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CASE REPORT

Lactobacillus pneumonia in a patient with oesophageal carcinoma

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Lactobacillus pneumonia in a patient with oesophageal carcinoma, J.M. Querol, F. Manresa, J. Izquierdo, M. Cisnal.
ABSTRACT: We report a case of necrotizing pneumonia caused by *Lactobacillus* secondary to a tracheo-oesophageal fistula created by an oesophageal carcinoma. We emphasize the presence of resistance of *Lactobacillus* to clindamycin and cotrimoxazole, previously reported to be effective.
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Lactobacilli are non-sporeforming, catalase-negative, Gram-positive rods that are generally strictly or facultatively anaerobic bacteria, and are part of the normal flora of the mouth, vagina and gastrointestinal tract [1, 2]. They are involved in the pathogenesis of dental caries. Since the organism resembles diphtheroids morphologically, it is often considered to be a contaminant when isolated from specimens other than blood [1, 2].

Until 1978, 25 cases of serious human infections caused by *Lactobacillus sp.* had been reported [2]: 13 endocarditis, 9 sepsis arising from localized suppuration especially involving the oropharynx, 2 pneumonia with empyema and one case of meningitis. In 19 of the 25 patients the portal of entry was adequately identified (oropharyngeal 11 cases, female genital tract 4 cases, and gastrointestinal tract 4 cases). In the remaining 6 cases no focus of sepsis was identified. In 1986 two more cases of endocarditis due to *Lactobacillus* were published by DAVIES *et al.* [3].

Lactobacilli are usually sensitive to penicillin, ampicillin, clindamycin and cephalothin. Penicillin-tolerance has been described; DAVIES *et al.* [3] in 1986 reported a case and concluded that in deep infections the treatment of choice would be the combination of a penicillin in high dosages with an aminoglycoside.

Case Report

A 40 yr old man presented with an eight week history of cough with fetid expectoration, fever, anorexia and a 10 kg weight loss. He was a smoker of more than 40 packs per year and a heavy drinker (daily alcohol intake >100 $g \cdot day^{-1}$).

Physical examination revealed a patient with toxic appearance, malaise, a temperature of 39.2°C, multiple carious teeth, basilar râles on auscultation and hepatomegaly. Laboratory studies on admission revealed a haematocrit value of 35%, haemoglobin 10.5 $g \cdot dl^{-1}$, ESR (erythrocyte sedimentation rate) 128 $mm \cdot h^{-1}$, Quick 82%, total proteins 60 $g \cdot l^{-1}$, (albumin 16.8 $g \cdot l^{-1}$), urea nitrogen 3.4 $mmol \cdot l^{-1}$, sodium 132 $mmol \cdot l^{-1}$, white blood cell count 15,200·mm^3 (2% bands, 77% polymorphonuclears and 17% lymphocytes) and platelets 427,000. Two blood cultures on admission were negative. A chest film (fig. 1) showed an alveolar pattern with microcavitation on the right upper lobe suggesting necrotizing pneumonia.

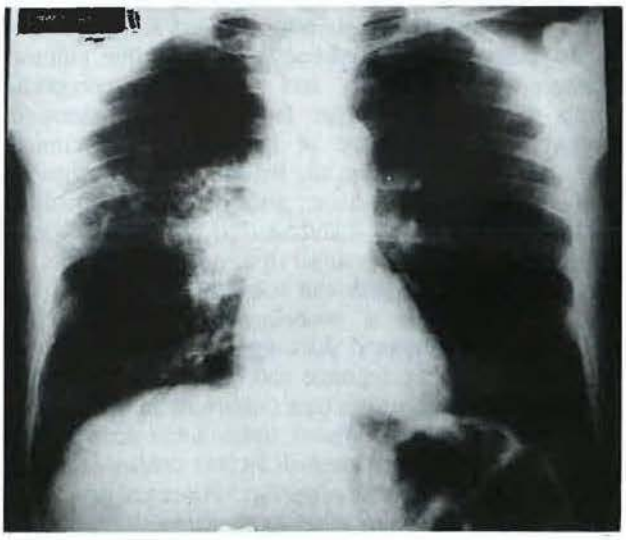

Fig. 1. – Chest roentgenogram on admission showing an alveolar pattern with microcavitation on the right upper lobe suggesting necrotizing pneumonia.

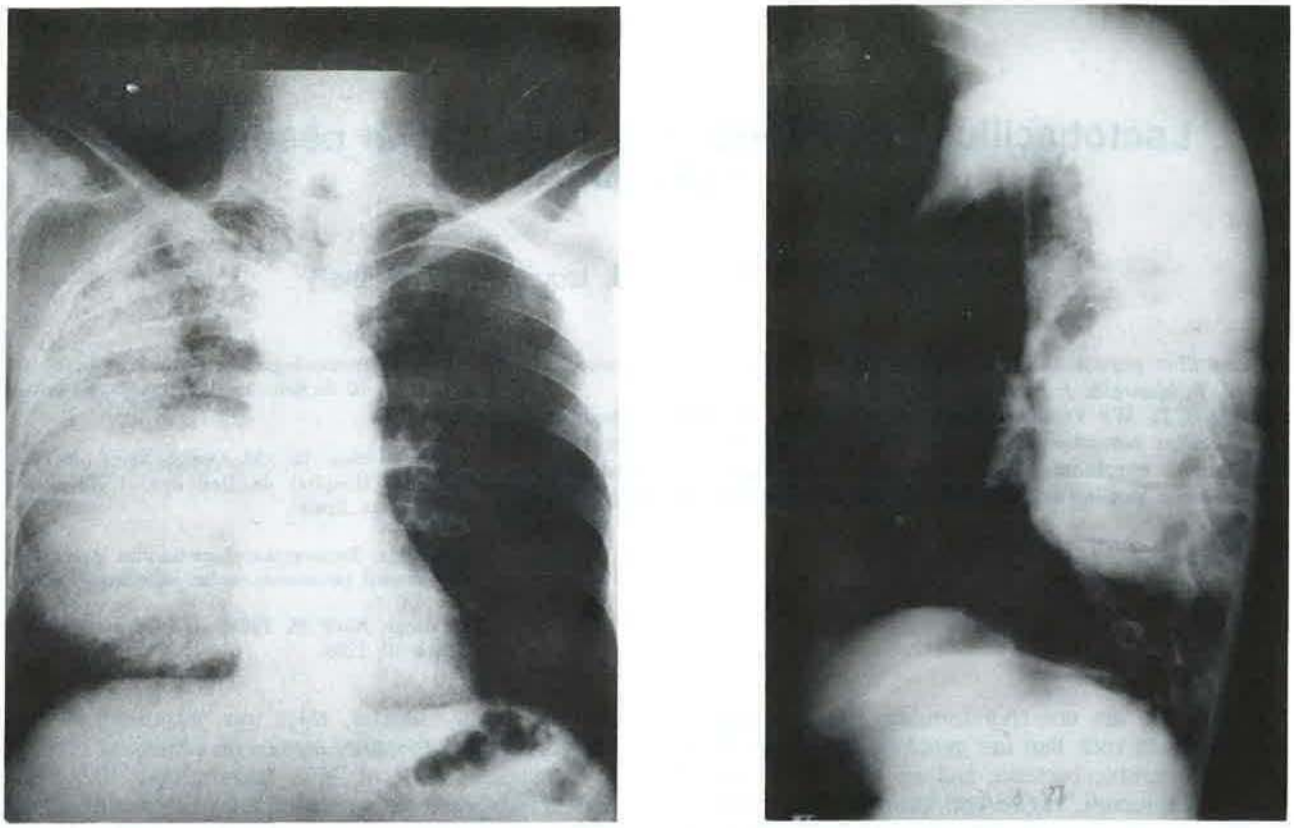

Fig. 2. – Chest roentgenogram 14 days after admission showing radiological progression to include the right lower lobe; uniform central area corresponds to a posterior empyema. PA view left, right lateral view right.

The patient was treated with 12 million U·day^{-1} of penicillin intravenously (*i.v.*). Four days later the persistence of fever and radiological progression with larger cavitation led us to consider a change of therapy to clindamycin 600 mg per 6 h *i.v.* After two days of clindamycin, a transthoracic needle aspiration (TNA) was performed yielding many polymorphonuclear white blood cells and Gram staining showed Gram-positive bacilli identified as *Lactobacillus sp*; routine cultures (Löwenstein, anaerobic, and fungi) in appropriate media were negative. After four days of clindamycin therapy with persistence of high fever and clinical radiological deterioration, the treatment was changed to cefmetazole 2 g per 8 h *i.v.*, after obtaining two blood cultures that yielded *Lactobacillus sp.* Antibiogram revealed penicillin sensitivity and resistance to clindamycin, tobramycin and cotrimoxazole.

Three days later a bronchoscopy with bronchial brushing was performed showing a stenosis of the apical right lower lobe segment and infiltration of the mucosa suggesting neoplasia (not confirmed in the biopsy); specimen from brush catheter (quantitated according to WIMBERLEY *et al.* [4]) yielded 50,000 colony- forming units (CFU)·ml^{-1} of *Lactobacillus*. Fourteen days after admission a chest film (fig. 2) revealed the existence of posterior empyema that required chest tube drainage; the cytological study of this pleural fluid showed vegetative cells and striated muscle suggesting a tracheo-oesophageal fistula.

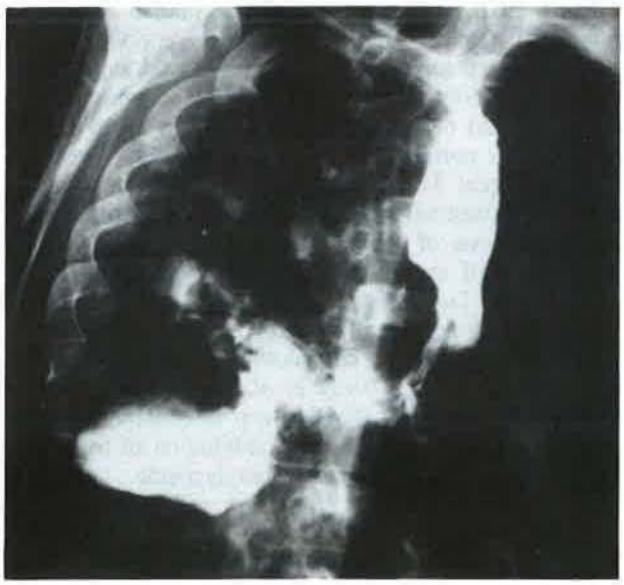

Fig. 3. – Oesophagogram showing the oesophageal carcinoma and barium in the abscess cavity.

An oesophagogram (fig. 3) confirmed the fistula and the endoscopic procedure suggested oesophageal carcinoma; the biopsy was compatible with squamous-cell carcinoma. An oesophageal prosthesis was inserted obtaining a rapid clinical improvement of the patient who could then tolerate an oral diet.

Twenty one days after admission, bleeding through the aspirate drainage was observed, and six days later the patient died from massive haemoptysis.

Discussion

We present a patient with risk factors for anaerobic lung infection (septic mouth, neoplasia, hypoproteinaemia and fistula) whose fatal clinical course was determined not by the *Lactobacillus* pathogenicity but by his basal status.

The radiographic persistence and worsening of condensation with lung abscess formation must be attributed to: 1) a persistent tracheo-oesophageal fistula which maintained an open and direct communication perpetuating infection, and creating an abscess and empyema: and 2) carcinomatous infiltration that caused the fatal massive haemoptysis.

Therapeutic changes to different antibiotics were unable to control the lung infection, even though the initial antibiotic regimen with penicillin was adequate. The inefficacy of penicillin can be attributed to: 1) deficient host defence mechanisms (alcoholism, neoplasia); and 2) local anatomical alteration that maintained broncho pulmonary infection preventing adequate drainage.

The tolerance of *Lactobacillus* to clindamycin was confirmed in our case (minimal inhibitory concentration (MIC) >8 $\mu g \cdot ml^{-1}$ and minimal bactericidal concentration (MBC) >8 $\mu g \cdot ml^{-1}$. MICs and MBCs were determined by a broth microdilution technique.

Sensitivity testing, in this case, showed no discrepancy between MICs and MBCs for penicillin, as previously reported [2]. In our case MIC was 0.25 $\mu g \cdot ml^{-1}$ and MBC 0.25 $\mu g \cdot ml^{-1}$ for penicillin.

Lactobacillus growing as a single bacteria in different procedures (TNA, brush catheter and blood cultures) confirm the pathogenicity of this bacterium and indicate that pneumonia was probably the focus of sepsis. Positive blood cultures showed bacteraemia by *Lactobacillus*.

We know of only two cases of pneumonia with empyema attributed to *Lactobacillus* reported in the literature [2, 5]. One of the two cases, was a patient with an empyema with a gastropleural fistula and the aetiological relationship between the infection and the fistula was similar to our case. The second case involvedbacteraemia by *Lactobacillus* in a patient with pneumonia, in which this relationship had not been proven [5]. Our case is, therefore, probably the first report of pulmonary infection proven to be caused by *Lactobacillus sp.* In 1982 a case of thoracic infection by *Lactobacillus casei vr. rhamnosus* isolated from sputum was reported [6].

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RÉSUMÉ: Observation d'une cas de pneumonie nécrosante provoquée par un Lactobacillus et consécutive à une fistule trachéo-oesophagienne due à un carcinome de l'oesophage. Nous insistons sur la présence d'une résistance du Lactobacillus à l'égard de la clindamycine et du cotrimoxazole, dont on avait singnalé antérieurement l'efficacité.
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