

Possibly also at a later date the psychiatrist might become involved in these centres with group therapy for such conditions as obesity, alcoholism, tobacco addiction.

Summary

Joint psychiatric consultations over the four-year period at a general practitioners' diagnostic health centre have been reviewed—124 patients were seen, this number has been analysed as regards diagnosis and management. The value and further development of the scheme has been discussed.

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CLINICAL NOTE

Normal pregnancy in a case of active chronic hepatitis complicated by vertebral collapse

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ACTIVE CHRONIC HEPATITIS IS A FORM of relapsing hepatitis which has been variously named active juvenile cirrhosis (Read *et al.* 1963), plasma cell hepatitis (Good 1956) and lupoid hepatitis (Mackay *et al.* 1956). Active chronic hepatitis (Cook *et al.* 1968) appears to describe the condition most closely. The clinical features include recurrent jaundice, pyrexia and poly-arthritis with hepatosplenomegaly and fleeting rash. LE cells have been found in ten per cent of cases and there is a hypergammaglobulinaemia.

It is unusual for successful pregnancy to occur in patients suffering from chronic liver disease (Slater 1954). The only published reports of successful pregnancy in active chronic hepatitis that are available are those of Maclachlan *et al.* (1965), Seedat and Raine (1965), Joske *et al.* (1963), Read *et al.* (1963), and Bearn *et al.* (1956). In those cases where sufficient detail is given, the patients were receiving corticosteroids when they became pregnant and throughout pregnancy.

The present case is unusual because pregnancy was successfully completed without exacerbating the activity of the hepatitis and corticosteroid therapy, though not intensively used, resulted in vertebral collapse.

Case history

In February 1964 the patient was first seen at the age of 23 having had pyrexia for one week. She had a fleeting macular rash on the face and limbs and became jaundiced two days later. She was found to have grossly abnormal liver function tests (table I), raised gamma globulin and an E.S.R. of 112mm/hr. The latex test for rheumatoid arthritis was negative and LE cells were not found. A liver biopsy showed portal polymorphonuclear cell infiltration. In view of this, she was treated with chloramphenicol after which both pyrexia and jaundice subsided.

Four months later she again became jaundiced and pyrexial and for the first time hepatosplenomegaly was noted. LE cells were found on one occasion only, anti-nuclear factor was detected and the latex test for rheumatoid arthritis was positive. A further liver biopsy showed a plasma cell infiltration of the portal areas with cirrhosis and a diagnosis of active chronic hepatitis was made. The patient was treated with prednisolone 15mg daily from 20 September.

In October she was clinically much improved and bromsulphthalein retention was 19 per cent after 45 minutes, having been 35 per cent before prednisolone. She was maintained on 15mg prednisolone daily.

She had amenorrhoea since the start of her illness in February 1964 and in February 1965

TABLE I
TABLE OF LIVER FUNCTION TESTS

Date	Serum bilirubin mg/100ml	Serum alkaline phosphatase K-A unit	Thymol turbidity	SGOT sigma-Frankel units	SGTP sigma-Frankel units	Serum albumin G/100ml	Serum globulin G/100ml
*Feb. 12 1964 ..	2.3	68.8	18	175	180	3.4	4.1
*Feb. 28 1964 ..	1.3	80.5	18	116	96	3.3	4.1
*Mar. 10 1964 ..	2.0	85	16	230	370	3.7	5.6
*July 27 1964 ..	2.2	47.5	9	136	160	4.0	4.6
*Aug. 15 1964 ..	1.2	74.8	16			3.2	6.3
*Aug. 25 1964 ..	1.2	75.6	18	810	680		
*Sept. 2 1964 ..	2.3	70.4	18	830	530	4.5	5.5
Nov. 19 1964 ..	0.7	49.8	9	360	320	3.9	4.2
Dec. 14 1964 ..	0.5	23				3.3	4.6
June 11 1965 ..	1.3	35	8	180	280	3.6	3.2
July 21 1965 ..	0.9	81	6		142		
Dec. 13 1965 ..	7.5	38.7	14	730	660		
June 2 1966 ..	1.2	48.3	10	460	450	4.1	4.5
April 20 1967 ..	1.8	60.7	10	400	240		
Nov. 5 1967 ..	1.2	28	3	288	152		
Jan. 18 1968 ..	1.2	24.2	8	90	62		
April 25 1968 ..	1.9	27		152	83		

*=L.F.T.s before treatment with prednisolone

she was found to be four months pregnant. Conception must have occurred about one month after starting corticosteroid therapy.

In April 1965 she was rested because of increasing back pain. Serum calcium was 9.2mg/100ml and a 24-hour urine calcium was 0.3G. An x-ray taken in January had shown only a mild vertebral osteoporosis (figure 1a). She was not jaundiced and liver function was relatively normal.

Labour was induced on 16 July and a normal male baby was born. The labour was normal except for an increase in the backache. An x-ray of the spine taken after delivery showed multiple wedging of the lumbar and thoracic vertebral bodies (figure 1b). A Roberts-Jones corset was supplied and the patient given calciferol, potassium chloride supplement and a reducing dose of prednisolone. By November, prednisolone had finally been withdrawn.

In December 1965, she developed a severe relapse of the hepatitis and was started on ACTH gel 40 units and hydrocortisone 100mg im twice daily. After three days, treatment was continued with ACTH gel 40 units on alternate days. She rapidly improved (figure 2) and this dose was reduced slowly over a period of 14 months to be discontinued in February 1967. Up to the time of this report she has had no treatment for a year and a half. She has remained well, though mildly icteric, with occasional short attacks of fever and increasing jaundice. She is over four inches shorter than before her illness.

Discussion

In most cases of active chronic hepatitis previously described, deterioration occurred during pregnancy (Joske *et al.* 1963, Read *et al.* 1963, Seedat and Raine 1965) although some had little or no deterioration (Bearn *et al.* 1956, Jackson 1962, Maclachlan *et al.* 1965). In the present case the hepatitis remained completely quiescent. There is no guide as to which case will

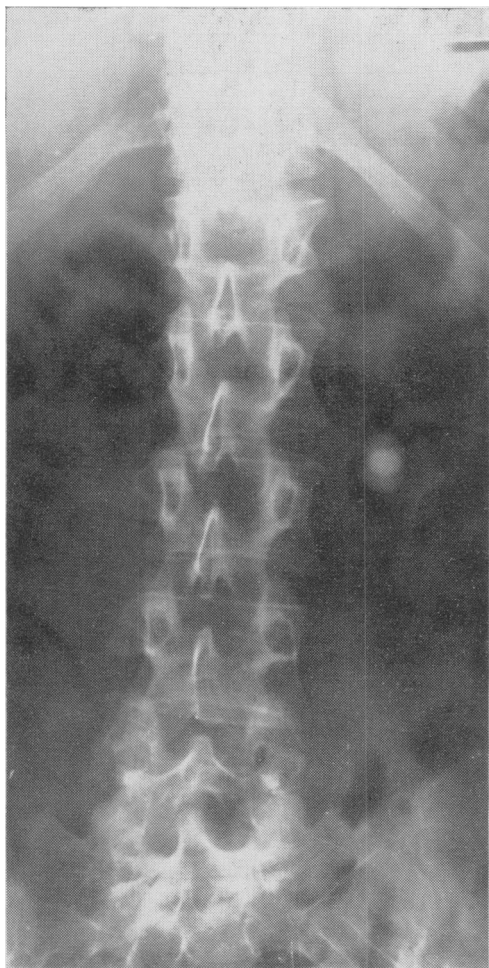


Figure 1a
Showing mild vertebral osteoporosis



Figure 1b
Showing multiple wedging of lumbar and thoracic vertebral bodies

relapse and a survey of cases previously described indicates that relapse is not related to the severity of the hepatic condition nor to the dose of corticosteroids controlling it.

Another unusual feature was the development of multiple vertebral collapse even though the patient had been treated with a comparatively low dose of corticosteroid for only a short period. Other factors may have been associated with corticosteroid treatment in bringing about this rapid vertebral degeneration. The heavy protein requirement of pregnancy may have been a contributing factor and in the present case serum protein fell during the pregnancy from 8.1G/100ml in October 1964 to 6.8G/100ml in July 1965. A progressive fall of serum protein during pregnancy in active chronic hepatitis has been reported in the case described by Seedat and Raine (1965). Another factor may have been the increased mobility and excretion of calcium which could follow a co-existing metabolic acidosis. This is suggested in this case in that the serum bicarbonate levels ranged from 14.1 to 21mEq/L. and a 24-hour urine calcium was 0.3G, the upper limit of normal. There was also a persistent hypokalaemia (average 3.3mEq/L).

The patient has remained fairly well without medication for the last 18 months under observation. However, evidence of active hepato-cellular damage persists clinically, with

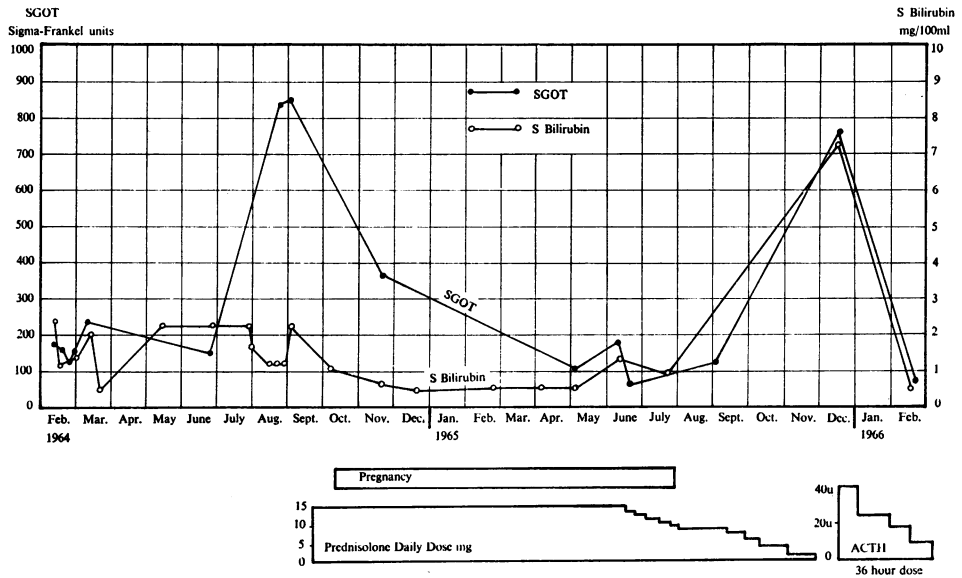


Figure 2
S. Bilirubin and SGOT in relation to treatment and pregnancy

recurrent attacks of pyrexia and jaundice, and serologically with serum bilirubin 1.9mg/100m and SGOT 152u (April 1968). The prognosis now that the patient has survived four-and-a-half years of active disease is somewhat better than at the onset. In the series of Read *et al.* (1963), of 26 patients who died, 17 did so in the first four years. Long term survival is most commonly seen in women and some are alive and well over ten years after diagnosis.

Summary

A case of active chronic hepatitis is described in which the primary illness remained quiescent during pregnancy unlike most others reported where pregnancy led to a deterioration. Multiple vertebral collapse developed despite comparatively low dose corticosteroid therapy during pregnancy. The possible reasons are discussed.

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