

lesson of the month (2)

Pig trotters lung - novel domestic transmission of *Pasteurella multocida*

Pasteurella multocida is a non-motile, facultatively-anaerobic, gram-negative bacillus associated with a spectrum of human disease.¹ Direct and indirect zoonotic transmission is recognised with animal bites being most frequently encountered as a result of salivary colonisation in farm and domestic animals.² Despite the prevalence of *P. multocida* in swine herds,³ the relationship between porcine colonisation and human disease is poorly established. This lesson reports a previously unrecognised mode of zoonotic transmission in respiratory pasteurellosis; domestic cooking of pig trotters.

Lesson

An 81-year-old man presented with a four-day history of worsening shortness of breath, mucopurulent cough and fever. He was an ex-smoker (120 pack-years) with a background of chronic obstructive pulmonary disease (COPD). On admission he was tachypnoeic, pyrexial (temperature 37.9°C), tachycardic (122 beats per minute) and hypotensive (92/48 mmHg) with decreased breath sounds, dull percussion note and coarse inspiratory crackles at the right lung base. Blood investigations revealed leucocytosis $16.1 \times 10^9/l$ with neutrophilia, C-reactive protein (CRP) 341 mg/l and type 1 respiratory failure (PaO₂ 6.7 kPa, pH 7.367, PaCO₂ 5.6 kPa).

Chest radiograph and ultrasound imaging confirmed a moderately-sized right pleural effusion with multiple septae and consolidated lung (Fig 1). After initial resuscitation and empirical intravenous co-amoxiclav, a chest drain was inserted. Pleural fluid analysis revealed an exudate (30 g/l protein) of numerous neutrophils and no malignant cells. Gram stain revealed gram-negative bacilli identified as *P. multocida* on culture. Sputum culture grew *Pseudomonas aeruginosa* only. Despite amoxicillin sensitivity of *Pasteurella spp.* resistance was identified to *Pseudomonas spp.* and

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accordingly single-agent imipenem was employed to target both organisms.

Following an initial fall in inflammatory markers CRP remained elevated at approximately 100 mg/l and repeat ultrasound imaging showed multiple small loculations unsuitable for tubal drainage. Thoracoscopic decortication was considered too high risk. Consequently, medical treatment continued with a 21-day course of intravenous imipenem followed by four-weeks of oral amoxicillin. Complete recovery was observed eight weeks after presentation.

In view of isolation of *P. multocida*, detailed questioning established that the patient had no domestic pets and denied feline, canine or farm exposure, foreign travel or exposure to unwell contacts. The only possible exposure to *P. multocida* was five days prior to the onset of symptoms when he soaked pig trotters in salted tap water at room temperature for 48 hours before boiling uncovered for one hour and ingesting as his evening meal.

Discussion

Human respiratory tract pasteurellosis has been noted as epiglottitis, sinusitis, tracheobronchitis, pneumonia and empyema formation.² Pathophysiology of infection is underpinned by oropharyngeal colonisation. In healthy individuals and asymptomatic patients with COPD, colonisation has been reported at 5% and 25% respectively, accounting for variation in



Fig 1. Anteroposterior erect chest radiograph demonstrating a right pleural effusion with consolidated lung and a dual pacemaker in situ.

isolation and clinical significance.^{2,3} Aerosol inhalation of the microorganism is therefore an important step in the development of oropharyngeal and tracheobronchial tree colonisation. Underlying lung disease, advancing age as well as concomitant immunosuppression are thought to further determine subsequent development of respiratory tract disease and in part correlate with an associated mortality of 31% in *P. multocida* associated pneumonia.^{2,3}

P. multocida is a commensal of swine respiratory and gastrointestinal tracts and survival of *P. multocida* at scalding water tank temperatures is thought to explain contamination of most pigs during slaughter.³ It is therefore speculated that *P. multocida* colonises pig trotters and in this case physically agitating the organism during soaking and cooking allowed fragmentation of biological material originating from porcine saliva or faeces. Transmission is likely to have been facilitated by uncovered boiling of the pig trotters which would not have killed *P. multocida* but rather pro-

moted further airborne spread of droplets containing this unusual and virulent pathogen.

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letters to the editor

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'With a pinch of salt' revisited

Editor – I read with interest but some concern the recent lesson of the month (*Clin Med* February 2010 pp 86–7). The report highlights a case of severe hyponatraemia which the authors attribute to excessive sweating, poor fluid consumption and low salt intake in a hot environment. There is little evidence to suggest that a low salt intake would contribute to this event and the authors have not explored alternative likely explanations. During evolution, mankind has survived with very little salt in the diet. Even in modern times, this evidence is detectable in the Yanomano and Xingu Indians living in the humid and hot environment of the Amazon jungle.¹ Their average salt intake, when measured by 24-hour urine collections, varies between 1 and 10 mmol/day. These levels, however, are almost unseen in the western world

due to the high salt intake we are exposed to, even when adhering to a low salt diet. Under conditions of exercise in a hot environment, a low salt intake does not impair the ability to exercise, and it does not cause changes in plasma sodium, potassium, osmolality or sweat rate, although the salt content of sweat is reduced on a low salt intake diet.² Gangopadhyay *et al* misquote the evidence in athletes and the military where the high morbidity from hyponatraemia is due to overhydration (ie too much water) rather than a low salt intake. The case presented here is clearly a case of diuretic abuse, surreptitious vomiting or laxative abuse, as we described in the past in a different scenario.³ While hyponatraemia may possibly have been caused by water intoxication, it would not have caused plasma potassium to fall so low, or the renin–angiotensin system to be so stimulated. The authors do not seem to

have considered screening for diuretics. Diuretic abuse would explain hyponatraemia, alkalotic hypokalaemia and activation of the renin–angiotensin–aldosterone system as described here. A moderate reduction in salt intake (up to 3 g per day) does not raise cause for concern and should be recommended to everyone to prevent cardiovascular disease and other common conditions like kidney stones and osteoporosis.⁴

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