cefixime (400 mg daily) orally instead of linezolid because of the high cost and potential toxicities associated with long-term use of linezolid, such as bone marrow suppression, lactic acidosis and peripheral neuropathy.

Discussion

Nocardia species are common natural inhabitants of the soil throughout the world. Pulmonary nocardiosis is usually acquired by direct inhalation of nocardia species from contaminated soil. One study reported the presence of underlying disease including chronic obstructive pulmonary disease, HIV infection, neoplastic disease, alcoholism and corticosteroid therapy in 8 of 10 cases of pulmonary nocardiosis. [2]. Pulmonary nocardiosis has a tendency towards remissions and relapse. There is often necrotizing pneumonia, commonly associated with cavitations. The most common symptoms include fever, cough, expectoration, dyspnoea, chest pain and, to a lesser degree, haemoptysis and abdominal pain [3].

The choice of therapy depends on the severity of the infection, host immunity, potential drug interactions and toxicity, and whether the infection is localized or has spread to other organs [1]. Historically, sulphonamides were the treatment of choice but certain types of Noracia, such as farcinica and ostitidiscaviarium, are resistant to sulphonamides. Linezolid is another antibiotic that has recently shown excellent in-vitro activity against all of the clinically relevant species of Nocardia [4]. The duration of treatment is usually 6–12 months for immunocompetent patients with pulmonary nocardiosis or disseminated nocardiosis outside the central nervous system. For immunosuppressed patients, however, treatment should continue for at least 1 year. Nocardial localized abscesses generally require prompt surgical therapy. Surgery should be performed for large and readily accessible brain abscesses, and also for progressive lesions beyond 2 weeks in spite of adequate medical therapy.

In conclusion, the diagnosis of pulmonary nocardiosis requires a high degree of suspicion because of nonspecific presentation, difficulty in isolating the organism and high mortality with delayed treatment. Although the disease mainly affects immunocompromised patients, our case shows the importance of considering the diagnosis in immunocompetent patients who face certain occupational hazards.

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Respiratory distress in children: another cause to be considered

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Acute respiratory distress in children has a differential diagnosis including asthma, bronchiolitis, pneumonia, anaphylaxis, sepsis, airway obstruction, pain, arrhythmia, pneumothorax, anxiety and metabolic acidosis [1]. Respiratory compensation for metabolic acidosis in children with diabetic ketoacidosis is well known [2]. We are not aware of prolonged hyperventilation after exercise being reported in a child with metabolic acidosis and raised capillary lactate.

An 11-year-old girl presented to the emergency department in acute respiratory distress after completing a 1 km run. Her tachypnoea failed to resolve after she stopped and was still evident 45 min after exercise was completed.

Examination showed no wheeze or crackles on chest auscultation. She had good air entry throughout, oxygen saturations were 100% and a chest radiograph was normal. She was an otherwise healthy girl with appropriate development for her age. Venous blood investigations were unremarkable with values within normal limits for her age and normal inflammatory markers.

A bedside test was available for capillary blood lactate measurement, and a capillary blood gas was sent to the laboratory. Her capillary blood analysis at 45-min post exercise showed a pH of 7.3 and an elevated capillary lactate of 9.2 mmol/l. The clinical examination findings at the time of presentation and when rechecked after an hour are summarized in Table 1.

On the basis of these parameters, without any evidence for another cause found, the only positive findings suggested a diagnosis of a metabolic acidosis secondary to raised lactate post exercise.

Her hyperventilation settled with conservative treatment over the following hour. A repeat capillary blood lactate had reduced to 2.1 mmol/l and pH returned to normal range. Peak expiratory flow rate when performed was 100% of expected for her sex and height.

Capillary blood lactate levels increase in exercise as skeletal muscle lactate production exceeds elimination [3]. Acidosis because of a failure of lactate buffering is a major determinant for hyperventilation in exercise [4].

In children, a lactate concentration above 2.5 mmol/l has been proposed as a level beyond which further exercise would lead to lactate accumulation in capillary blood [5].

Table 1 Summary of clinical examination and investigations

	End of race time, 0 min	Acute presentation time, 45 min	Repeat time, 105 min
Pulse	–	135/min	102/min
Respiratory rate	–	90/min	24/min
Peak expiratory flow rate	–	Unable to do	320 l/min 100% of predicted
Oxygen saturations	–	100%	100%
Temperature	–	36.9°C	36.9°C
Chest auscultation	–	Air entry equal Breath sounds normal	Air entry equal Breath sounds normal
Chest radiography	–	Unremarkable	–
Capillary blood glucose	–	5.3 mmol/l	5.7 mmol/l
Capillary blood pH	–	7.30	7.37
Capillary blood HCO3	–	18.9 mmol/l	25.4 mmol/l
Capillary blood base excess	–	– 6.8 mEq/l	– 0.2 mEq/l
Capillary blood lactate	–	9.2 mmol/l	2.1 mmol/l

This accumulation of lactate in exercise and subsequent metabolic acidosis are important stimuli to drive the respiratory compensation of hyperventilation [4].

However, 9.2 mmol/l is an exceedingly high capillary lactate accumulation in a child 45 min after exercise had stopped and one would have expected metabolic compensation to have occurred. Our patient's fitness was described as compatible with her peer group and on review in the respiratory clinic no respiratory or metabolic cause has been found for her symptoms to date.

The most important point from this case is that it would have been easy, but incorrect, to treat this young girl as an acute asthma presentation. The early detection of the lactate accumulation was facilitated by a bedside blood lactate monitoring system. Over breathing in children may have a metabolic component that is important to consider in any differential diagnosis of hyperventilation.

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