A sting in the tail of Hurricane Charley

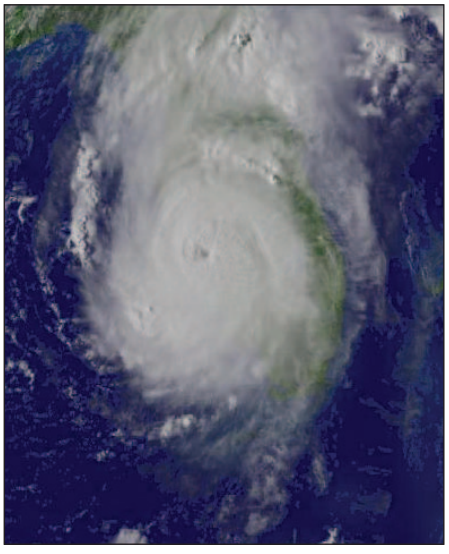

Figure 1. National Oceanic and Atmospheric Administration satellite image of Hurricane Charley over Punta Gorda in Florida, taken at 4.15pm Eastern Daylight Time on August 13 2004, after making landfall around Cayo Costa, just north of Captiva Island.

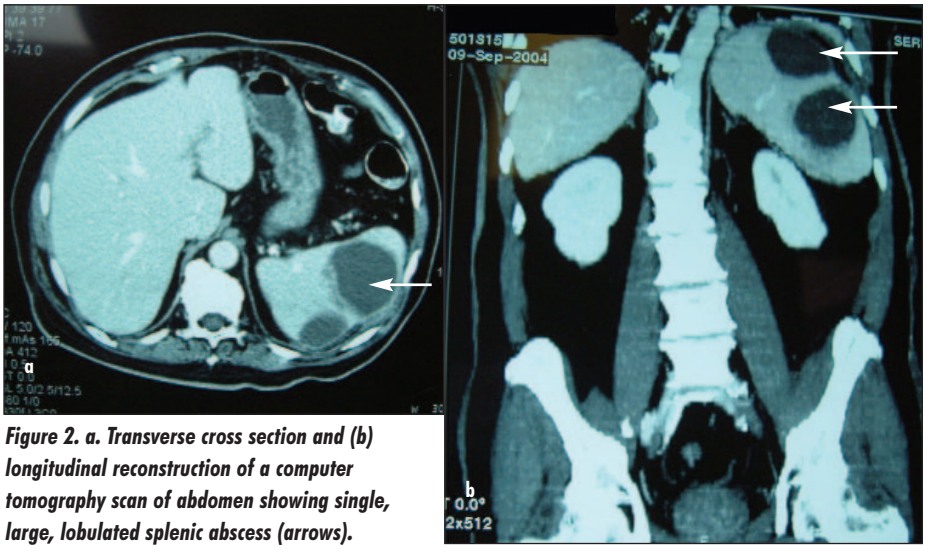

Figure 2. a. Transverse cross section and (b) longitudinal reconstruction of a computer tomography scan of abdomen showing single, large, lobulated splenic abscess (arrows).

Introduction

Category 4 Hurricane Charley made landfall at Cayo Costa, Florida, USA at 3.45pm Eastern Daylight Time on Friday 13 August 2004 (*Figure 1*). Winds were estimated at 145 mph. In the Naples area, the maximum storm tide was 10–11 feet above the mean sea level and between 4 and 8 inches of rainfall occurred along Charley's path. Moving in a north-north-east direction, Charley took 9 hours to traverse the Florida peninsula.

It was the strongest hurricane to make landfall in the state since Hurricane Andrew in 1992, with gusts topping 180 mph in Punta Gorda, north of Fort Myers – causing deaths and injuries, and destroying houses and buildings (National Oceanic and Atmospheric Administration, 2004). Besides several fatalities, several billion dollars worth of damage was caused in Florida alone, but little is recorded about the adverse, collateral medical effects of such a disaster. This case illustrates an unusual medical complication.

Case Report

A 76-year-old man with maturity onset diabetes mellitus, hypercholesterolaemia, hypertension and a history of coronary artery bypass surgery was well until his home in Punta Gorda was devastated by Hurricane Charley. He and his wife had remained in their home as it was torn apart by the ferocious wind and rain but amazingly they survived. He received numerous insect bites while searching among the flooded ruins. Within 48 hours, he became unwell, with fatigue, lethargy, myalgia, rigors and anorexia. Despite deteriorating, he did not seek medical attention until returning to the UK 3 weeks later by which time he had lost 2 stones in weight. He had received oral cephalosporin but this was not based on the evidence of infection or the result of any investigation.

On examination, he looked unwell with a sallow complexion. He was initially apyrexial. An abdominal examination revealed fullness but no palpable mass. Investigations showed leucocytosis 19.0 x 10^9/litre – 95% neutrophils. C-reactive protein (CRP) was 160 mg/litre. Abdominal ultrasound confirmed splenomegaly with a suggestion of an intrasplenic mass. Computed tomography (CT) scanning demonstrated a large multilobulated, low density mass within the spleen extending close to the capsular surface, indicative of an abscess (*Figure 2*). Five days after cessation of antibiotics, he was pyrexial (37.8°C) and blood cultures yielded Gram-negative bacillus, *Escherichia coli*. Echocardiography showed no evidence of infective endocarditis.

Percutaneous drainage of the intrasplenic abscess using a pigtail catheter revealed 500 ml of chocolate-brown coloured pus. A Gram stain showed Gram-negative bacilli and culture of the aspirate revealed *Escherichia coli* sensitive to ceftriaxone. He was treated with a single intravenous dose of gentamicin 360 mg, intravenous ceftriaxone 1 g twice daily for 2 weeks and metronidazole 500 mg three times daily for 2 weeks and the abscess resolved. He improved rapidly and 7 days after draining the abscess, the CRP fell to 13 mg/litre and the leucocyte count to 6.9 x 10^9/litre. Subsequent CT scanning showed resolution of the splenic abscess. Although genitourinary investigation revealed no abnormality, a barium enema showed a 5 mm polyp and diverticula in the sigmoid colon.

Discussion

Splenic abscess is a rare clinical entity with an incidence of 0.2–0.7% in autopsy-based studies (Chun et al, 1980; Ooi and

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Leong, 1997). It has diverse aetiologies. While it may arise from contiguous spread of infection or from direct trauma to the spleen, haematogenous spread of infection is the usual mode of development. Infective endocarditis is the most common associated infection. Alcoholics, diabetics, immunosuppressed individuals and those with haemoglobinopathies and haematological disorders are particularly susceptible.

The degree of clinical suspicion for splenic abscess needs to be high, as this condition is frequently fatal if left untreated. While approximately 50% of patients have abdominal pain, the pain is localized to the left upper quadrant in only 50% of these cases. Other symptoms include weakness (22%), chills (22%), anorexia (15%) and weight loss (11%). Splenomegaly is found in 50%. Fever (95%) and leukocytosis (70%) are generally present, fever preceding hospitalization by an average of 22 days in one series (Jang and Fung, 1995).

Left-sided chest findings may include abnormalities to auscultation, and chest radiographic findings may include an infiltrate or a left-sided pleural effusion. Ultrasonography can show hypoechoic lesions but lacks specificity and cases have been missed with this modality, whereas computed tomography (CT) scanning of the abdomen is the most sensitive diagnostic test – approaching 100% (Alonso-Cohen et al, 1990; Tikkakoski et al, 1992; Ng et al, 2002). A liver-spleen scan or a gallium scan may be useful. Abscesses are solitary and unilocular in 65%, multilocular in 8% and multiple in 35%.

Streptococcal species are the most common bacterial isolates from splenic abscesses with *Staphylococcus aureus* being the next common, reflecting infective endocarditis as the commonest cause of splenic abscess formation. An increase in Gram-negative aerobic organisms has been reported including *Escherichia coli*, *Proteus mirabilis*, group D streptococcus and *Klebsiella pneumoniae* – often originating from the urinary tract with associated bacteraemia or some other intra-abdominal source (Chun et al, 1980; Jang and Fung, 1995; Ooi and Leong, 1997; Brook and Frazier, 1998; Ng et al, 2002). Anaerobic species account for only 5% of isolates but 'sterile abscesses' may indicate that optimal techniques for isolating anaerobes were not used. Polymicrobial (36–50% of cases), fungi and rare organisms may be responsible.

If left untreated, the mortality rate from splenic abscess approaches 100%. Complications include rupture into the peritoneal cavity (6.6%), bowel, bronchus or pleural space (Ooi and Leong, 1997).

In this case, infective endocarditis and genitourinary sources were excluded by subsequent investigations. It was unlikely that the unremarkable gastrointestinal pathology was responsible for the septicaemia. The authors suspect that it probably arose from the numerous insect bites which he suffered after his property was extensively flooded by polluted water as a result of Hurricane Charley.

Because of the high mortality figures reported for splenic abscesses, splenectomy with adjunctive antibiotics is often advisable, particularly for multiple or multiloculated abscesses. However, percutaneous drainage for single abscesses is frequently successful, and ultrasound or CT guidance is particularly helpful (Jang and Fung, 1995; Ooi and Leong, 1997; Liu et al, 2000; Thanos et al, 2002). Percutaneous drainage should be considered as the first-line treatment and allows preservation of the spleen.

Conclusions

Septicaemia following numerous insect bites in the aftermath of Hurricane Charley resulted in a large splenic abscess in a 74-year-old diabetic man. CT scanning confirmed the diagnosis and intravenous antibiotics and percutaneous drainage effected a cure without any serious sequelae. **BJHM**

The authors would like to thank the National Oceanic and Atmospheric Administration (NOAA) for permission to use the satellite image of Hurricane Charley.

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