P. aeruginosa (Table 2). Rural waterways were sampled at random and all sampling took place within an area that represented prime agricultural grazing land for sheep and cattle. Sampling at each site was performed by aseptically obtaining 100 mL water in a sterile disposable plastic universal container (Sterilin). All samples were processed within 6 h of sampling. Individual water samples (100 μ L) were inoculated on PIA medium and spread uniformly on the surface of the agar with the aid of an L-shaped spreader, and the plates were incubated at 37°C for 72 h. Presumptive positive isolates were further characterised by sequence analysis of their near complete 16S rDNA gene locus.

P. aeruginosa was not detected in any air sampled, nor was it found in any surface water examined. However, eight surface waters were positive for other *Pseudomonas* species, including *P. fluorescens* (n=2) and *P. tolaasii* (n=1). Five other waters sources were positive for other species of *Pseudomonas* (Table 2). All resulting 16S rDNA sequences relating to these organisms have now been deposited in GenBank and their accession numbers are given in Table 2.

Water is a well-documented environmental source of this organism;³ however, there are a limited number of published studies describing its presence in water-related activities associated with agriculture and agricultural practices. Many CF patients and their families are concerned to avoid the acquisition of this organism in the lower airways and hence try to avoid high-risk situations/environments where transmission may occur, including swimming, contact with other CF patients infected with *P. aeruginosa* and avoidance of spas and jacuzzis. As a result, many CF patients ask their clinicians and nurses about the risk from recreational use of water in rural areas, where agriculture is the most important factor, as well as the avoidance of inhaling air contaminated with the smell of animal slurry during periods of high slurry spreading activity.

The inability to isolate this organism from intensive agricultural environments is surprising and suggests that the organism may not be as ubiquitous as thought originally. However, given that other species of *Pseudomonas* were cultured from environmental surface waters, CF patients should still consider water and associated work or recreational activities as high risk, where such water may be contaminated with several species of *Pseudomonas*.

The aerial sampling component of this study was funded through the Small Grants Initiative Scheme, awarded by the Environmental Protection Agency, Dublin, Ireland.

References

- 1 Anon. About cystic fibrosis; What is CF? 2003 (www.cff.org).
- 2 Clarke L, Moore JE, Millar BC *et al.* Development of a diagnostic PCR assay that targets a heat-shock protein gene (*groES*) for detection of *Pseudomonas* spp. in cystic fibrosis patients. *J Med Microbiol* 2003; **52**: 759–63.
- 3 Govan JRW, Deretic V. Microbial pathogenesis in cystic fibrosis: mucoid *Pseudomonas aeruginosa* and *Burkholderia cepacia*. *Microbiol Rev* 1996; 60: 539–74.
- 4 Lyczak JB, Cannon CL, Pier GB. Lung infections associated with cystic fibrosis. *Clin Microbiol Rev* 2002; 15: 194–222.
- 5 Rajan S, Saiman L. Pulmonary infections in patients with cystic fibrosis. *Semin Respir Infect* 2002; 17: 47–56.

Congee: a cause of gross but transient elevation in plasma creatinine concentration

H. C. H. LEE*, M. C. MAK*, P. C. TONG^{\dagger}, T. S. SIU^{\dagger}, P. L. S. CHEN^{\$}, C. K. CHING^{\$}, W. T. POON^{\ast}, W. L. T. MAK^{\$}, S. TAM^{$\dagger$} and Y. W. A. CHAN^{$\ast$}

^{*}Chemical Pathology Laboratory, Department of Pathology; ^{*}Department of Paediatrics and Adolescent Medicine, Princess Margaret Hospital; [‡]Division of Clinical Biochemistry, Queen Mary Hospital; and [§]Hospital Authority Toxicology Reference Laboratory, Princess Margaret Hospital, Hong Kong SAR, China.

A previously healthy 14-month-old boy was admitted to hospital with diarrhoea. Clinical examination was unremarkable. Apart from a markedly elevated plasma creatinine level (222 μ mol/L; Roche Modular Analytics, Indianapolis, USA), routine blood tests, including plasma urea, were normal. By the next day his plasma creatinine had spontaneously normalised to 39 μ mol/L (Table 1). He was discharged after the diarrhoea settled and subsequently referred for exclusion of renal pathology.

When seen three months later, there was no abnormality on examination but plasma creatinine concentration was 214 μ mol/L (Beckman Coulter Synchron, Fullerton, USA) which when repeated four days later was normal (45 μ mol/L). Other biochemical and haematological parameters were normal, as were ultrasonography of the renal system and a dimercaptosuccinic acid renal scan. As the creatinine assay based on the Jaffé reaction is subject to interference,¹ creatinine was re-analysed using the creatininase-creatinase enzymatic method (Vitros Chemistry system, Ortho-Clinical Diagnostics, Rochester, USA), which confirmed the elevated plasma creatinine and provided no evidence for assay interference (Table 1). Urinary toxicology screen revealed no abnormality.

On further specific questioning, the parents denied giving any herbs, health products, traditional medicine or drugs to the child but stated that the child had been given pork congee (concentrated meat broth) cooked from 500 g lean pork prior to each hospital visit. Therefore, plasma creatine (high-performance liquid chromatography [HPLC]-tandem MS) was measured and found to be 424 µmol/L and 167 µmol/L (reference interval: 17–109 µmol/L) in the plasma samples with a creatinine of 214 µmol/L and 45 µmol/L (Table 1).

Dietary creatinine and creatine affect plasma creatinine concentrations. The effect of plasma creatine interference in the Jaffé creatinine assay is small.² *In vivo* conversion of creatine to creatinine occurs at an average rate of 1.6% daily,³ and therefore normally only makes a small contribution to circulating creatinine concentration. Jacobsen *et al.*⁴ and Mayersohn *et al.*⁵ reported that plasma creatinine levels increase by 50% one and a half to three and a half hours after ingestion of a cooked meat meal. As raw meat ingestion does not give similar effect, the authors suggest that creatinine is produced from creatine during cooking and ingested from the cooked meat.

Correspondence to: Chloe Miu Mak Chemical Pathology Laboratory, Department of Pathology, Princess Margaret Hospital Hong Kong SAR, China.

Email: makm@ha.org.hk

14 April 15:05 15 April 09:20 Interval 10 July 16:30 14 July 10:21 Range 132-145 139 135–149 Sodium (mmol/L) 137 140 138 Potassium (mmol/L) 4.4 4.8 3.1-5.1 4.2 4.2 3.5-4.7 5.9 3.3-7.0 Urea (mmol/L) 5.9 <8.4 4.9 5.3 Creatinine (µmol/L) Jaffé method 222 39 21-36 214 45 30-90 Enzymatic method 45 10-60 232 Creatine (µmol/L) 424 167 17-109

 Table 1. Plasma electrolytes, creatinine and creatine concentrations.

Creatinine is readily absorbed in the gastrointestinal tract⁶ and has a half-life of 3.85 hours. One hundred grams of cooked lean pork contains 0.73 mmol (96 mg) creatinine.⁷ Assuming that the child consumed congee from 250 g cooked lean pork meat per meal, each meal provided him with 1.83 mmol (240 mg) creatinine. Therefore, the authors suggest that the plasma creatinine was markedly elevated after ingestion of congee and that it normalised in the absence of congee consumption.

Plasma creatinine concentration, a measure of the glomerular filtration rate, is the single most commonly used index of renal function in clinical practice.⁶ The effect of cooked meat consumption on plasma creatinine concentration is often not considered as important. Preiss *et al.*, however, reported an increase in plasma creatinine following ingestion of cooked meat, from 80.5 μ mol/L to post-prandial concentrations of 101 μ mol/L and 99 μ mol/L at 1–2 h and 3–4 h, respectively.⁷ Therefore, it is recommended that blood samples should be taken in fasting or fed status without cooked meat to avoid misclassification of chronic kidney disease.⁷

In summary, the authors report a dramatic increase in plasma creatinine following ingestion of congee in a child, which normalised rapidly after brief abstinence from consumption of congee. This case illustrates the potential effect of cooked meat ingestion on plasma creatinine concentration. It is important for the clinician to recognise this entity in order to avoid unnecessary investigations, hospitalisation and parental anxiety.

References

- 1 Weber JA, van Zanten AP. Interferences in current methods for measurements of creatinine. *Clin Chem* **1991**; **37**: 695–700.
- 2 Taes YE, De Vriese AS. Analytical and biochemical aspects associated with supraphysiological creatine intake. *Clin Chim Acta* 2005; **351**: 217–9.
- 3 Heymsfield SB, Arteaga C, McManus C, Smith J, Moffitt S. Measurement of muscle mass in humans: validity of the 24-hour urinary creatinine method. *Am J Clin Nutr* 1983; **37**: 478–94.
- 4 Jacobsen FK, Christensen CK, Mogensen CE, Andreasen F, Heilskov NS. Pronounced increase in serum creatinine concentration after eating cooked meat. *Br Med J* 1979; **1** (6170): 1049–50.
- 5 Mayersohn M, Conrad KA, Achari R. The influence of a cooked meat meal on creatinine plasma concentration and creatinine clearance. *Br J Clin Pharmacol* 1983; **15**: 227–30.
- 6 Perrone RD, Madias NE, Levey AS. Serum creatinine as an index of renal function: new insights into old concepts. *Clin Chem* 1992; **38**: 1933–53.
- 7 Preiss DJ, Godber IM, Lamb EJ, Dalton RN, Gunn IR. The influence of a cooked-meat meal on estimated glomerular filtration rate. *Ann Clin Biochem* 2007; **44**: 35–42.