***Salmonella bonariensis* Salmonellosis, Rhabdomyolysis, and Acute Renal Failure**

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Dear Sir,

In contrast to *Salmonella typhi* infections, infections due to *Salmonella choleraesuis* and *Salmonella enteritidis* occur primarily in animals. Accidental cross-contamination in man can occur and results most frequently in self-limited acute gastroenteritis. In 1941 a new *S. enteritidis* serotype '*Salmonella bonariensis*' was isolated from pig mesenteric lymph nodal tissue [1]. We report a case of *S. bonariensis* salmonellosis with acute enteritis followed by rhabdomyolysis and acute renal failure.

Mrs. L.C., 84 years old, had been previously in good health when she developed acute diarrhea on July 13, 1985. The diarrhea resolved spontaneously within 3 days, but was followed immediately by severe diffuse myalgia. The serum potassium level was 4.4 mmol/l and the serum creatinine level 500 µmol/l (1 month previously 98 µmol/l). On July 20, the patient was hospitalized with severe edema of the lower extremities and persisting diffuse myalgia. There was no gastrointestinal symptomatology. Her temperature was 37.8 °C, and the blood pressure was 130/80 mm Hg. The remainder of the physical examination, in particular the neurologic examination, was normal. The urine was clear. The electrocardiogram was normal, as was a neuromuscular biopsy. Laboratory findings revealed a hemoglobin level of 10 g/dl, a leukocyte count of 9,900/mm³ (84% polynuclear cells), and a platelet count of 277,000/mm³. The following serum levels were obtained: urea 36 mmol/l, creatinine 876 µmol/l, sodium 134 mmol/l, chloride 94, potassium 4.8, bicarbonate 19 mmol/l, creatinine phosphokinase 24,360 IU/l (normal < 100), lactate dehydrogenase 3,676 (normal < 330) serum glutamic-oxaloacetic transaminase 782 (normal < 26), serum glutamic-pyruvic transaminase 472 (normal < 27), and aldolase 7.3 IU/l (normal < 3). Three

blood cultures were negative. The urine leukocyte count was 4,000/ml, red cell count 4,800/ml, proteinuria 0.30 g/24 h, and no bacteriuria was present. Urine myoglobin levels were determined twice and were both negative. Two stool cultures obtained 24 h apart both revealed *S. bonariensis* (6,8: i: e, n, x). On July 25, the serum creatinine level was 1,266 µmol/l. The patient required hemodialysis twice. No therapy was administered for the salmonellosis, since the intestinal infection had become asymptomatic at the time of hospitalization. Upon discharge, on August 5, the serum creatinine level was 300 µmol/l, and serum enzyme levels had returned to

normal. On October 20, the patient was in good health with a serum creatinine level of 130 $\mu\text{mol/l}$.

This is the second time *S. bonariensis*, a rare serotype, has been isolated in France and the first case isolated in man [Le Minor, Centre National des Salmonelles, personal commun]. Its pathogenicity in man has never been previously reported (negative Medline search from 1971 to 1987). In this case report, this serotype induced a self-limited acute enteritis and constituted the only possible etiology for the rhabdomyolysis and acute renal failure. The diagnosis of rhabdomyolysis was established primarily by the elevated serum creatinine phosphokinase level. The absence of myoglobinuria with a normal muscle biopsy does not exclude this diagnosis [2]. There were no other traumatic, metabolic, toxic, or enzymatic causes for the rhabdomyolysis. Rhabdomyolysis with acute renal failure has been previously reported with *S. typhi* infections [3] and other *Salmonella* species [4]. Muscle energy metabolism disturbances have been demonstrated experimentally in rats infected by *Salmonella typhimurium* [5]. It is possible that this same mechanism is responsible for the rhabdomyolysis.

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References

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- 5 Friman G, Ilbäck NG, Beisel WR: Effects of *Streptococcus pneumoniae*, *Salmonella typhimurium* and *Francisella tularensis* infections on oxidative, glycolytic and lysosomal enzyme activity in red and white skeletal muscle in the rat. *Scand J Infect Dis* 1984;16:III–119.

Book Review

Graeme R.D. Catto, David A. Power *Nephrology in Clinical Practice*

Clinical Practice Series Arnold, London 1988 XI+ 324 pp.; £16.95 ISBN 0–340–41311–5

The texts' aim is to introduce renal medicine to doctors preparing for the MRCP (UK) exam, as a foundation for those intending to subsequently pursue Nephrology in more depth, as a source for students of anesthesiology or surgery and as a reference from which lectures might be prepared. It is lucidly written and, in general, readily comprehensible. Like many other texts on the subject with relatively similar objectives, there is room for improvement. Perhaps because there are so many such texts, it becomes the reviewer's task to cast a more critical eye on a new one and, additionally, view it and its mission from the standpoint of how it might compliment standard larger US textbook of Internal Medicine.

Although renal physiology is not its focus and references to it are intended only to provide background to clinical disorders, there are all aspects where a more recent acquaintance with the relevant literature is in order. Examples include an outdated presentation of sodium reabsorptive mechanisms and renal ammoniogenesis, the localization of thiazide natriuresis to the ascending limb and the omission of 'type IV in dealing with renal tubular acidosis. Elsewhere, statements are periodically inaccurate such as crenation being the feature that distinguishes glomerular from nonglomerular

hematuria or more than 10 WBCs on an uncentrifuged specimen as a definition of pyuria.

Alternately, statements requiring modification or a cautionary note include the safety of administering radiocontrast to diabetics with significant renal impairment or magnesium salts as phosphate binders to chronic renal failure patients. Oversights which this reviewer was sensitized to, perhaps reflecting his clinical bias, included omitting β -agonists from the list of causes of hypokalemia and diphosphonates from the table of treatments for hypercalcemia and the claim that hypophosphatemia is relatively rare. Better coordination would have avoided tables duplicating the causes of nephrogenic DI and the management of hyperkalemia or reference to an earlier chapter to find an unforthcoming explanation of the symbols used in calculations of free water clearance or the implication that the minimal change lesion mentioned in table 13.3 is the same as that described on page 129. The absence of hypertension and diabetes from a table listing the nine most common causes of chronic renal failure is in marked contrast to such tables in the US. The prominent inclusion of lactic acidosis and status epilepticus as causes of urate nephropathy surprised this reviewer.

The subjects which were better presented for their stated purposes than in a number of textbooks with similar objectives included the role of the kidney as an endocrine organ, renal replacement therapy, the kidney in systemic disease, preventive and rehabilitative aspects of Nephrology and financial aspects of ESRD.

Edmund Bourke Brooklyn, N.Y.

Announcement

International Meeting on Nutritional and Pharmacological Strategies in Chronic Renal Failure
Montesilvano Lido, Italy, September 29–30, 1989

This meeting will be held in the Serena Majestic Hotel and Residence, Montesilvano Lido (Pescara), Italy, on September 29–30, 1989.

The topics will include: dietary compliance in different trials in Italy

Europe and USA; malnutrition in chronic renal failure and vasoactive drugs in preventing the progression of renal damage. There will be a special session for oral presentation and posters.

For further informations please contact: Prof. A. Albertazzi

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