RESEARCH ARTICLE

A CLINICAL STUDY OF ORAL GLUCOSE TOLERANCE TEST IN CHRONIC LIVER DISEASE

Abhilash Tadiboina and Kandula Venkateswara Reddy
Katuri Medical College.

Abstract

Background: As liver is the principal organ for metabolism of the carbohydrates, chronic liver disease (CLD) may affect carbohydrate metabolism. Hence we can utilise oral glucose tolerance test (OGTT) to study the disturbances in carbohydrate metabolism in CLD. Objectives include: 1) To know clinical features and laboratory manifestations of chronic liver disease. 2) To know various results of OGTT in CLD. 3) To study various classes of cirrhosis (as in child-grading)

Methods: Study was conducted over a period of 18 months in Katuri medical college where clinical, laboratory manifestations and OGTT results in 30 cases of CLD and 30 controls (age and sex matched) with cured respiratory tract infection. Age more than 65 years, pregnant women, patients with h/o diabetes, clinical features suggestive of chronic cholestatic diseases and h/o alcoholism in controls were excluded from the present study.

Results: CLD was found to be more common in males (73.3%) than females. CLD was more common in lower socioeconomic status (63%). Male: female ratio is equal for chronic hepatitis C equals to one. Alcoholic cirrhosis, chronic alcohol hepatitis and primary hepatoma with cirrhosis were seen only in male and cryptogenic cirrhosis was seen in only female. Most of the cases had anemia. Cirrhosis is the most common cause for CLD (66.6/20 cases). OGTT showed rise in blood sugar levels in cases which was significant when compared with control group (p<0.05). OGTT showed impaired response in 46.6% cases, impaired glucose tolerance (IGT) in 23.3%, diabetic response in 23.3% and normal response in 53.3% cases. Cirrhosis is the most common (78.5%) cause for impaired glucose response.

Conclusions: It can be concluded that CLD is more common in male and lower socioeconomic status. There is significant impaired response to glucose load in CLD. Cirrhosis, among CLD’s is the most common etiology for impaired response.
Introduction:

1. In view of the central role\(^1\) the liver plays in blood glucose regulation, it is not surprising that abnormalities of carbohydrate metabolism are found in CLD, like cirrhosis of liver, chronic hepatitis, hepatocellular carcinoma, etc.
2. Metabolic disorders are evident in patients with advanced liver disease, and the manifestations are similar regardless of the initial etiologic insult\(^2\). To a varying degree, similar abnormalities are observed in patients with severe chronic hepatitis, micronodular cirrhosis, and post necrotic cirrhosis.
3. The liver maintains a healthy level of blood sugar by a combination of gluconeogenesis, glycogenolysis, and glycogen. The metabolic changes in cirrhosis are complex and not fully understood. In fulminant acute hepatic necrosis the blood glucose levels may be low; this is rare in chronic liver disease.
4. In fasted patients with cirrhosis, the contributions of carbohydrates to energy production are reduced to (2% Vs. 38% in control) with contributions from fat increasing (86% Vs 45% in control)\(^3\). This may be caused by impaired release of hepatic glucose or a reduced reserve of glycogen in the liver.
5. However, in cirrhotics, following a meal\(^4\) or glucose load, there is hyperglycemia because of inability of liver to metabolize glucose, probably due to insulin resistance. Patients with cirrhosis may have elevated serum lactate levels, reflecting the decreased capacity of liver to utilize lactate for gluconeogenesis.
6. It has been shown experimentally that hepatectomised\(^5\) dog rapidly developed hypoglycemia unless glucose is maintained with intravenous glucose drip; this can be explained by the fact that G-6-Pase is present mainly in the liver.

Advanced genetic hemochromatosis causes diabetes mellitus; Diabetes is also associated with chronic hepatitis C virus infection\(^6,7\) and may occur in patients with chronic autoimmune hepatitis, probably due to shared immune genetic predisposition (HLA-B8 & HLA-DR3).

Disturbances of carbohydrate metabolism in chronic liver disease were directly correlated with degree of hepatocellular dysfunction\(^8\). The underlying mechanisms of these are sophisticated and not fully understood\(^8,9\). Oral glucose tolerance test (OGTT) can be used for assessing the glucose homeostatic power of the liver in chronic liver disease, where the reserve function reduced as there are many factors that alter the glucose tolerance curves, for example infections, old age, drugs, and many other systemic diseases. Hence other evidence like previous history of diabetes mellitus, history of taking diuretics and other causes of glucose intolerance should be excluded before interpreting the glucose tolerance test.

Study of OGTT is superior\(^10\) to intravenous GTT because glucose is presented to the body by a natural route and there is an opportunity for regular stimulation of insulin secretion by various hormones of gastrointestinal tract. Many studies with intravenous GTT have shown abnormal glucose tolerance curve in a significant proportion of cases.

Objectives:

1. To know clinical features and laboratory manifestations of chronic liver disease.
2. To know various results of oral glucose tolerance test in chronic liver disease.
3. To study various classes of cirrhosis [as in Child- glucose tolerance.

This study was carried out in Katuri Medical College, hospital and research Center, under the guidance of Dr. Gorantla RamaKrishna, Associate Professor, and Department of medicine.

I studied the clinical and laboratory manifestations and OGTT in 30 patients with chronic liver disease admitted in our hospital. 30 patients with cured respiratory tract infection acted as a control group. The results of the investigation and OGTT have been recorded and tabulated. Depending on the data obtained according to WHO criteria for OGTT (Table No, 3), I have evaluated the results and come upon a conclusion regarding the study.
Inclusion and exclusion criteria:

Inclusion criteria:
Patients were presenting to our hospital with clinical and/or histopathologically diagnosed chronic liver disease between the age group 18-65 years.

Exclusion criteria:
1. Know diabetes mellitus on treatment with insulin and oral hypoglycemic agents.
2. Patients on drugs that cause hyperglycemia are excluded.
3. Patients with any other condition causing hyperglycemia other than chronic liver disease
4. Pregnant women and chronic cholestatic illness are excluded from the study.
5. Among the control group, past h/o alcoholism or jaundice are excluded from the study.

Figure 5: Age and sex distribution in cases

Results:
Thirty cases of chronic liver disease admitted in our hospital have been collected as per the criteria mentioned in the materials and methods.

The control group included thirty cases of age and sex-matched, cured upper respiratory tract infection (I.P/O.P).

Table No.6: Age and Sex distribution.

| Age group | Male |  | Female |  | Total |  |
|-----------|------|---|--------|---|-------|---|
| Years     | No   | % | No     | % | No    | % |
| 10-20     | 1    | 3.3| 0      | 0 | 1     | 3.3|
| 21-30     | 2    | 6.6| 1      | 3.3| 3     | 9.9|
| 31-40     | 7    | 23.3| 4     | 13.3| 11    | 36.3|
| 41-50     | 6    | 20 | 2      | 6.6| 8     | 26.6|
| 51-60     | 3    | 10 | 1      | 3.3| 4     | 13.3|
| 61-70     | 3    | 10 | 0      | 0 | 3     | 10 |

Age: Age of the patients range from 18 years to 64 years in this study. The majority (43.3%) of chronic liver disease (CLD) occurred among 31-50 years. The mean age is years.

Sex: among 30 cases of CLD, 22 (73.3%) were male, and 8 (26.6%) were females. The ratio of male: female was 2.75:1.

Socioeconomic status: majority (63.3%) are low socioeconomic status, rest are middle socioeconomic status.
Anorexia was the most common (80%) symptom of CLD, seen in 24 cases.

Fatigue is the next common (70%) symptom seen in 21 cases.

Distention of abdomen, nausea and vomiting, jaundice, and pain abdomen are also frequent, seen in 36.6%, 33.3%, 33.3%, and 33.3%, respectively. Fever and body ache is seen in 30% each.

Hematemesis/ melena was least common (16.6%) symptom seen in severe cases of cirrhosis (83.3% of Child-Pugh’s Class-B).

### Table No.7: Symptomatology

| Symptom                    | No. of cases | Percentage |
|----------------------------|--------------|------------|
| Anorexia                   | 24           | 80         |
| Fatigue                    | 21           | 70         |
| Distention of abdomen      | 15           | 50         |
| Nausea and vomiting        | 11           | 36.6       |
| Jaundice                   | 10           | 33.3       |
| Pain abdomen               | 10           | 33.3       |
| Fever                      | 9            | 30         |
| Body ache                  | 9            | 30         |
| Hematemesis/melena         | 5            | 16.6       |

### Table No.8: Past History

| Past history                | No. of cases | Percentage |
|-----------------------------|--------------|------------|


Alcoholism 12 40
Blood transfusion 2 6.6
Childhood h/o 2 6.6
Extramarital sex 2 6.6
Needle prick 1 3.3
History in husband 1 3.3
Nil 10 33.3

**Table No.9:** Signs.

| Sign            | No. of cases | Percentage |
|-----------------|--------------|------------|
| Ascites         | 16           | 53.3       |
| Hepatomegaly    | 15           | 50         |
| Icterus         | 13           | 43.3       |
| Clubbing        | 13           | 43.3       |
| Palmar erythema | 11           | 36.3       |
| Splenomegaly    | 9            | 30         |
| Spider naïve    | 7            | 23.3       |
| Gynecomastia    | 5            | 16.6       |

Ascites is the most common (53.3%) sign seen in 16 cases.

Hepatomegaly is the next common sign seen in 50% of cases.

Icterus, clubbing, and palmar erythema, are seen in 43.3%, 43.3%, and 36.3%. Splenomegaly, spider naïve, and gynecomastia are also common.
There are any symptoms or sign of autoimmune disease. There are no symptoms or signs of hemochromatosis, Wilson’s disease, Antitrypsin deficiency. None of cases had any clinical features of chronic cholestatic diseases. Hepatic encephalopathy: of early-stage, is seen in 6 (20%) cases, all are severe cases of cirrhosis (all are cirrhosis of Child-Pugh’s Class-B).

Liver function tests:
Mean total serum bilirubin is 2.19 mg %, with 0.9mg% and 4.7mg% being lowest and highest values, respectively, 22 cases (73.3%) are having hyperbilirubinemia.

Mean serum albumin is 3.73 gm% is, with 2.5gm% and 4.5gm% being lowest and highest values respectively, the majority (86.7%) having normal limits, 4 cases have hypoalbuminemia, 75% are from Child’s class-B cirrhosis.

Mean serum globulin is 2.66 gm % is, with 2.2 gm % and 3.5gm% being lowest and highest values respectively, 6 cases (20%) show increased globulin levels, of which 5(83.3%) are cirrhotics.

Mean serum AST in IU/L is 132 with 65 and 252 being highest and lowest values, respectively, high costs are recorded in alcoholic liver disease. Serum ALT in IU/L is 101 with 23 and 356 being highest and most moderate values respectively; high values are recorded in chronic active hepatitis.

Mean serum alkaline phosphatase in IU/L is 50, with being lowest being 156, seen in primary hepatoma a with cirrhosis.

Mean hemoglobulin is 9.97 gm% with 7.8 and 12.8 lowest and highest, respectively; low values are seen in severe cirrhosis and hepatoma. Among cases, 63% of males are anemic (<12gm%), and 50% of females are anemic (<10gm%) Total count, differential count, and platelet count were within normal limits in all cases. Clotting time and bleeding time are within normal limits. In peripheral smear, 66.6 % (20cases) showed normocytic normochromic response rest showed microcytic hypochromic response, because of inadequate intake or chronic blood loss through varices. Serum creatinine was within normal range for all cases, and none of them were in hepato-renal syndrome.

Serum electrolytes levels, including K⁺ are within normal limits. In all patients with clinical ascites, ascitic fluid analysis was done and shown to be transudate. None of the cases had any autoimmune disease or showed any autoantibodies.

| Table No.10: Viral markers: |          |          |
|---------------------------|----------|----------|
| Viral marker              | No. of cases | Percentage |
| HCV Ab                    | 11       | 36.6     |
| HB s Ag                   | 7        | 23.3     |
| Nil                       | 12       | 40       |

Analysis for viral markers was done for all cases, HCV Ab is positive in 11 (36.6%) cases, HBs Ag is positive in 7 (23.3%), and no viral markers were found in 12 (40%) cases.

Figure No.9: Histopathological diagnosis of cases.
Figure No.10: Etiological diagnosis in cases.

Figure no.11: Micronodular cirrhosis. Patient name: n. Ramesh

Figure no.12: Macronodular (Post necrotic) cirrhosis Patient name: CH. Padma.
Figure No.13: Chronic hepatitis: Patient name: Subhashini

Figure No.14: Hepatocellular Carcinoma Patient name: Sk. Bude

Table No.11: Histopathology:

| Histopathological diagnosis       | No. of cases | Percentage |
|-----------------------------------|--------------|------------|
| Post necrotic cirrhosis           | 7            | 23.3       |
| Chronic active hepatitis          | 4            | 13.3       |
| Chronic persistent hepatitis      | 4            | 13.3       |
| Alcohol hepatitis                 | 2            | 6.6        |
| Micronodular cirrhosis            | 1            | 3.3        |
| Hepatoma with cirrhosis           | 1            | 3.3        |
Histopathological examination was done in doubtful cases to confirm chronic liver disease. Of 19 (63.3%) liver biopsies done, 7 (23.3%) showed post necrotic cirrhosis, 4 (13.3%) showed chronic active hepatitis, 4 (13.3%) showed chronic persistent hepatitis, 2 (6.6%) showed alcoholic hepatitis, 1 (3.3%) showed alcoholic cirrhosis, and 1 (3.3%) showed hepatoma with cirrhosis.

Table No.12: Etiological diagnosis for chronic liver disease:

| Etiology                           | No. of cases | Percentage |
|------------------------------------|--------------|------------|
| Alcoholic cirrhosis                | 9            | 30         |
| Post necrotic cirrhosis-C          | 7            | 23.3       |
| Post necrotic cirrhosis-B          | 2            | 6.6        |
| Chronic persistent hepatitis-C     | 3            | 10         |
| Chronic persistent hepatitis-B     | 1            | 3.3        |
| Chronic active hepatitis-C         | 1            | 3.3        |
| Chronic active hepatitis-B         | 3            | 10         |
| Chronic alcohol hepatitis          | 2            | 6.6        |
| Primary hepatoma with cirrhosis    | 1            | 3.3        |
| Cryptogenic cirrhosis              | 1            | 3.3        |

When the etiology of chronic liver disease is considered, of 30 cases, 20 (66.6%) were cirrhosis, and 10 (33.3%) were chronic hepatitis. Out of 20 cases of cirrhosis, 9 (30%) were alcoholic cirrhosis, 9 (30%) were post necrotic cirrhosis, one example was primary hepatoma with cirrhosis and one case of cryptogenic cirrhosis.

Out of 10 cases of chronic hepatitis, 4 (13.3%) were chronic active hepatitis, 4 (13.3%) were chronic persistent hepatitis, and 2 (6.6%) were chronic alcoholic hepatitis. The majority (36.6%) of chronic liver disease is due to chronic infection with the hepatitis C virus.

Table No.13: Etiological diagnosis according to Age group:

| Age group | AC | PNC | Cr-C | CH-C | CH-B | CAH | PH-C |
|-----------|----|-----|------|------|------|-----|------|
| 10-20     | -  | -   | -    | 1    | -    | -   | -    |
| 21-30     | -  | -   | -    | 1    | 1    | 1   | -    |
| 31-40     | 1  | 4   | -    | 2    | 3    | 1   | -    |
| 41-50     | 5  | 2   | 1    | -    | -    | -   | -    |
| 51-60     | 2  | 2   | -    | -    | -    | -   | -    |
| 61-70     | 1  | 1   | -    | -    | -    | -   | 1    |

Alcoholic cirrhosis was seen only in males, mainly (5 cases/16.6%) in age group 41-50 years. Female PNC is seen commonly (4 cases/13.3%) in age group 31-40 years.

Table No.14: Etiological diagnosis according to sex:

| Sex       | AC | PNC | Cr-C | CH-C | CH-B | CA-H | PH-C |
|-----------|----|-----|------|------|------|------|------|
| Male      | 9  | 5   | -    | 2    | 3    | 2    | 1    |
| Female    | -  | 4   | 1    | 2    | 1    | -    | -    |

Male: female ratio is equal for chronic hepatitis C; it is 1.25 for post necrotic cirrhosis; it is 3 for chronic hepatitis B. Alcoholic cirrhosis, chronic alcohol hepatitis, and primary hepatoma with cirrhosis is seen only in males, and cryptogenic cirrhosis is seen only in female.
When cirrhotic (20 cases) patients were graded according to Child- Pugh ‘s grading, class A, seen in 14 (70%) cases of cirrhosis, B is seen in 6 (30%) cases of cirrhosis. Class B is associated with the early stages of hepatic encephalopathy and Grade 1/2 esophageal varices. None of the class C is included in the study.

Ultrasound examination showed increased echogenicity and nodularity in 19 (63.3%), 10 (33.3%) showed coarse echo pattern, and one case showed increased echogenicity with mass lesions.

Endoscopic examination showed Grade 1 esophageal varices in 3 (10%) cases and Grade 2 esophageal varices in one case. All are severe cases of cirrhosis (Child-Pugh’s Class-B).

Cases who have volunteered and gave written consent were subjected to the Oral glucose tolerance test (OGTT). Cases were fed with minimum of 300g of carbohydrate per day for three days prior to OGTT.

Patients with cured respiratory tract infection, after age, sex, and BMI (mean-18.6 kg/m$^2$) matching were taken as controls. Persons who had previous history of jaundice, alcoholism, diabetes and exposure to other hepatotoxic drugs were excluded.
Controls were also examined in detail and investigated with Hb%, LFTs, ultrasonography, FBS, and serum creatinine was done. Those who have laboratory values within normal limits have volunteered and gave written consent were subjected to Oral glucose tolerance test (OGTT).

Controls were fed with a minimum of 300g of carbohydrate per day for three days prior to OGTT were subjected to OGTT, as for cases. The mean blood glucose level in case and control at fasting, 1/2hr, 1hr, 1 1/2 hr, 2 hr are as follows.

Table No.16:- Mean blood sugar in cases and controls:

| Time in hours  | Blood sugar level mg% | Control | Case  |
|----------------|-----------------------|---------|-------|
| Fasting        | 78.10                 | 80.40   |       |
| 1/2 hr         | 105.13                | 115.2   | 0     |
| 1 hr           | 123.33                | 148.3   | 7     |
| 1 1/2 hours    | 102.53                | 158.4   | 3     |
| 2 hours        | 88.70                 | 165.2   | 0     |

After OGTT, data is tabulated and analyzed in both case and control groups. Case and control are compared using ‘paired T-test’ and calculated the p-value.

Figure No.18:- Responses of OGTT in cases.
Table No.17:-Group statistics:

| Blood sugar in | Group     | No  | Mean  | Standard Deviation | Standard Error of mean |
|----------------|-----------|-----|-------|--------------------|------------------------|
| Fasting        | Control   | 30  | 78.10 | 5.95               | 1.09                   |
|                | Case      | 30  | 80.40 | 8.97               | 1.64                   |
| ½ hour         | Control   | 30  | 105.13| 9.65               | 1.76                   |
|                | Case      | 30  | 115.20| 13.7               | 2.51                   |
| 1 hour         | Control   | 30  | 123.33| 6.37               | 1.16                   |
|                | Case      | 30  | 148.37| 30.5               | 5.58                   |
| 1½ hours       | Control   | 30  | 102.53| 9.35               | 1.71                   |
|                | Case      | 30  | 158.43| 42.3               | 7.73                   |
| 2 hours        | Control   | 30  | 88.70 | 4.85               | 0.89                   |
|                | Case      | 30  | 165.20| 58.1               | 10.6                   |

Table No.18:-Independent samples test:

| Blood sugar during | Assumption             | Levene’s test for equality of variances | F    | Significance P-value |
|--------------------|------------------------|----------------------------------------|------|----------------------|
| Fasting            | Equal variances assumed| 3.007                                  | 0.088|
| 1/2 hour           | Equal variances assumed| 2.779                                  | 0.101|
| 1 hour             | Equal variances assumed| 94.854                                 | 0.0001|
| 1 ½ hours          | Equal variances assumed| 115.865                                | 0.0001|
| 2 hours            | Equal variances assumed| 73.294                                 | 0.0001|

When means of case and control are compared, there is a significant difference in 1hr, ½ hrs, 2- hrs glucose levels, (i.e., p:0.0001).

Table No.19:-Age, sex, and etiological distribution of impaired glucose response:

| Age group | Diabetic response | IGT response |
|-----------|-------------------|--------------|
|           | Male              | Female       | Male         | Female       |
| 10-20     | -                 | -            | 1-CH-C       | -            |
| 21-30     | -                 | -            | -            | -            |
| 31-40     | 1-PNC-C           | 1-AC         | 1-PNC-C      | 1-CH-C       |
|           | 1-AC              | -            | 1-AC         | 1-Cr-C       |
| 41-50     | 1-AC              | -            | 1-AC         | -            |
| 51-60     | 1-AC              | -            | 1-PNC-C      | -            |
| 61-70     | 1-PNC-C           | -            | 1-AC         | 1-PNC-B      |

OGTT shows impaired response in 14 (46.6%) cases, impaired glucose tolerance (IGT) in 7 (23.3%) cases, diabetic response in 7 (23.3%) and normal response in 16 (53.3%) cases. There was fasting hypoglycemia in a case of cirrhosis with hepatoma.

Among 22 (73.3%) males, 5 (16.6%) showed diabetic response, 5 (16.6%) showed IGT and 12 showed normal responses.

Among 8 (26.6%) females, 2 (6.6%) showed diabetic response, 2 (6.6%) showed IGT and 4 (13.3%) showed normal response.

The diabetic response is seen mainly (2 cases each) seen in the age group 31-40 in males and females. IGT is seen in one case each, in all age groups in males and one each in 41-50 and 61-70 age group in females.
Post necrotic cirrhosis and alcoholic cirrhosis are common (42.86% each) etiology, seen in three cases each (10%) diabetic response. Chronic hepatitis-C is the etiology for diabetic response in one case (14.28%).

Chronic hepatitis-C, Post necrotic cirrhosis-C and alcoholic cirrhosis are the etiology for IGT in two cases (28.5% each) in each. Chronic hepatitis-B and cryptogenic cirrhosis are etiology for IGT in one case (14.2% each) each. Alcoholic cirrhosis and Post necrotic cirrhosis are the common (35.7%) etiologies for impaired glucose response, seen in five (16.6%) cases each. Chronic hepatitis-C is a third stock (21.4%) etiology for impaired glucose response, seen in three (10%) of cases. Cryptogenic cirrhosis causes (7.1%) impaired glucose response in one case (3.3%).

Chronic infection with hepatitis virus C is the most common (50%) etiology for impaired glucose response seen in seven (23.3%) cases. Alcoholic cirrhosis is second common (35.7%) etiology for impaired glucose response seen in five (16.6%). Cirrhosis is the most common (78.57%) cause for impaired glucose response seen in 11 cases. Impaired glucose response in class A (70% /14 cases) and class B (30% /6 cases) Child-Pugh’ classification are 50%/7 cases and 83.3%/ 5 cases, respectively. Chronic hepatitis cause impaired response in 21.4%. Impaired glucose response is typical in females 4 cases (50%) than males 10 cases (45.4%).

Summary and conclusion:-
1. Chronic liver disease is common in males and in low socioeconomic status. Male are more malnourished than females.
2. Cirrhosis is the most common and typical cause of chronic liver disease. Child-Pugh’s grading is useful to know the severity of cirrhosis. Hepatitis C infection is the most typical cause of chronic hepatitis.
3. Among Liver function tests, synthetic functions like serum bilirubin, serum albumin, serum globulin, and prothrombin time are useful in assessing the functional reserve in chronic liver disease. Enzyme estimations like AST, ALT, AP, etc., are helpful in the probable etiology of chronic liver disease. Liver biopsy is useful in confirming the condition and severity.
4. Impaired glucose response is most common in cirrhosis, more common in class B Child-Pugh’s grading. In hepatitis, an impaired response is common in Hepatitis C. Impaired response is more common in females than males. Fasting hypoglycemia can be seen in hepatoma with cirrhosis.
5. Ninety-three cases of chronic liver disease were studied in detail. Data regarding the clinical features and investigations done are present in the master chart.
6. Chronic liver disease is more common in males (73.3%) than females.
7. Chronic liver disease is more common in lower socioeconomic status (63%). Male: female ratio is equal for chronic hepatitis C; it is 1.25 for post necrotic cirrhosis; it is 3 for chronic hepatitis B. Alcoholic cirrhosis, chronic alcohol hepatitis, and primary hepatoma with cirrhosis is seen only in males, and cryptogenic cirrhosis is seen only in female. Among cases males have lower BMI (50.09%), compared to females (35.7%) are malnourished.
8. Anorexia, fatigue, and distention of abdomen are common symptoms, seen in 80%, 70%, and 50%, respectively. History of alcoholism is most common (40%). Others like blood transfusion, extramarital sex, childhood history, etc. also contribute significantly to development of chronic liver disease. Ascites is the most common (53.3%) sign; hepatomegaly, icterus, and clubbing are also common.
9. Most of the cases are anaemic (mean-9.97mg%), 63% of males are anaemic, and 50% of females are anemic.
10. In peripheral smear, most of them show anemia of chronic disease (normocytic normochromic anemia).
11. 66.6% (20 cases) showed normocytic normochromic response rest showed microcytic hypochromic response, because of inadequate intake or chronic blood loss through varices.
12. Cirrhosis is the most common cause (66.6% /20 cases) for chronic liver disease, with post necrotic cirrhosis and alcoholic cirrhosis causing 50% each. Child-Pugh’s grading is very useful to know the severity of cirrhosis. Among cirrhosis, 70% were in class-A, and 30% were in class-B. Class-B cirrhotics are associated with hepatic encephalopathy in 20% and esophageal varices in 16.6%. Among hepatitis virus infection, Hepatitis C cause majoritv (36.6%) of chronic liver disease.
13. Thirty patients of cured respiratory tract infection (I/P/O/P) were studied in detail and investigated; age and sex-matched were taken as control group.
14. Those who have given volunteered and gave written consent for OGTT were subjected to OGTT, venous blood, and urine was serially analyzed for sugar at fasting, 1/2hr, 1hr, 1 1/2hr, and 2hr. Data according to WHO criteria (Table 3 & 4) is collected and compare between case and control groups using ‘paired T’ test to know the significance.
15. There is a significant (p-0.00001) rise in blood sugar levels in cases compared to control group. After glucose load and there was diabetic response in 23.3% cases and IGT in 23.3% cases.
16. Cirrhosis is the most common (78.57%) cause for impaired glucose response. There is a correlation between impaired glucose response and Child-Pugh’s classification (83.3% in class-B and 50% in class-B). Chronic hepatitis cause impaired response in 21.4%. Impaired glucose response is typical in females (50%) than males (45.4%). Fasting hypoglycemia is seen in the case of cirrhosis with hepatoma.