

Typhoid hepatitis with pancytopenia

Sir,

We read with interest the article entitled 'Typhoid hepatitis' by S.N. Khosla.¹ We present here a case of typhoid fever with two rare features, namely hepatitis and pancytopenia (prior to treatment with chloramphenicol). The combination of hepatitis and pancytopenia in a single case of untreated typhoid has not been reported previously, to our knowledge.

A 16 year old male was admitted with a history of continuous fever and jaundice for 15 days. The patient also complained of pain in the abdomen, vomiting, and loose motions for 10 days, prior to admission. The patient did not receive chloramphenicol or any other antibiotic prior to admission. On examination, the patient was toxic, febrile, jaundiced, grossly dehydrated and had altered sensorium. Hepatic flap was present. The abdomen was mildly distended, and meteorism was present. The liver was palpable 4 cm below the right costal margin in mid-clavicular line and was soft and tender; the spleen was not palpable. His haemogram at admission revealed a total leucocyte count of $0.9 \times 10^9/l$, (neutrophils 80%, lymphocytes 20%), Hb 58 g/l, packed red cell volume (PCV) = 0.19, platelets $90 \times 10^9/l$. Peripheral smear showed normocytic, mildly hypochromic anaemia and white cells and platelets were markedly reduced, all suggesting pancytopenia. Protamine sulphate paracoagulation test was negative. Widal test was positive with antibody titres of 1:320 for *Salmonella typhi* 'O' antigen and 1:160 for 'H' antigen (Widal test repeated after 4 weeks during convalescence showed a rising titre of 1:640 for both 'O' and 'H' antigens). Blood culture was negative. Blood urea, blood glucose, serum sodium, potassium and chloride were normal. Serum total bilirubin was 111 $\mu\text{mol/l}$ (normal 3.5–20.5 $\mu\text{mol/l}$). Direct (conjugated) – 51 $\mu\text{mol/l}$ (normal 1.7–5.1 $\mu\text{mol/l}$). Serum alanine aminotransferase activity (SGPT) – 23 IU/l (normal 3–26 IU/l) and serum alkaline phosphatase – 16 KA° units (normal 5–13 KA° units).

The patient was initially put on ampicillin injection, 1 g 6-hourly (chloramphenicol was not used as the patient had pancytopenia); but as the fever pattern remained unchanged with 5 days of treatment with ampicillin, the patient was switched over to chloramphenicol injection, 500 mg 6-hourly. Forty-eight hours after, the patient became afebrile.

The haemogram repeated at that time showed total leucocyte count $5.9 \times 10^9/l$, differential leucocyte count (neutrophil 68%, eosinophil 4%, lymphocyte 23%, monocyte 5%), Hb 34 g/l, PCV 0.20, platelet count $280 \times 10^9/l$. Bleeding time and clotting time were 2 min 40 s and 3 min 10 s respectively. Liver function test showed total bilirubin 37.6 $\mu\text{mol/l}$, direct (conjugated) 18.8 $\mu\text{mol/l}$, serum alanine aminotransferase activity (SGPT) 55 IU/l, serum alkaline phosphatase 15 KA° units.

The patient's jaundice disappeared totally by the sixth day of treatment with chloramphenicol and the patient was discharged from hospital with the advice to complete a 2-week course of chloramphenicol at home.

His investigations, repeated 2 weeks after discharge, showed a normal blood count and liver function tests.

Jaundice is known to occur rarely in typhoid fever when it can have either a hepato-cellular or a cholestatic

pattern.² However, when present, it is almost always associated with a rise in serum aminotransferase levels.³ Reduction of single cell component like leucopenia or thrombocytopenia is not unknown in typhoid fever,³ but pancytopenia in an untreated case of enteric fever is perhaps extremely rare. That the pancytopenia was related to disease was proved by the fact that blood count improved with recovery of the patient after treatment with chloramphenicol. Blood culture in this patient was negative, probably because the patient came to us in the third week of illness.

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References

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Nasogastric catheter fastening technique

Sir,

Nasogastric catheterization is always an unpleasant procedure. The complaints of the patients are related to its prolonged maintenance. Nose, throat and epigastric pain are the most common complications of this procedure. The tubes also induce nausea and vomiting.

Since catheters are usually necessary in medical practice, and sometimes for a long period of time, the troublesome nature of nasogastric tubes should be decreased to be better tolerated. The way of fastening the tubes should be able to lessen the inconvenience. Not only must the catheters be firmly attached to be kept in place during the necessary time, but also they have to be accepted by the patients.

Since 1979, we have fastened nasogastric tubes to the upper lip with two adhesive tapes.¹ The first tape is attached directly to the lip. The second tape surrounds the catheter completely, and after an attachment to itself for 1 cm, this tape is attached to the first tape (Figure 1).

If the patient has a moustache, an inverted adhesive tape can be used on the opposite side of the first tape, to protect the moustache. In this case, the first tape must be attached to the face. This fastening also centralizes the tube in the nasal hole, far from the septum or walls. Even if the patient moves the head to any side, the catheter does not touch the nasal walls.

The technique presented in this communication avoids trauma to the nose. Consequently, the patients do not exhibit nasal pain, haemorrhage, ulceration or necrosis. Secondary infections, perforations and sinusitis are