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RETROSPECTIVE ASSESSMENT OF LATENT JAUNDICE DETECTED DURING LIVER FUNCTION TESTS

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ABSTRACT

Jaundice, as clinical sign, has been recorded for over 2000 years; however the jaundiced patients are frequently ill, uncomfortable, slightly unable to work, thus it is worthwhile to review this problem from time to time. It is apparent clinically (clinical jaundice) when serum bilirubin concentration reaches 2-3 mg/dl. If serum bilirubin concentration is below 2 mg/dl it is called latent jaundice (sub-clinical-jaundice since at this stage it is not yet detectable). Latent jaundice is often an ignored aspect, hence the study was planned retrospectively to evaluate this aspect. The study was conducted in the main clinical lab, Department of Biochemistry, Government Medical College, Jammu, for a period of 3 months, i.e. May 2015 to July 2015. Sampling, reagents delivery, mixing, processing and printing of results were automatically performed by the Siemens Dimension Clinical Chemistry system. There is a need for an earlier investigation to identify latent jaundice patients, inspite of the fact that they tend to show very little or no clinical symptoms. The reason for identifying latent jaundice and comparing it with clinical jaundice is significant enough as even on random sampling the normal subjects may be as much poised to be suffering with the disease as the overtly jaundiced patient. Serum bilirubin is of immense value in diagnosing and monitoring the cases of latent jaundice. Latent jaundice should be addressed properly by further work-up of the patients and close follow up.

KEY WORDS: Latent jaundice, subclinical, bilirubin, liver.

INTRODUCTION

The liver is the first stop for all nutrients, toxins, and drugs later absorbed by the digestive tract.[1] Liver performs a variety of different biochemical, synthetic and excretory functions. Bilirubin is a product of the breakdown of hemoglobin inside red blood cells and its serum levels are associated with the function of the liver. If bilirubin does not leave the body, it accumulates and discolors other tissues: termed jaundice.[2] Hyperbilirubinemia seen in acute viral hepatitis is directly proportional to the degree of histological injury and the longer course of the disease.[3] Jaundice is a physical sign characterized by yellow appearance of the patient due to deposition of bile pigments (bilirubin) in the skin, mucous membrane and sclera due to high elastin content in these tissues. It is apparent clinically (clinical jaundice, Table 1) when serum bilirubin concentration reaches 2-3 mg/dl whereas if serum bilirubin concentration is below 2 mg/dl it is called latent jaundice (sub-clinical-jaundice since at this stage it is not yet detectable clinically).[2-5] Latent jaundice is an often neglected issue in healthcare practice and this was the focus of the present study.

Table 1: Biochemical differentiation of three types of jaundice based on blood tests.

Sample	Biochemical parameter	Type of jaundice		
		Pre-hepatic	**Hepatic**	**Post-hepatic**
Blood	Serum bilirubin	↑	↑↑	↑↑↑
	Types of bilirubin	Unconjugated	Mixed	Conjugated
	Serum transaminase (ALT/SGPT)	N	↑↑↑	↑
	Serum alkaline phosphate	N	↑	↑↑↑
	Serum 5'-nucleotidase	N	↑	↑↑↑
	Prothrombin time (PT)	N	↑	↑
	Effect of parental vitamin K on PT	-	Remains ↑	↓(normalizes)
	Causes	Abnormal red cells; Antibodies; Drugs and toxins; Thalassemia; Hemoglobinopathies; Gilbert's syndrome; Criggler-Najjar syndrome	Viral hepatitis; Toxic hepatitis; Intrahepatic cholestasis	Extrahepatic cholestasis; Gallstones; Tumours of bile duct; Carcinoma of pancreas; Lymph node enlargement in porta hepatis

MATERIALS AND METHODS

The study was conducted in the main clinical lab, Department of Biochemistry, Government Medical College, Jammu, for a period of 3 months, i.e. May 2015 to July 2015. All the

subjects were admitted in the Medicine Ward with some complaints, advised for serum total bilirubin. Investigations done were included in the study irrespective of gender or age. 2ml of venous blood was collected from antecubital vein under aseptic conditions and serum was separated. TBI method was evaluated for interference according to CLSI/NCCLS EP7-A.[5] It is a modification of Doumas reference method,[6] (which is a modification of Diazo method described by Jendrassik and Grof in 1938).[7] Briefly, diazotized suphanilic acid is formed by combining sodium nitrite and suphanilic acid at low pH. Bilirubin (unconjugated) in the sample is solubilized by dilution in a mixture of EDTA. Upon addition of diazotized suphanilic acid the solubilized bilirubin including conjugated bilirubin and the delta forms is converted to diazobilirubin, a red chromophore which absorbs at 540nm.Sampling, reagents delivery, mixing, processing and printing of results were automatically performed by the Siemens Dimension Clinical Chemistry system.

Reference range [5,8]

Normal subjects:	total serum bilirubin: 0.2- 1mg/dl.
Clinical jaundice:	total serum bilirubin ≥2 mg/dl.
Sub clinical/Latent jaundice:	total serum bilirubin 1.0 to 2.0 mg/dl

RESULTS AND DISCUSSION

Total no of samples investigated in consecutive three months, i.e. May, June and July, 2015, in the main lab, Department of Biochemistry, GMC, Jammu were 12235. Out of these, total number of patients investigated for bilirubin was 8191. The number of patients diagnosed with frank jaundice after biochemical investigations revealing the bilirubin range > 2.0 mg/dl was 949 (11.6% of 8191) “Fig.1”. On the other hand, number of patients not diagnosed for clinical jaundice but falling under latent or subclinical jaundice after biochemical investigations (bilirubin range between 1.0 to 2.0 mg/dl) was 747 (9.12% of 8191) “Fig.1”. Number of patients diagnosed normal based upon their range below 1.0 mg/dl was 6493 (79.27% of 8191) “Fig.1”.

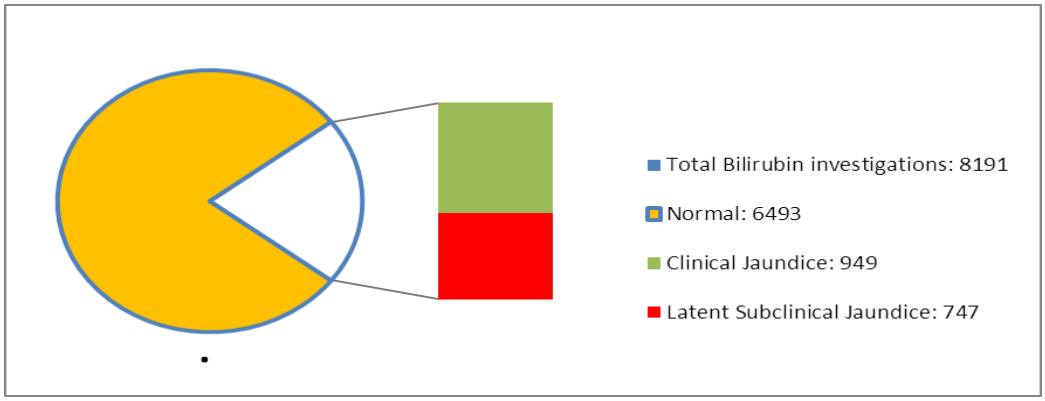

Fig. 1: Distribution of Patients Based on Clinical, Latent Subclinical Jaundice

However, on comparing, we found the ratio of patients diagnosed with clinical jaundice to patients possibly under latent subclinical jaundice to be 949: 747 = 1: 0.787 (which is very significant). This shows that the patients, here diagnosed for latent jaundice, were still in the safer zone of serum bilirubin values (1 to 2 mg/dl) and hence their treatment can proceed in a better manner. Further, the ratio of healthy normal patients is not evenly distributed in the complete range (0.1 to 2.0 mg/dl) as the 'assumed normal' patients (747) are only 10.32% of total no of normal(i.e. 7240) which is very insignificant. Hence, for practical reasons, this is an abnormal outcome of investigative results. Either the normal range of bilirubin needs to be reassessed or 'latent jaundice' is too significant a phenomenon to be ignored.

There is a need for an earlier investigation to identify latent jaundice patients, inspite of the fact that they tend to show very little or no clinical symptoms. Reason for identifying latent jaundice and comparing it with clinical jaundice is significant enough as even on random sampling the normal subjects may be as much poised to be suffering with the disease as the jaundiced patient.

CONCLUSION

No single biochemical test can detect the global functions of the liver. All laboratories usually employ a battery of tests for initial detection and management of liver diseases known as liver function tests. Often, latent jaundice is the only aspect suggestive of liver disease which is clinically silent until late in its course and the clinicians are faced with reports that do not tally with clinical condition of the patient and they face difficulty in interpreting the jaundice. For this reason, serum bilirubin is of immense value in diagnosing and monitoring the cases of latent jaundice. Latent jaundice should be addressed properly by further work-up of the patients and close follow up.

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