The three trichomonad species could not be experimentally transmitted to each other's environment (Westphal, 1936; Stabler and Feo, 1942).

Discussion

Tumka (1956) used morphological and cultural criteria to identify this pulmonary flagellate as the oral *T. tenax*. Other common mouth inhabitants such as the spirochaetes and fusiform bacteria found in the lungs along with the trichomonads were probably also aspirated. Anaerobic bacteria from the mouth undoubtedly play an important role in the production and maintenance of the foul-smelling, purulent and necrotic pulmonary milieu commonly described. I believe my patient's pulmonary *Trichomonas* was *T. tenax*. His poor oral hygiene, prolonged comatose state, tracheostomy with periodic cuff deflation, and lack of primary gastrointestinal disease or intra-abdominal communication all point to *T. tenax* aspirated from the mouth as the agent. Tumka's pulmonary trichomoniasis prevalence rate of 17% in chronic pulmonary disease is surprisingly high. The increased use of *Trichomonas* cultures and the closer attention to wet-preparations, gram-stained smears, Papanicolaou-stained smears, and, perhaps, serology will confirm or contradict Tumka's statistics. The predominance of males in table I is consistent with the male predominance of purulent and malignant pulmonary disease.

Trichomonas species and strains have variable genetically-determined pathogenicity. While some trichomonads are completely harmless, strains of the avian species *T. gallinae*, for example, will kill their natural hosts within a week (Honigberg, 1961). Teras and Roigas (1966) demonstrated a marked difference in virulence among strains of *T. vaginalis*. Honigberg *et al.* (1971) demonstrated that the pathogenicity of a normally benign *Trichomonas* strain may be enhanced by a DNA-RNA mediated virulence transformation. Future work will show the degree of *T. tenax* virulence. Do the *Trichomonas* strains found in the mouth have the same virulence as the *Trichomonas* strains found in the lungs? Is there a synergistic effect between *T. tenax* and other pulmonary organisms found in the debilitated hosts?

The presence of the organism in a diseased host does not mean the organism caused the disease. Until the question of pathogenicity is resolved, no definitive statement regarding the indications for therapy, the kind of therapy nor its route of administration, can be made. Until these organisms are proved to be harmless, a trial of metronidazole would seem worthwhile. Vigorous therapy should be directed at the underlying pulmonary disease.

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