Cholecystitis and subphrenic abscess caused by *Salmonella virchow*

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**Summary**

A case of cholecystitis caused by *Salmonella virchow* is reported. Following cholecystectomy a subphrenic abscess due to the same organism developed.

**Introduction**

Infection of the gall-bladder is well described and occurs almost invariably in enteric fever caused by *Salmonella typhi*. Despite this, however, symptoms of acute cholecystitis occur in only 2% of cases (Stuart and Pullen, 1946). Systemic invasion is a much rarer sequel of infection with food poisoning *Salmonella*, and this has been associated with certain types, e.g. *S. choleraesuis*. But, if systemic invasion does occur, focal infection in almost any organ may result.

In a review of clinical syndromes associated with non-typhoidal *Salmonella* infection (Sapra and Winter, 1957) only 146 of 7779 (1.9%) cases had focal infection directly or indirectly related to the gastrointestinal tract. Non-typhoidal salmonellae are a rare cause of infective cholecystitis, being responsible for 0.6% of the cases of Fukunaga (1973) and none of those of Keighley and Graham (1973); they are an equally rare cause of subphrenic abscess (Dineen and McSherry, 1962). The authors therefore report a case of cholecystitis complicated by a subphrenic abscess caused by *S. virchow*.

**Case report**

A 36-year-old woman presented with a 3-month history of gall-stone colic. A cholecystogram confirmed the presence of gall-stones and she was admitted for routine cholecystectomy. At operation the surgeon was surprised at the degree of thickening and inflammation of the gall-bladder which was difficult to remove and adherent to the hepatic flexure of the colon. An operative cholangiogram showed no evidence of obstruction in the common bile duct, and the gall-bladder contained multiple small stones. A swab of bile was sent for culture and grew a pure growth of *S. virchow*.

On the first postoperative day the patient developed a high fever, tachycardia and became hypotensive. Blood cultures were taken and she was treated with chloramphenicol 500 mg i.v. 8 hourly, to which she failed to respond; so amoxycillin one i.v. 8 hourly with gentamicin 80 mg i.v. 8 hourly were substituted. Chest X-ray at that time showed a right pleural effusion. She improved and was discharged after 10 days on oral amoxicillin 250 mg thrice/day.

She was given another week of amoxicillin at home, because of persistent, right subcostal pain and slight fever, but 18 days later she was re-admitted. With the clinical features of a subphrenic abscess, X-rays and ultrasound confirmed the diagnosis. 800 ml of thick serous fluid was drained from her subphrenic space. The fluid was sent for culture and again a pure growth of *S. virchow* was isolated. She was therefore treated postoperatively with amoxycillin 1.5 g i.v. 6 hourly for 7 days and made an uneventful recovery.

Her stool grew *S. virchow* throughout this period, but it disappeared after drainage of the subphrenic abscess and the course of high dose i.v. amoxicillin. The blood cultures taken during the postoperative septicaemia were sterile.

**Discussion**

It is unusual for infection with a food poisoning *Salmonella* to present with cholecystitis. On further questioning the patient gave no history of previous gastroenteritis or systemic illness which might have indicated the acquisition of the organism. Whether the occult infection of the gall-bladder with *S. virchow* was the cause of her gall-stones is uncertain, but evidence now suggests a primary chemical abnormality producing precipitation of gall-stones which become secondarily infected (Keighley and Graham, 1973). The patient had been on ethynodiol diacetate BP for the past 13 years, and there is an increased incidence of gall-bladder disease in users of oral contraceptives (Boston Collaborative Drug Surveillance Programme, 1973) due to increased

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cholesterol saturation and a shift in relative proportions of bile acids (Bennion et al., 1976).

In the absence of a clinical episode of bacteraemia or gastroenteritis, the organism may have reached the gall-bladder via the bile rather than the blood stream, and its persistence in the gall-bladder facilitated by stasis and obstruction due to pre-existing gall-stones. The degree of inflammation of the gall-bladder is surprising in view of the absence of symptoms and signs of infection pre-operatively.

Despite antibiotics the organism was not eradicated and subsequently caused a subphrenic abscess requiring further surgery. In view of this the minimum inhibitory concentration (MIC) and minimum bactericidal concentration (MBC) for amoxycillin and chloramphenicol were determined on the first and second isolates, but the organism was sensitive to these antibiotics and showed no significant change in MIC or MBC.

*S. virchow* is not mentioned among the serotypes causing cholecystitis (Sapra and Winter, 1957; Fukunaga, 1973) but this may change with the recent increased incidence of infection with this organism. In 2 outbreaks of *S. virchow* infection (Semple, Turner and Lowry, 1968; Mani, Brennand and Mandal, 1974) there was a high incidence of systemic invasion with positive blood cultures, as well as gastrointestinal symptoms.

This case emphasizes the need for routine culture of bile from inflamed gall-bladders as this may represent a commoner source of occult infection and carriage than currently appreciated. Similarly, all non-lactose fermenting coliforms isolated from extra-intestinal foci of infection should be fully identified, as salmonellae have been described as a cause of focal sepsis in many sites.

**Acknowledgment**

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**References**


