Biliary Pneumonia in Malawi

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SUMMARY
A survey was made over a two and a half year period (July, 1970-December, 1972), of pneumonias complicated by jaundice. The incidence was 4 per cent. (24 cases).

The possible pathogenesis is discussed.

INTRODUCTION
In many older textbooks of medicine the association of pneumonia and jaundice (biliary pneumonia or pneumonia biliosa) is mentioned as not at all rare.
In the more recent textbooks it is only touched upon. Much forgotten pathology from the Western world is fairly frequently seen in developing countries and is therefore worth recording. We report our own observations and discuss the pathogenesis.

Patients Studied
In a 180-bed general hospital in Malawi 24 patients were observed with a biliary pneumonia during a two and a half year period (July, 1970-December, 1972).

All patients had evidence of lobar pneumonia, the evidence being based on physical examination (dull or impaired percussion, bronchial breathing, prolonged expiration). Only two patients had a chest X-ray because of lack of facilities.

The jaundice was diagnosed on clinical grounds and supported at least by urinalysis; all urines were positive for bilirubin.

A thick blood film was taken from all patients: those with a malaria positive blood film were excluded from the study. No history of alcohol abuse was elicited.

Thirteen patients had liver function studies done, mostly within two hours of admission.

Clinical Features
Apart from the classical symptoms of pneumonia the patients often complained of pain in the right upper quadrant of the abdomen; in these patients the liver was tender and palpable. One patient had a light-coloured stool.

Two female patients developed a symptomatic psychosis and three had S. mansoni infection.

The temperature was elevated for 1-8 days; the mean stay in hospital was 10 days. No patient died.

Age: There was only one child of one and a half years; all other patients were over fifteen, with an estimated mean age of 35 years.

Sex: Sixteen men, seven women and one male child were included.

Lung involvement: Six patients had more than one lobe involved; 11 left lower lobe; 10 right lower lobe; six right upper lobe; two left upper lobe and one right median lobe.

All patients did well on penicillin treatment except one who required tetracycline because of failure to respond.

Laboratory Results
Bilirubins: Total: 3,0-10,0 mgm/100 ml (mean: 5,0).
Direct: 2,3-7,8 mgm/100 ml (mean 3,5). SGOT: 23-115 units.

SGPT: 5-75 units.
SGOT values were always higher than for SGPT.

Alkaline phosphatase was always normal. The thymol turbidity tests were abnormal in four patients (8,2-21,2 units). Facilities for bacteriological examination were not available during this period.

Discussion
The incidence of biliary pneumonia in the medical department in this hospital was 4 per cent. of all pneumonias and bronchopneumonias.

Shaper and Shaper (1958) mention an incidence of 1,5 per cent. in Mulago Hospital; Hall and Parry (1963) mention 13 patients; Kibukamusoke et al (1964) described 21 patients from Kampala during a period of 15 months.

Trowell (1960) stresses its liability to appear in Africans.

Pathogenesis of Jaundice
The icterus may be caused by hepatocellular damage. Evidence has been based on biochemical tests. Zimmerman and Thomas (1950) showed that liver function is widely and almost invariably impaired in pneumonia; liver function tests returned quickly to normal after the febrile stage was over. They found abnormal bromsulfalein retention in 93 per cent. and abnormal thymol turbidity in 30 per cent. An abnormal bromsulftalein test, however, is not very reliable in febrile patients. Some authors have done liverbiopsies during the recovery phase. Hall and Parry (1963) studied eleven patients during the first week of the illness. They found cloudy swelling or necrosis of the parenchymal cells, with leucocytes in the sinusoids and portal tracts. Foci of degeneration and bile thrombi were also seen. Kibukamusoka et al (1964) studied 21 patients: changes suggestive of hepatitis were found with a marked cholestatic element. Radford and Rhodes (1967) also found hepatitis-like changes, but no cholestasis. A post-mortem liver biopsy on one patient with biliary pneumonia who was admitted after this survey and died within 24 hours, showed minimal cholestasis and nuclear variability both in the number and size of nuclei of parenchymal cells, but no necrosis. The hepatocellular damage might be due to toxic haematogenous factors (e.g. bacterial toxins). A dormant liver virus might be activated in the same way as

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the herpes virus (causing herpes labialis) with a pneumonia, as was suggested by Kibukamusoke et al. (1964). Many systemic infections produce histological and functional changes in the liver, which occasionally are accompanied by jaundice (Eley et al., 1965). In children and especially during the neonatal period a pyelitis or pyelonephritis can present with jaundice (Hamilton et al., 1963). Cellular abnormalities and bilestasis were observed in post-mortem liverbiopsies. The author assumed that the jaundice in pyelitis was due to the inability of partially injured cells to excrete bile. Fahrlander et al described electron microscopic studies of liverbiopsies in two patients, one with broncho-pneumonia, one with cystitis: a marked dilatation of bile canaliculi was seen; microvilli were reduced in number and flattened (1964).

However, the jaundice cannot be explained on the base of hepatocellular damage only. The raised transaminase levels (especially SGOT) may be due to death of lung tissue or destroyed red cells. During red hepatisation when the affected lobe is filled with polymorphonuclear leucocytes, fibrin, oedema and erythrocytes, haemoglobin becomes freed, which may give rise to an increase in the indirect serum bilirubin. Haemolysis seems to be a minor factor as there are high levels of conjugated bilirubins and the urobilinogen levels in the urines were not very high.

One wonders if the combination of pneumonia and jaundice deserves a special distinction (biliary pneumonia), as many infections are accompanied by jaundice and the mechanisms involved seem to be the same.

References


