Concurrent emphysematous cholecystitis and emphysematous cystitis in a non-diabetic patient

Introduction

Emphysematous cholecystitis and emphysematous cystitis occurring concurrently has not been reported before. This article discusses a non-diabetic patient suffering from both these conditions concurrently with *Escherichia coli* as the causative organism – a first in the medical literature.

Discussion

This is the first reported case of emphysematous cholecystitis with concurrent emphysematous cystitis. Both these conditions are commonly seen in diabetics (Garcia-Sancho et al, 1999; Bobba et al, 2004), but this patient was not a diabetic.

Emphysematous cholecystitis typically presents with pain in the right hypochondrium with or without radiation, fever with chills and rigors, and vomiting. On the contrary, emphysematous cystitis sufferers may have non-specific features such as fever, dysuria and haematuria or they may be asymptomatic. A history of pneumaturia is highly suggestive of the diagnosis but is rarely offered by the patient (Bobba et al, 2004). Patients are usually diagnosed on imaging in both conditions.

The causative organisms in emphysematous cholecystitis include *Clostridium perfringens* and Gram-negative organisms such as *E. coli* (Bhansali et al, 2004). In addition to poor glycaemic control and an immunocompromised state, recurrent cholecystitis and the presence of gall stones predispose patients to emphysematous cholecystitis. The most common organism implicated in emphysematous cystitis is *E. coli* (Bailey, 1961) but other organisms include *Enterobacter aerogenes*, *Klebsiella pneumonia*, *Proteus mirabilis*, *Staphylococcus aureus*, streptococci and *C. perfringens* (West et al, 1981).

Gas appears in the gall bladder wall and bladder wall as a result of transluminal dis-

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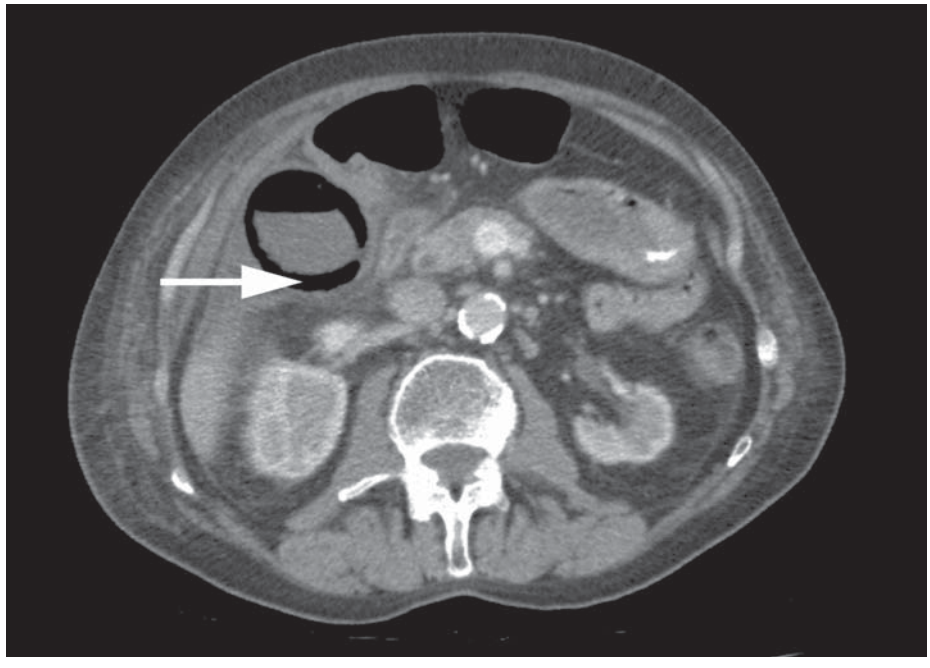
Figure 1. Air in the gall bladder wall (arrow).



Case Report

A 84-year-old non-diabetic man presented with central colicky, abdominal pain. The pain was worse on movement and radiated to his back. It began 2 weeks before admission and had progressively become worse. During this time, he had two episodes of vomiting and one episode of diarrhoea. There was no associated jaundice or weight loss. He had long-standing anaemia as a result of angiodysplasia. He did not complain of any urinary symptoms but had a previous history of chronic renal failure, hydronephrosis and transurethral resection of the prostate. On examination he had a temperature of 36.8°C, a regular pulse rate of 85/min and blood pressure of 109/95 mmHg. He had tenderness in the right para-umbilical region and an ejection systolic murmur in the aortic region without any radiation (he had a previous history of aortic valve replacement). Blood tests showed a haemoglobin of 10.7 g/dl, white cell count of 17.5×10^9/litre, urea of 11.8 mmol/litre and creatinine of 181 µmol/litre. Blood glucose on admission was 4.6 mmol/litre. Urine dipstick showed ++ leucocytes but was negative for nitrites. Urine microscopy showed the presence of $>10^5/cm^3$ of *Escherichia coli* and numerous white cells but no red cells. Blood culture was negative. Computed tomography of the abdomen showed an enlarged gall bladder with intraluminal air within the gall bladder wall and an air fluid level within the gall bladder itself (*Figure 1*). No calculi were seen within the gall bladder. Coincidentally the scan also showed air pockets within the bladder wall (*Figures 2a* and *b*).

He was treated with intravenous cefuroxime and metronidazole for 5 days as well as being fluid resuscitated. Once his temperature subsided he was discharged on a 5-day course of amoxicillin as advised by the microbiologists. A follow-up computed tomography scan showed complete resolution of the emphysematous cholecystitis and emphysematous cystitis.

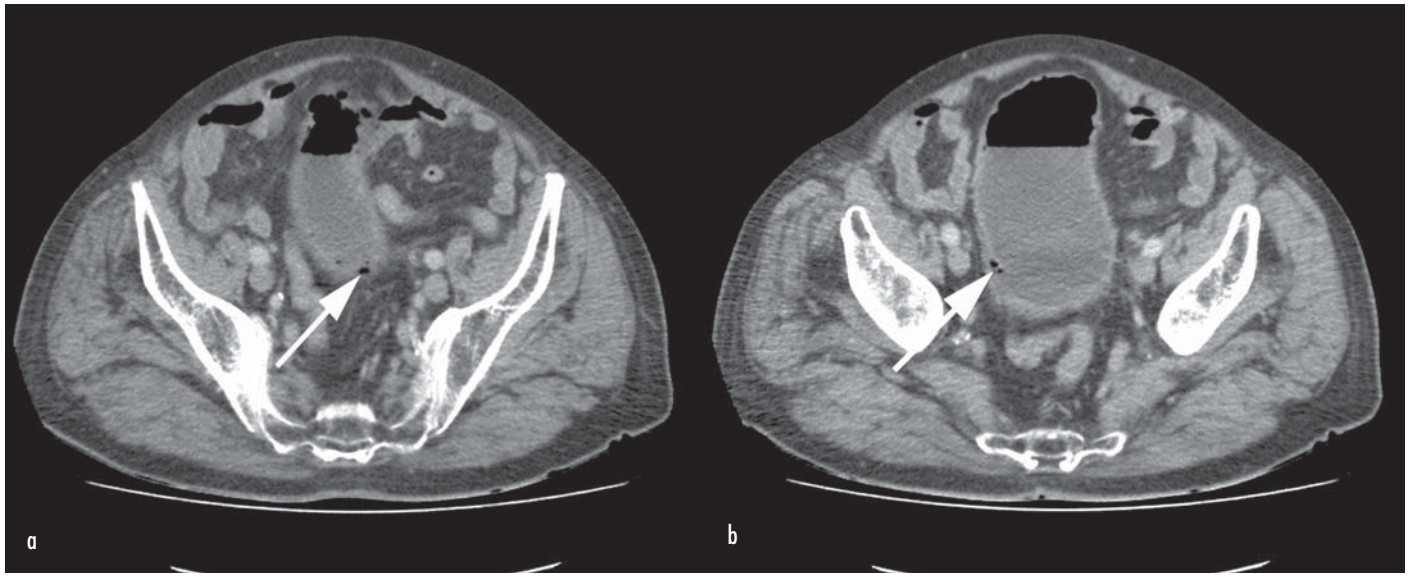


Figure 2. a and b. Air in the bladder wall (arrows).

section of gas or true infection of the bladder wall with pathogens. *E. coli* was found on urine microscopy in this patient. Facultative anaerobes, such as *E. coli*, ferment glucose to hydrogen and carbon dioxide in the low oxygen environment of the bladder, leading to gas formation. This is the most likely mechanism for the emphysematous cystitis seen in this patient. The patient had no factors which might predispose him to developing emphysematous cholecystitis. It is likely that transient bacteraemia from the *E. coli* infection in the urinary tract may have lead to dissemination of the organism via the blood stream to the gall bladder.

Treatment of both these conditions involves broad-spectrum antibiotics covering Gram-negative organisms and anaerobes, and surgery if appropriate. In the reported cases of emphysematous cholecystitis, the authors have used intravenous antibiotics for 2 weeks followed by a 6-week course of oral antibiotics in patients with acute cholecystitis and diabetes (Bhansali et al, 2004). In this case, a 5-day course of intravenous cefuroxime and metronidazole was used followed by a 5-day course of oral amoxicillin. A repeat computed tomography scan, 4 weeks after completing antibiotic therapy, showed total resolution. Infections in the presence of hyperglycaemia are more severe and complicated than those seen in euglycaemic states (Wheat, 1980). The shortened course of antibiotic therapy may have been suitable because this was a less aggressive form of the condition as this patient was not a diabetic.

Conclusions

This is the first ever reported case of concurrent emphysematous cholecystitis and emphysematous cystitis. Cases in the literature focus on patients with diabetes, but this patient was a non-diabetic. Thorough clinical assessment and early radiological assessment helps institute early antibiotic therapy and improves outcome. **BJHM**

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