Table showing response of 8 cases of N.M.A. to the different liver extracts

Number	Initial rbc in million	Initial Hb. in gm.	Final rbc in million	Final Hb. in gm.	Days	Dosage	Remarks
I	2.2	8.65	3.76	11.76	14	2 c.c. × 6 days Plexan (12 c.c.)	Optimum response.
II	1.46	5.19	3.4	9.51	19	2 c.c. × 6 days Plexan (12 c.c.)	Optimum response.
III	0.88	3.70	2.32	7.26	16	1 c.c. × 8 days Chemilon (8 c.c.)	Optimum response.
IV	1.08	3.76	2.2	7.09	22	1 c.c. × 8 days Chemilon (8 c.c.)	Suboptimal response.
V	1.00	3.76	3.76	10.38	16	1 c.c. × 3 days Examen (3 c.c.)	Optimum response.
VI	1.84	8.40	3.72	12.11	21	1 c.c. × 3 days Examen (3 c.c.)	Optimum response.
VII	1.09	4.84	2.24	7.78	14	1 c.c. × 2 days Examen (2 c.c.)	Slightly less than optimal response.
VIII	1.82	7.95	3.08	10.38	17	Examen (1 c.c.)	Slightly less than optimal response.

Since Wills' work, crude liver extract was considered effective in the treatment of N.M.A. and purified liver extracts were presumed to lose the anti-anæmic principle which is supposed to be curative in the N.M.A. in the process of purification. Since then, however, it has been found that purified liver preparations are effective, but in larger dosage, than that for the P.A. The four cases quoted above show that purified liver extract Examen (New Potency) is also effective even in a small dose, 1 c.c., though it can be said that 3 c.c. produce better response than 1 or 2 c.c. Since the comparatively small dosage of refined liver extract used has produced optimum response, the cases in question seem to point the way to further investigations on similar lines.

The dosage of crude liver extract employed in the treatment of N.M.A. has been usually 2 to 4 c.c. daily (and even a bigger dosage for the first 2 or 3 days) for above 10 days, that is a total of 20 to 40 c.c. In the 4 cases of N.M.A. treated with crude liver extract the dosage was only 8 to 12 c.c. So far it has been the smallest dose found effective in our experiments.

Summary

1. The activity of three different types of liver extracts was investigated in cases of nutritional macrocytic anæmia.

2. It was found that pure liver extract (Examen, New Potency) gave optimum response in this anæmia; even small doses proved effective.

I take this opportunity of thanking the Dean, K. E. M. Hospital, and Honorary Director, Singhanee Hospital, for allowing me to report these cases. I also express my acknowledgment to the firms concerned for generously supplying me with liver extracts.

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STUDY OF LIVER DISEASES

CORRELATION OF CLINICAL AND LIVER FUNCTION STUDIES WITH LIVER BIOPSY

By P. N. WAHI, M.D., M.R.C.P. (Lond.)
and
M. G. CHAKRAVARTI, D.I.C., M.Sc., Ph.D. (Lond.)
Departments of Pathology and Biochemistry, Agra Medical College

In cases of hepatic diseases it is at times difficult to arrive at a correct diagnosis from clinical findings only. To help the physician a number of laboratory tests have been devised. They include (*i*) the study of biopsy material, (*ii*) routine examination of blood and urine, and (*iii*) 'function tests' either to evaluate the impairment of the hepatic function or to study the derangement of some specific function of the liver. Such studies are necessary in cases where clinical signs like jaundice or ascites are present; and also in those where such signs are absent. It must be admitted that the liver 'function tests' have limited usefulness. The

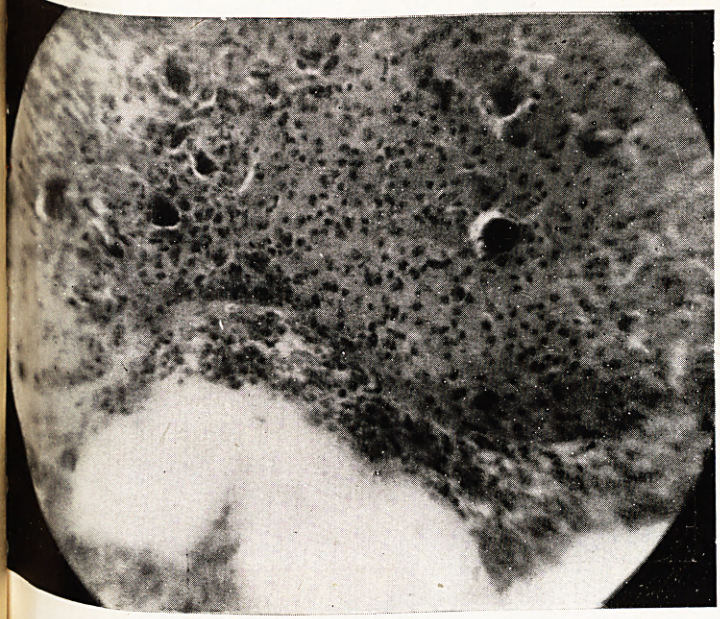

Fig. 1.—*Case I.*

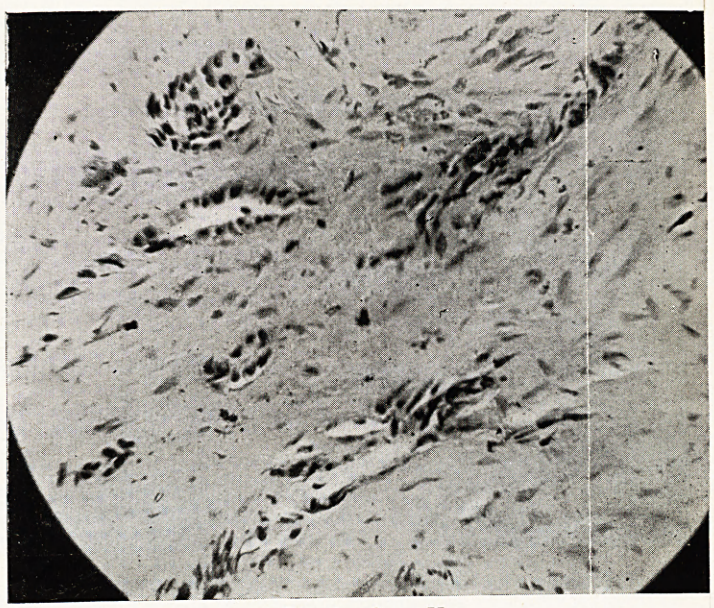

Fig. 2.—*Case II.*

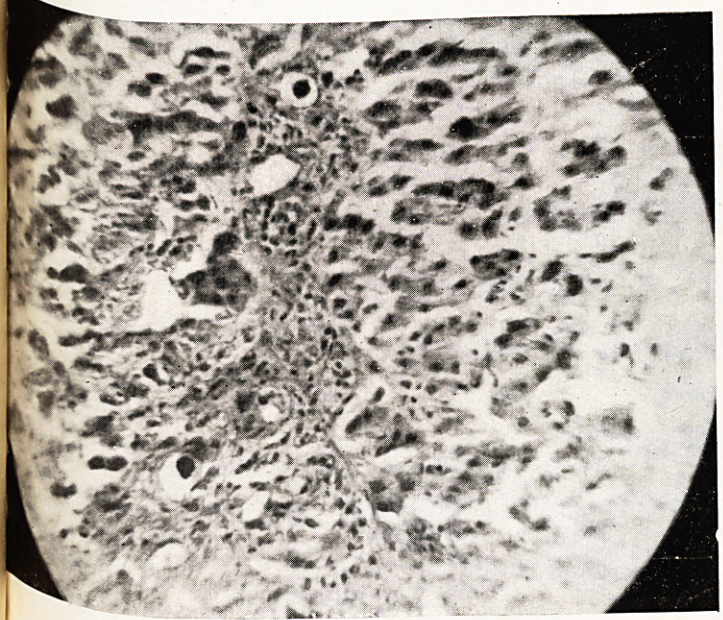

Fig. 3.—*Case III.*

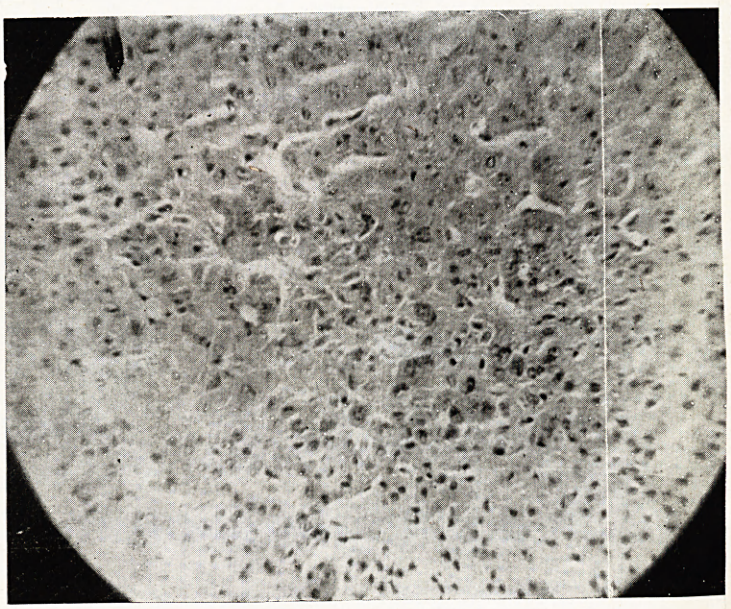

Fig. 4.—*Case IV.*

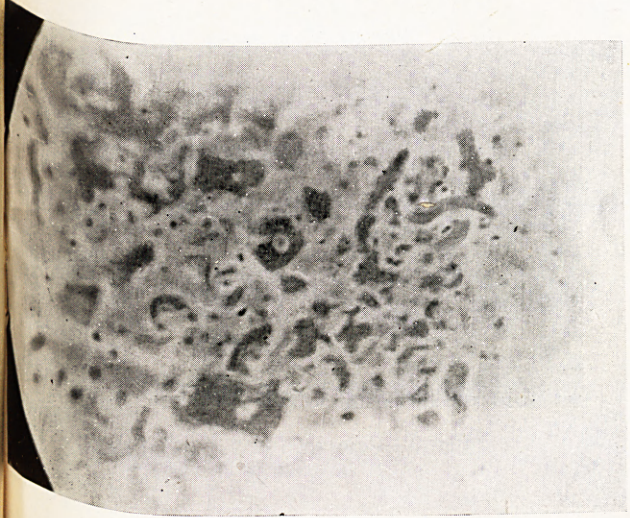

Fig. 5.—*Case V.*

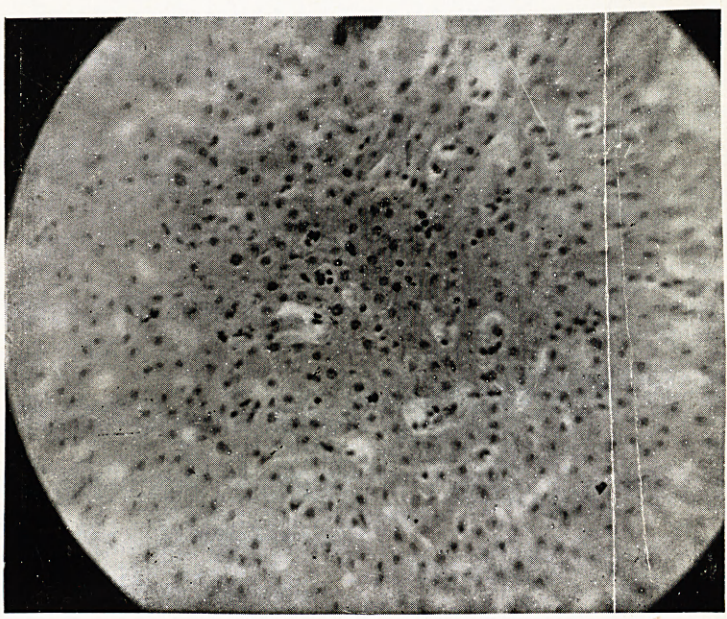

Fig. 6.—*Case VI.*

STUDY OF LIVER DISEASES : P. N. WAHI & M. G. CHAKRAVARTI. (O. A.) PAGE 454

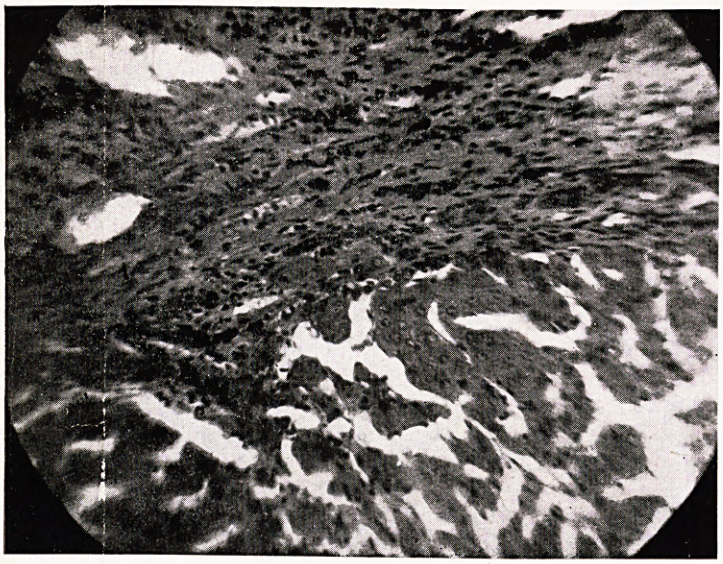

Fig. 7.—*Case VII.*

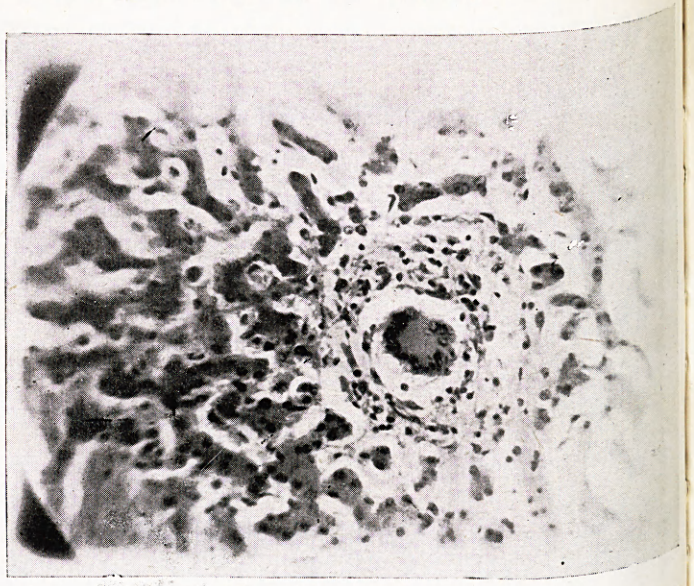

Fig. 8.—*Case VIII.*

MYOEPITHELIAL TUMOURS OF THE SALIVARY GLANDS : M. V. SIRSAT. (O. A.) PAGE 460

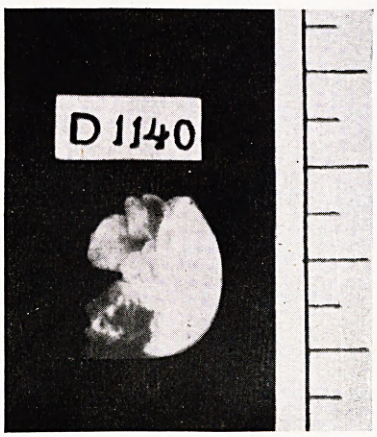

Fig. 1.—*Case* 1.

Fig. 2.—*Case* 1.

Fig. 3.—*Case* 2.

possibility of impairment and the extent of derangement of the multiple functions of the liver are very large. No test is yet known which can be of a general diagnostic use. Till such test becomes available, which seems doubtful, the physician has to depend on the existent function tests of the liver, and choose therefrom those of value in particular cases. A second approach open to him will be to correlate the information available from the biopsy studies with the chemical tests.

We have been interested for some time in correlating the histology of the diseased liver with the tests of hepatic insufficiency. Studies of this type have been made by Hoffbauer, Evans and Watson (1945) in cases of cirrhosis of the liver.

In this paper, cases have been recorded where a definite histological diagnosis has been made from biopsy studies. In some, the necropsy material has also been employed.

Some of the 'function tests' performed are common both in the non-jaundiced and jaundiced cases. A few others have been taken up in jaundiced cases only. In non-jaundiced cases the tests performed were icteric index, quantitative serum bilirubin, quantitative urobilinogen, total and fractional serum proteins, hippuric acid synthesis, prothrombin time and response before and after administration of vitamin K, blood urea and non-protein nitrogen. In jaundiced cases, all these and fæcal urobilinogen, serum cholesterol, van den Bergh's reaction and galactose tolerance test have been performed. The tests chosen were of general usefulness. The tests which are cumbersome to perform, have limited application and cause inconvenience to patients have been omitted. For our preliminary work cephalin-cholesterol flocculation test, bromosulphalein test and Takata-Ara test were performed. From them very little additional information was available, and as such they were abandoned at a later stage in our work.

The techniques employed are those commonly used in clinical laboratories. The estimations of total and fractional proteins have been made by the microkjeldahl method before and after precipitation with 22 per cent sodium sulphate. The blood urea has been determined by the urease-nesslerization method. The hippuric acid synthesis test has been performed by the intravenous method of Quick. The impairment of renal function, if any, was evaluated side by side by an injection of 1 c.c. of phenolphthalein and by determining the dye excretion after one hour; and also by the van Slyke's urea clearance test. For the determination of prothrombin time, one stage modified method of Quick has been employed. The prothrombin response was measured after an administration of 5 or 10 mg. of vitamin K parenterally. For urobilinogen the method of Wallace and Diamond has been employed.

Liver biopsy was performed with the Vim-Silverman needle. The technique has already been described in detail by one of us (Wahi, 1946).

Illustrative cases

Case I. Hindu female, aged 60 years, was admitted into the Thomason Hospital with the complaint of a painful lump in the right lumbar region of about two months' duration. There was a marked loss of weight, and signs of emaciation were present. The patient had jaundice, but there was no ascites or œdema. The liver was found enlarged, about four fingers below the costal margin, and was hard to feel. The pelvic examination revealed no abnormality. The patient's past history showed little of note. The working diagnosis of cancer of the liver was made.

The function tests performed in this case gave the following results : The stools were light yellow in colour and urobilinogen, as repeated examinations showed, was completely absent. Urine urobilinogen was 2 mg. The prothrombin concentration was 80 per cent, which, after an administration of 5 mg. of vitamin K, showed a rise to 88 per cent after 24 hours. The van den Bergh's reaction was immediate direct positive ; serum bilirubin 3 mg.; icteric index was 35; blood cholesterol was 333.0 mg.|100 c.c. and galactose 20 mg. ; 0.25 gm. of hippuric acid was synthesized and the plasma proteins were found to be altered.

The function tests, by themselves, as can be seen from the data, could lead to no positive conclusion, some pointing towards hepatic lesions and others suggesting normal liver function. The photomicrograph of the needle biopsy (figure 1, plate XXV) shows no evidence of tumour. The canaliculi are dilated and contain bile thrombi. The liver cells show cloudy swelling. This suggests the diagnosis of extrahepatic biliary obstruction.

A detailed analysis of the case, therefore, shows that clinically the case labelled as cancer of the liver, when subjected to function tests, gave findings supporting the diagnosis either of cancer of the liver or extrahepatic biliary obstruction. The information obtained from the liver biopsy was of great value, and the case was labelled as one of extrahepatic obstruction.

Case II. T. S., Hindu male, aged 42 years, was admitted with a painful lump in the right hypochondrium of about three months' duration. He had jaundice four months before admission and had suffered from irregular fever during this period. A fortnight before admission he developed œdema of the feet. He had been a habitual alcoholic.

The examination revealed a poorly nourished, deeply jaundiced, slightly anæmic, middle-aged man. The liver was found to be irregularly enlarged from ½ to 4 inches below the costal margin and was hard and slightly tender. Ascites was present and the spleen was not

palpable. After a few days' stay in the hospital there was a noticeable increase both in ascites and jaundice. The working diagnosis of cancer of the liver was made.

The prothrombin concentration was 70 per cent and was raised to 80 per cent after vitamin K administration; icteric index was 90; van den Bergh's reaction was immediate direct positive and serum bilirubin was 5.6 mg.; W.R. was negative and Takata-Ara test was positive; galactose was 40 mg. Blood urea was 35.6 mg; N.P.N. 32 mg.; total protein 4.8 per cent ; serum albumin 2.9 grammes and globulin 1.9 grammes. The low plasma proteins and slightly altered A : G ratio indicated liver damage. Blood cholesterol was raised to 266.6 mg.|100 c.c.; fæcal urobilinogen was absent and hippuric acid synthesis was 0.42 gramme.

The liver function tests suggested the diagnosis of obstructive jaundice and liver damage and are quite in keeping with the clinical diagnosis of cancer of the liver.

The needle biopsy study of the liver (figure 2, plate XXV) revealed the presence of cancer and confirmed the diagnosis.

Case III. O. P., Hindu male, aged 23 years, was admitted with ascites of short duration. Five years back he suffered from fever with rigor which was controlled by quinine after a fortnight. He had also been getting epigastric pain.

The examination revealed a poorly nourished anæmic young man with no jaundice. He had ascites. The liver and the spleen were not palpable. The clinical diagnosis of endemic ascites was made.

Laboratory investigations showed: W.B.C. 5,000 per c.mm.; P—66; L—32; M—2; E.S.R. 14 mm.; van den Bergh's reaction was negative, icteric index 10 and serum bilirubin 0.35 mg. ; prothrombin concentration of 66.6 per cent was raised to 80 per cent after administration of vitamin K. Takata-Ara test was positive. Blood urea was 34.2 mg.; N.P.N. 31 mg.; total proteins 5.87 grammes; serum albumin 3.55 grammes and globulin 2.32 grammes. Urine examination revealed the presence of urobilinogen and synthesis of 0.28 gramme of hippuric acid. Fæcal urobilinogen was 100 mg. Examination of ascitic fluid showed cell count 37 per c.cm. which were all lymphocytes. Total proteins in ascitic fluid were 1.4 grammes.

Analysis of the findings does not lead one to any definite conclusion. The prothrombin concentration was low but showed a good response to vitamin K. Hippuric acid synthesis was very low and was the only finding which pointed towards liver damage.

The needle biopsy was done through the 10th intercostal route. The histological examination showed that the liver cells were markedly pigmented. The bile canaliculi were dilated and contained bile thrombi. The portal connective tissue was greately increased and cellular. Newly formed bile ducts were present (figure 3, plate XXV). Diagnosis : Cirrhosis of liver with jaundice.

The diagnosis of this case by needle biopsy was thus of great help as the case was believed to be one of endemic ascites. This also points to the fact that many of the so-called cases of endemic ascites if histologically examined may turn out to be those of cirrhosis of the liver.

Case IV. C., Hindu female, aged 45 years, was admitted into the Thomason Hospital with complaints of fever, cough with expectoration and constipation. There was history of previous attacks of malaria. The examination showed a fairly nourished woman with jaundice. The spleen was enlarged 5 inches and was hard and smooth. The liver was 5 inches below the costal margin, was hard and smooth. The working diagnosis of chronic malaria and cirrhosis of the liver with jaundice was made.

The laboratory investigations showed that the prothrombin concentration of 84.2 per cent was raised to 89 per cent after an administration of 10 mg. of vitamin K; icteric index was 17 ; van den Bergh's reaction indirect positive and serum bilirubin was 1.3 mg. Blood urea was 37.6 mg. and N.P.N. 34 mg./100 c.c. Total protein 5.2 per cent; serum albumin 3.8 per cent and serum globulin 1.4 per cent. Blood cholesterol was 160 mg.|100 c.c. W.R. was negative and Takata-Ara test was positive. Galactose was 10 mg. Urine urobilinogen was absent and 0.39 gramme of hippuric acid was synthesized. Fæcal urobilinogen was 150 mg.

The above findings show that most of the data are in favour of normal liver function except the low hippuric acid synthesis.

The liver biopsy (figure 4, plate XXV) revealed cloudy swelling, and no evidence of cirrhosis was present. Jaundice, probably of the hæmolytic type, was a result of malarial infection.

Case V. P. H., Hindu male, aged 40 years, was admitted for treatment. He gave a history of cough and expectoration, pain in abdomen and anorexia of about six months' duration. The patient had an attack of malaria four months back. He gave no history of amœbic dysentery.

He was markedly wasted and anæmic. There was no jaundice or ascites. The liver was found enlarged 1½ inches and was smooth, soft but not tender. The spleen was enlarged 5 inches below the costal region. It was hard and regular. Soft systolic murmurs were present in the pulmonary and mitral areas and crepitations were present in both lungs. The vocal resonance was diminished over the right base. The skiagram of the chest showed infiltration of the right lower and left middle zones. The working diagnosis of pulmonary tuberculosis was made. The cause for the enlargement of the spleen and the liver could not be definitely ascertained.

The laboratory findings in this case were R.B.C. 2,850,000 per c.mm.; hæmoglobin 7.5

grammes ; W.B.C. 4,600 per c.mm.; P—76 ; L—21; M—2; E—1; M.C.V. 100 cu μ; M.C.H. 26 $\gamma\gamma$; M.C.H.C. 26 per cent; reticulocytes 1 per cent; prothrombin concentration was 61 per cent and was raised to 76 per cent after an administration of 10 mg. of vitamin K. Icteric index 6; van den Bergh's reaction was negative and serum bilirubin was less than 0.25 mg. Blood urea was 39.8 mg.; N.P.N. 36 mg./100 c.c. ; total protein 4.7 per cent ; serum albumin 2.8 per cent and serum globulin 1.9 per cent ; W.R. was negative. Urine urobilinogen was 10 mg. Fæcal urobilinogen was normal. Hippuric acid synthesis was 0.9 gramme. The sputum on repeated examination was negative for acid-fast bacilli. The analysis of the function tests showed a low prothrombin response after vitamin K injection pointing towards liver damage.

The needle biopsy of the liver was performed and showed the presence of amyloid disease (figure 5, plate XXV), a finding which was rather unexpected in view of the absence of any chronic suppurative lesion or evidence of neoplasm. The *x*-ray examination had shown pulmonary tuberculous infiltration but there was no evidence of caseation or cavity formation.

The case was one of amyloid disease of the liver following pulmonary tuberculosis.

Case VI. D. H. M., aged 15 years, was admitted into the Thomason Hospital with complaint of enlarged spleen and liver. He suffered from malaria for six months about two years back. The spleen was enlarged since then while the liver has been enlarged for the last six months.

He was a young boy of good build, slightly anæmic. There was no jaundice or ascites. The spleen was hard and smooth, 8 inches below the costal margin. The liver was felt 3 inches below the costal margin. It was smooth, moderately hard and not tender. The working diagnosis of chronic malaria and cirrhosis of the liver was made. Kala-azar was also thought to be a possibility and so also megalocytic anæmia.

The laboratory investigations showed : R.B.C. 2,150,000 per c.mm.; Hb. 5.5 grammes; W.B.C. 4,400 per c.mm.; P—55 ; L—42 ; M—3 ; P.C.V. 21 per cent ; M.C.V. 100 cuμ; M.C.H. 26 $\gamma\gamma$; M.C.H.C. 26 per cent. The prothrombin concentration of 84 per cent was raised to 100 per cent after vitamin K. Icteric index was 8 and serum bilirubin was 0.3 gramme. Blood urea was 28.5 mg.; N.P.N. 25.6 mg.; total proteins 5.6 grammes ; serum albumin 3.2 grammes and globulin 2.4 grammes. Aldehyde and antimony tests for kala-azar were negative. Hippuric acid synthesis was 0.9 gramme. Urine showed excess of urobilin.

From these findings the liver could be assessed as perfectly normal, the cause for the enlargement remaining undetected. The needle biopsy of the liver showed increased cells in the sinusoids and portal tracts. The cells gave the impression of being formed from sinusoidal epithelium. The picture (figure 6, plate XXV) suggested histiocytic medullary reticulosis. No changes suggesting cirrhosis were present.

The case illustrates that the part liver biopsy may play in the detection of the cause of liver enlargement where the function tests are perfectly normal.

Case VII. S., Hindu female, aged 38 years, was admitted with a history of fever for seven months, pain in abdomen and indigestion. She had been having attacks of dysentery for the last 1½ years. There was an attack of malaria about an year back lasting for about two months.

She was a poorly nourished and anæmic woman. There was no jaundice but there was slight ascites. She had a low temperature ranging between 99° and 100°. Oedema was present in both the legs. The liver was enlarged 6 inches below the costal margin. It was smooth, hard and slightly tender. The spleen was not palpable. Working diagnosis of amœbic hepatitis was made.

Laboratory investigations showed : R.B.C. 2,000,000 per c.mm.; Hb. 4 grammes; W.B.C. 6,150 per c.mm.; P—86; L—13; M—1; P.C.V. 15 per cent; M.C.V. 75 cuμ; M.C.H. 80 $\gamma\gamma$; M.C.H. 26 per cent ; prothrombin concentration was 100 per cent ; icteric index 18 ; serum bilirubin 0.4 mg.; blood urea 36.4 mg.; N.P.N. 33 mg.; total proteins 5.24 grammes ; serum albumin 2.8 per cent ; serum globulin 2.44 per cent ; blood cholesterol was 230 mg. per 100 c.cm.; W.R. negative. Urine urobilinogen was 10 mg. and 0.38 gramme of hippuric acid was synthesized. Fæcal urobilinogen was 200 mg. The sputum showed pus cells and the usual oral flora.

The analysis of the findings show that liver damage was indicated by the low hippuric acid synthesis and jaundice. The patient was given 12 emetine hydrochloride injections of 1 grain each with no improvement.

The liver biopsy was done (figure 7, plate XXVI) and the histological examination showed portal cirrhosis. In spite of the cirrhotic condition, the liver-prothrombin concentration was normal. Though damaged liver was diagnosed the cause of it was only located after biopsy.

Case VIII. L., Hindu male, aged 45 years, was admitted with the complaints of fever for 4 months, swelling of feet, scanty urine and ascites, all of about 3 months' duration. The examination revealed that the patient was anæmic and had ascites. The spleen was enlarged 3½ inches and the liver was not palpable. After paracentesis abdominis, the liver was still not felt.

The working diagnosis of ascites secondary to atrophic cirrhosis of the liver was made.

The laboratory investigations revealed prothrombin concentration of 53.3 per cent raised to 64 per cent after vitamin K. Icteric index 3; serum bilirubin normal. Blood urea 47.6 mg.;

N.P.N. 42.8 mg.; total protein 4.9 grammes; serum albumin 2.5 grammes; serum globulin 2.4 grammes and hippuric acid synthesis of 0.5 gramme. Urine urobilinogen was normal. Fæcal urobilinogen was present. The protein of the ascitic fluid was 3.8 per cent.

The liver biopsy by needle did not enable us to secure specimen for the histological examination. Later, after the patient died, post mortem was performed. The histological examination of the liver tissue (figure 8, plate XXVI) showed miliary tuberculosis of the liver and the capsule. Low prothrombin response and low hippuric acid synthesis suggest a damaged liver but the cause of the damage presumed to be cirrhosis of the liver was due to tuberculosis of the liver. Ascites was due to tuberculous peritonitis.

Conclusions

1. A few cases of hepatic diseases have been presented in which from clinical findings alone difficulty was experienced in making a correct diagnosis.
2. Liver biopsies have been carried out with the Vim-Silverman needle, and the histological picture of each has been presented as micro-photographs.
3. A number of function tests have been performed in each case and the findings have been presented.
4. An attempt has been made to correlate the clinical, chemical and histological findings, and difficulties in the interpretation of function tests have been pointed out.
5. Single tests have been found useless in the assessment of the nature and extent of hepatic impairment. It has been found that a combined study of the liver functions and the histological findings gives a better information about the nature and extent of the liver diseases. The cause of liver damage in most cases can be definitely established by biopsy studies alone and from the characteristic profiles it is hoped that the extent of hepatic impairment can be properly assessed.
6. Comparative value of the various liver function tests has been assessed, as the correct diagnosis of the nature of the liver affection was possible by the histological examination of the biopsy material.

Our thanks are due to Major-General H. C. Buckley, I.M.S., Superintendent, Thomason Hospital, and Principal, Medical College, Agra, for the facilities given to us for carrying on this work; to Drs. G. N. Vyas, K. N. Gaur and B. K. Dube, physicians, for allowing us to perform the liver biopsy and function tests on their cases; and to Drs. Kashi Nath and V. N. Tiwari, for assisting us in the work.

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BASAL METABOLIC STUDIES IN THE PUNJAB

By LAL CHAND KHANNA, P.C.M.S.
Professor of Physiology, K. E. Medical College, Lahore
and
S. S. MANCHANDA, P.C.M.S.

It had long been felt that while valuable work on basal metabolism had been done in Bengal (Bose and De, 1934; Mukherjee and Gupta, 1931), Madras (Krishnan and Vareed, 1932; Mason and Benedict, 1931), Bombay (Sokhey and Malandkar, 1939; Niyogi, Patwardhan and Mordecai, 1939; Niyogi, Patwardhan and Sirsat, 1941), Lucknow (Banerji, 1931), Hyderabad, Deccan (Rahman, 1936) and Coonoor (Rajagopal, 1938) no normal standards were available for the Punjab which differs from other provinces of India chiefly in having wheat-eating population, better physique and extremes of climate. There is considerable evidence that dietetic, climatic and occupational factors influence basal metabolism. Basal metabolic rate in other provinces of India deviates markedly from the accepted American and European standards. This observation made the demand for an investigation of the standards in the Punjab all the more important.

Though this work was undertaken with this limited objective yet the greatest possible accuracy in the experimental technique was aimed at and full data were recorded with a view to make this study serve a somewhat wider purpose. The investigation is limited to one hundred individuals only, comprising sixty males and forty females, mostly medical students and members of the college staff, between the ages of 16 and 36 years, representing all communities.

Experimental

The usual standard precautions, namely 14 to 16 hours' fasting, least muscular exertion, mental repose, etc., were strictly adhered to in the preparation of the cases who were otherwise quite fit and healthy. The investigation was carried