JAUNDICE IN PREGNANCY

(A 13 Year Review)

by

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Introduction

Jaundice in pregnancy, though rare, is a dreaded complication. However, during the last decade newer and changing concepts of hepatic diseases have evolved newer investigative techniques which could be applied with advantage to this dreaded complication in pregnancy, thus reducing both morbidity and mortality.

With this in view, a retrospective study was undertaken over a period of 13 years

TABLE I

Published Causes of Jaundice in Pregnancy

Diagnosis	Author					
	Thorling	Sheehan's survivors	Sheehan's autopsy	Singler-Keyser	Per cent*	Present series
No. of patients	72	36	50	23	—	35
Recurrent Jaundice of pregnancy	38	19	0	1	44	Nil
Viral hepatitis	26	7	2	10	34	30
Drugs	0	5	10	3	6	? 1 Criminal abortion
Obstructive Jaundice	—	3	11	2	4	—
Hemolytic anaemia	2	1	9	1	3	? 1 Blood transfusion
Infection	—	0	—	2	3	1 Septicaemia
Hyperemesis	6	—	3	2	—	? 1
Obstetric acute fatty liver	—	1	8	0	—	? 1 Had severe pet
Frequency of Jaundice	—	1 in 2000 or 3000 deliveries	—	1 in 3500 deliveries	—	1 in 1063 pregnancies

* Sheehan's autopsy experience is excluded in calculating the per cent.

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Received for publication on 1.4.74.

(1960-1972) of such cases admitted into the maternity wards of Christian Medical College Hospital, Vellore.

Observation

There were 35 such cases over the 13 year period under review, out of a total of 37,219 obstetrical inpatients giving an incidence of 1:1063.

The pathology leading to jaundice in these 35 cases is shown in Table I. The commonest pathology was due to infective or viral hepatitis.

Table II shows the incidence of jaundice in relation to the gestational period. The incidence appears to be commonest during the third trimester of pregnancy (53.9 per cent). This confirms the observation of Bhasker Rao and Ganpathy (1955) who analysed such cases during an epidemic of infective hepatitis.

TABLE II

Incilence of Jaundice According to Duration of Pregnancy

Duration of pregnancy	Number of patients		
	Total	Fatal	Non-fatal
First trimester	1 (2.9%)	—	1
Second trimester	7 (20.6%)	—	7
Third trimester	19 (53.9%)	4	15
Post-partum	8 (22.6%)	3	5
Total	35 (100.0%)	7	28

Table III shows the presenting clinical features observed in these cases under review. Jaundice was present in all cases. The clinical features attributable to the systemic, gastrointestinal and abdominal system appeared to be the commonest. Clinical features attributable to haemorrhagic disorders, cerebral and renal system were not so common.

TABLE III

Clinical Features of Jaundice in Pregnancy

Clinical features		No. of cases	Percentage
Systemic			
Jaundice	—	34	100.0
Pyrexia	—	12	35.3
Oedema	—	8	23.5
Pruritis	—	4	11.8
Gastro-intestinal			
Nausea	—	8	23.5
Vomiting	—	5	14.7
Anorexia	—	7	20.6
Diarrhoea	—	3	8.8
Abdominal			
Hepatomegaly	—	9	26.5
Abdominal pain	—	6	17.6
Abdominal distension	—	3	8.8
Bleeding tendency			
Bleeding gums	—	2	5.9
Petechial heamorrhages		1	2.9
Epistaxis	—	2	5.9
Antepartum haemorrhages	—	1	2.9
Post-partum haemorrhages	—	6	17.6
Cerebral			
Convulsions	—	1	2.9
Coma	—	7	20.6
Renal			
Oliguria	—	3	8.8

Though the diagnostic clinical manifestations were present in all the cases, the associated symptomatology varied in some cases making a definitive diagnosis of the pathogenesis without biochemical investigations well nigh impossible.

Liver function was investigated in all the cases. Table IV shows the values of the other diagnostic liver function tests matched against the period of gestation. Closer scrutiny of these tests clearly demonstrated that there was a certain degree of hepatocellular damage. The extent of this damage appears to be more with the increase in gestational period.

The high incidence of maternal mortality in the third trimester and during postpartum, is obvious from Table V which shows maternal prognosis in relation to jaundice in pregnancy.

Table VI shows the fetal prognosis which demonstrates high fetal loss associated with premature labour as well as still births.

TABLE IV

Mean Values of Liver Function Tests and Gestation Period

Gestation period in weeks	Alkaline phosphatase K.A. units/100 ml.	Transaminase		Serum proteins	
		SGOT	SGPT	Albumin	Globulin
Less than 20	13.00	12.77	101.33	3.320	2.430
21–25	20.50	450.00	540.00	3.20	2.30
26–30	22.40	430.00	520.00	3.50	1.95
31–35	32.00	388.00	420.20	3.35	2.00
36–40	31.00	722.67	612.50	2.38	2.88

TABLE V

Jaundice in Pregnancy and Maternal Prognosis

Duration of pregnancy	Total No. of cases	Delivered		Undelivered	
		Fatal	Non-fatal	Fatal	Non-fatal
Ist Trimester	1	—	1	—	—
IInd Trimester	7	—	1	—	6
IIIrd Trimester	19	3	14	1	1
Post partum	8	3	5	—	—
Total	35	6	21	1	7

x^2 Test of significance showed that the difference in the total percentage of fatal cases with or without delivery is not significant.

TABLE VI

Jaundice in Pregnancy and Fetal Prognosis

Type of labour	Total	Still birth	Neonatal death	Alive
Premature labour	19 100%	12 63.2%	2 10.5%	5 26.3%
Labour at term	16 100%	6 30.5%	1 6.2%	9 56.3%

The difference in the percentage of still birth and neonatal death between the premature cases and labour at term is not significant.

Table VII shows the analysis of the clinical features and biochemical investigations in the 7 patients who expired. The most significant common feature is the high values of transaminases, denoting extensive hepato-cellular dysfunction. There is also an associated blood coagulation and renal failure.

Table VIII shows the analysis of one case with similar clinical features and biochemical liver function profile, as in these expired cases. She had two exchange transfusions and recovered.

Discussion

Jaundice in pregnancy is a rare condition as observed from the incidence here and abroad. From national literature, the

TABLE VII

Analysis of Patients who Expired

No.	Age	Gravida	Week of Gestn.	Type of delivery	Associated conditions	Bleeding tendency	SGOT KA usits	SGPT KA units	Bilirubin mg%	Prothrombin time
1	25	G4	28	Premature still birth	Uraemia convulsions	Epistaxis	1420	2500	16.6	60'-13"
2	21	G2	36	Normal asphyxiated baby neonatal death	Pyrexia abdominal distension hepatomegaly	Bleeding gums petechial heamorrhages	1230	1130	9.0	Bleeding time 15'
3	28	G7	35	Undelivered	Oedema hepatomegaly pyrexia		1460	2200	17.0	26'-14"
4	29	G7	28	Premature labour still born	Coma	Post partum haemorrhage	1300	1850	17.1	44'-12"
5	22	G3	32	Still born	Coma oedema pyrexia	Epistaxis	1450	2600	13.0	46'-10"
6	24	G4	40	Twins prolonged labour post partum shock one still birth	Severe pet coma		740	225	10.8	20'-14"
7	20	G1	34	Premature dead born	Eclampsia haematuria	Post partum haemorrhage shock	1430	2600	16.6	26'-14"

TABLE VIII

Analysis of Patient Data Treated with Exchange Transfusion

No.	Age	Gravida	Week gestn.	Type of delivery	Associated conditions	Bleeding tendency	SGOT KA units	SGPT KA units	Bilirubin mg%	Prothrombin time
1	28	G6	30	Premature dead born	Coma oliguria	Nil	1420	2340	30.0	—

MANAGEMENT
4 L Blood
2 Exchanges required

incidence appears to be high ranging from 1 in 50 (Malkani and Grewal, 1957), 1 in 67 (Rao *et al*, 1969) to 1 in 386 (Bhasker Rao *et al*, 1955). These incidences were most probably associated with epidemics of viral hepatitis and therefore would not represent a true incidence. The incidence reported from this hospital is 1 in 1063 of obstetrics admissions and this incidence probably represents a truer picture.

From Table I, it can be observed that the commonest pathology responsible for syndrome of jaundice in pregnancy is viral hepatitis and this is probably the single important cause for most of the jaundice in pregnancy. This observation substantiates previous such reports (Sheehan, 1961).

Jaundice in pregnancy appears to be most common from our data in the third trimester and this substantiates a similar observation made by Rao *et al* (1969). This could be directly attributable to the impairment of the liver to excrete the conjugated bilirubin in the third trimester (Frank *et al*. 1965; Hsia *et al*, 1960; Hsia *et al*, 1963).

The cause of jaundice is multifold, but is basically one of the three viz pre-hepatic, hepatic or obstructive. The first two are sinister and carry with them a high mortality and/or morbidity. The diagnosis is further rendered difficult by the multiplicity of liver function. The assortment of clinical signs often makes the diagnosis difficult, particularly in the presence of other complications of pregnancy. Clinical signs though of great importance have their own limitations and ultimately a definitive diagnosis to enable proper management is made on laboratory investigations. From the many tests available, a judicious choice should be made to constitute a "liver profile" to determine hepatic excretory function, extent of hepatic parenchymal damage and certain specific hepatic metabolic functions. The basis of diagnosis employed in this series is a measurement of direct and indirect bilurubin, serum alkaline phosphatase, serum proteins and their ratio, and the estimation of serum transaminases. This gives a good profile to arrive at a definite diagnosis. Plasma prothrombin time is helpful in the management of such cases.

From this profile it can be seen that the maximum hepatocellular damage occurs from 21 weeks of gestation onwards as evidenced by the high values of the transaminases obtained (Table VII).

Considering the problem of this syndrome in relation to maternal prognosis a clinical consideration leads to the hypothesis that maternal mortality is higher

in those cases where termination of pregnancy has occurred. However, when chi square test of significance is applied there is no statistical significance to such an observation. It can be seen that the maximum mortality occurs in the third trimester and in the postpartum period.

Similarly, the fetal prognosis is poor when patients develop jaundice in pregnancy. There is a high rate of premature labour with high fetal loss.

Analysis of the 7 deaths that occurred clearly shows certain facts which may be of some prognostic value. The clinical symptoms grossly reflect the state of hepatic dysfunction. The patients were all multigravidae leading to a conjecture whether repeated pregnancies take a toll on hepatic function. These points could therefore be taken in the ultimate analysis of the prognosis of the case. The serum transaminases are very highly elevated and are also associated with very high levels of serum bilirubin. Further disturbances of blood coagulation and renal failure are also prominent.

The most recent case had all the above mentioned criteria as is shown in Table VIII. This patient was given an exchange transfusion on two occassions with a total exchange of 4 litres and 2 litres of fresh blood. She made a steady progress after this and recovered completely. This mode of management perhaps may be the management of choice in such cases in the future.

Summary

A 13 year review was undertaken wherein 35 cases of jaundice in pregnancy were studied. The analysis of these cases indicated the need for biochemical analysis to give a profile of liver functions in the management of jaundice in pregnancy. The management has been mainly conservative, without any obstetrical interference. The analysis of the cases wherein death occurred showed severe hepato-cellular damage. One case with similar findings could be salvaged with exchange transfusion.

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