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Assessment of Lipid Profile in Patients with Chronic liver disease in children Welfare Teaching Hospital / Medical City

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Abstract

Background: Liver plays a central role in lipid metabolism, Alteration of lipid metabolism are well defined in liver disease. It is important to assess severity of chronic liver disease since management varies accordingly,
Aim of Study: The aim of the study is to assess changes that occur in lipid parameter (low-density lipoprotein, high-density lipoprotein, serum triglyceride and total serum cholesterol) levels in relation to severity of chronic liver diseases.
Patients and Method: An analytical cross-sectional study, was carried out for 100 patients from age of 2 months up to 12 years old in Children Welfare Teaching Hospital and gastrointestinal center outpatient in medical city who were complaining from chronic liver disease , another group of fifty normal healthy children as a control group and seven cases were excluded, from the period of 1st of May 2019 to the 30th of November 2020, patients were assessed by proper history, physical examination and Fasting blood sample were aspirated after consent for all patient and control group enrolled in study to investigate the serum lipid profile which include total cholesterol (TC), Triglyceride (TG),), high-density lipoprotein (HDL) and low-density lipoprotein(LDL).
All the patient and control group results were submitted to the PELD calculator and PELD score was estimated
Results: In this study the total number of patient was 100 diagnosed with chronic liver disease of different etiologies and 50 healthy as a control group, with maximum age of 12 years and minimum age of 2 months .There were 57 (57%) males and 43 (43%) females in diseased group, according to their diagnosis : 44 patients Progressive familial intrahepatic cholestasis (27%), 17 patients Metabolic Liver Disease (17%) , 12 patients cytomegalovirus (12%), 10 patients Biliary atresia (10%), 9 patients Hepatitis c (9%), 9 patients Wilson disease (9%) , 2 patients Idiopathic neonatal hepatitis (2%), 14 patients Undiagnosed chronic liver disease (14%).
Forty-nine patients their pediatric end stage liver disease score was < 11, 39 patients their pediatric end stage liver disease score was 11 – 18.
Six patients were 19 – 24, and same (6 patients) have pediatric end stage liver disease score ≥ 25.
there were 62 (62%) of patients having growth failure and 38 (38%) were in the normal growth according to distribution of growth chart.
Regarding serum cholesterol and serum triglyceride there was no significant difference between both groups, while both LDL and HDL showed significant low level between disease and healthy group.
Conclusion: There is decrease in lipid parameters (low density lipoprotein, high density lipoprotein) in disease group in comparison with control group
And both TG and LDL were had significantly lower than the mean in under growth group.

Keywords: Chronic liver disease; Children; Lipid profile; Lipoproteins; Pediatric end-stage liver disease (PELD) score; Growth failure.

1. Introduction

Liver disease in paediatric age group is one of the most significant causes of morbidity and mortality and includes a broad spectrum of disorders including; infectious, metabolic, genetic, drug induced, idiopathic, structural and autoimmune diseases and affect the economy of the family as a result of premature death, illness, and disability [(1)].
Chronic liver disease is determined either by duration of liver disease (typically >3–6 mo) or by evidence of either severe liver disease or physical stigmata of chronic liver disease (clubbing, spider telangiectasia, hepatosplenomegaly) [(1)].
liver contributes both in exogenous and endogenous cycles of lipid metabolism and transport of lipids through plasma, Synthesis of many apolipoproteins takes place in liver [(2)]. Hepatocytes play a vital role in regulating lipid metabolism; liver is considered the primary site for cholesterol and lipoprotein synthesis [(3)]. In healthy organisms, a complex balance is maintained between the biosynthesis, utilization and transport of lipid fractions, so in cirrhosis the lipid metabolism is altered such that glycogen reserves are substantially decreased, inducing lipolysis and malnutrition [(3)].
Previous studies have shown that patients with cirrhosis have an altered lipid metabolism, in particular hypocholesterolemia and hypo beta lipoproteinemia [(3)], Alterations of serum lipid profile is a common observation in chronic liver disease, Lipids are essential component of biological membranes, free molecules and metabolic regulators that control cellular function and homeostasis, Liver plays a central role in lipid metabolism as several pathways are at least in part dependent to this site[(2)].

Major metabolic processes take place at this level involving the production transportation and storage of apoproteins and lipoproteins as well ascatabolism of various lipids and excretion of cholesterol and phospholipids and alteration in liver function resulting from cellular injury leads to change in the serum concentration of cholesterol and lipoproteins [(2)].
Such modifications, evolve along with liver disease progression and can be used as a prognostic indicator for decompensated CLD [(4,5)]
many mechanisms involved in the reduction of lipid fractions in cirrhotic patients, all are complex and will still require many studies to understood, Enzymatic (acylCoA: cholesterol acyltransferase ACAT), protein (microsomal triglyceride transfer protein - MTP) and apoprotein (Apo AI) reductions are thought to be related to such changes [(6,7,8).]
The apolipoproteins are required for structure of lipoproteins, Lipoproteins play an important role in the absorption of dietary cholesterol, long chain fatty acids and fat soluble vitamins. The transport of triglycerides, cholesterol and fat soluble vitamins from the liver to peripheral tissue and transport of cholesterol from peripheral tissue to liver is by lipoproteins [(9)]. Apolipoproteins activate enzymes important in lipoprotein metabolism and to mediate the binding of lipoproteins to cell surface receptors. Liver is the principal site of formation and clearance of lipoproteins. This shows liver is involved in many steps of lipid metabolism and lipid transport. Thus, in severe liver disease lipid metabolismis profoundly disturbed [(9)].

Aims Of The Study:

The aim of the study is to assess changes that occur in lipid parameter (low-density lipoprotein (LDL), high-density lipoprotein (HDL), serum triglyceride and total serum cholesterol) levels in patients with chronic liver disease and assess correlation with the severity.

Patient and Methods

This cross-sectional study was done including one hundred and fifty-seven Children, taken from Welfare children Teaching Hospital and gastrointestinal center outpatient in medical city, from the period of 1st of May 2019 to the 30th of November 2020. One hundred inpatient with chronic liver disease regardless of the causes were included in this study (Chronicity of liver disease is determined either by duration of liver disease (typically >3–6 mo) or by evidence of either severe liver disease or physical stigmata of chronic liver disease (clubbing, spider telangiectasia, hepatosplenomegaly*)* , in addition to fifty healthy child sex- and age-matched used as control cases was taken from outpatient clinic suffering from simple flu, tonsillitis and seven cases were excluded from this study. age range of patient were included in this study was from 2 month to 12 years.

Data including age, sex, for all patients were recorded, patients were assessed by proper history, physical examination and investigations (LFT, serum albumin, PT, PTT, INR) which were aspirated after taking the patient and control family consent, also 3 ml of blood was collected in special tube (EDTA tube) by using the Randox kit, enrolled in study to investigate the serum lipid profile which include total cholesterol (TC), Triglyceride (TG), HDL and LDL.

PELD score was calculated to all cases depending on their age, gender, growth parameter (height and weight) and laboratory investigations results (TSB level, albumin, INR).

Exclusion Criteria

All patient with diseases and factors causing lipid profile derangement including diabetes mellitus, renal problem like nephrotic syndrome, thyroid dysfunction, familial dyslipidemia and taking drugs which might affect blood lipids and lipoproteins were excluded from study.

Statistical Analysis

Excel sheet, SPSS version (25) was used for data analysis, descriptive statistic, frequencies, percentages, associations, tests of significance (t test, ANOVA) was used for analysis of quantitative- continuous variables. Means and standard deviations were used to present data of continuous variables. Correlation was performed to recognize the independent predictors. Chi-square, fissure exact test for qualitative variables, where P-value < 0.05 was considered statistically significant.

Results

Demographic Characterization

The mean age of diseased group was 3.5 ± 3.7 years with maximum age of 12 years and minimum age of 2 months.

There were 57 (57%) males and 43 (43%) females in diseased group, while the male representing (58%) and female representing (42%) of control group.

The mean of weight, height, and head circumference for disease group were 13.2 ± 10.4 Kg, 79.8 ± 25.3 cm, and 45.48 ± 6.5 cm respectively. The mean of weight, height, and head circumference for disease group were significantly lower in comparison to the mean of control group (p value = 0.011, 0.001, and 0.042 respectively). Table 4.

Table 4: Comparisons of weight, height, and head circumference between diseased and control group.

		N	Mean	SD	P value
Weight /Kg	Disease	100	13.28	10.42	0.011
	Control	50	17.88	10.01	
Height /cm	Disease	100	79.81	25.36	0.001
	Control	50	98.30	25.66	
Head Circumference /cm	Disease	100	45.48	6.53	0.042
	Control	50	47.76	6.17	

By applying the growth chart for all disease group patients after measuring the weight for age and height, there were 62 (62%) of patients having growth failure and 38 (38%) were in the normal distribution of growth chart.

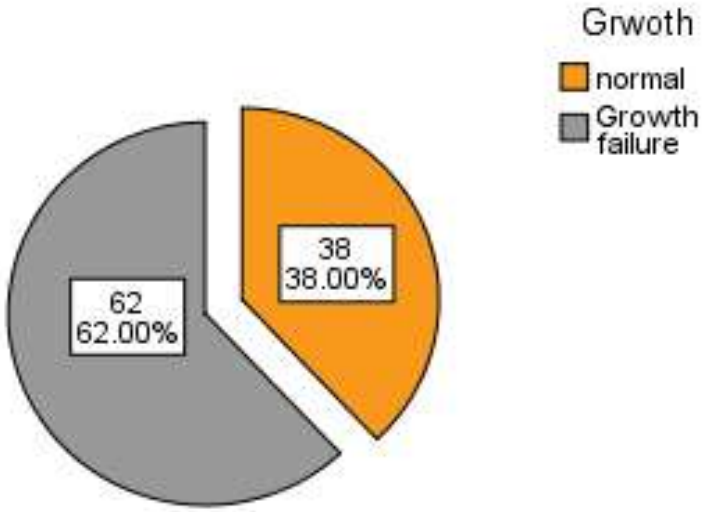


Figure 4: *Growth failure distribution across diseased group*

Diagnosis of Chronic Liver Disease

Progressive familial intrahepatic cholestasis was the most frequent diagnosis of chronic liver disease in our study 27 (27%), this was followed by metabolic liver disease 17 (17%) diagnosed patients, 12 (12%) patients were diagnosed with CMV, biliary atresia diagnosis was counted in 10 (10%) of patients, 9 (9%) of patients diagnosed with hepatitis C virus and same for Wilson disease, and 2 (2%) patients were diagnosed with idiopathic neonatal hepatitis. There were 14 (14%) patients undiagnosed for chronic liver disease.

Table 5: Frequency Distribution of diagnosis of chronic liver disease

Diagnosis	Frequency	Percent
Progressive familial intrahepatic cholestasis	27	27%
Metabolic Liver Disease	17	17%
CMV	12	12%
Biliary atresia	10	10%
Hepatitis c	9	9%
Wilson disease	9	9%
Idiopathic neonatal hepatitis	2	2%
Unknown etiology	14	14%

The PELD Score

The mean of PELD score for disease group was 9.85 ± 8.92, with highest value of 33 and lowest value -8. Categorizations of PELD score showed 49 patients have PELD score of <11, 39 patients have PELD score of 11 – 18, 6 patients have PELD score of 19 – 24, and same (6 patients) have PELD score of ≥ 25.

Table 6: The distribution of PELD score

PELD score	Frequency and Percentage
< 11	49 (49%)
11 – 18	39 (39%)
19 – 24	6 (6%)
≥ 25	6 (6%)

Association between PELD score and growth

The patients with PELD score of < 11, 31 (31%) of them were have normal growth and 18 (18%) of them have growth failure. For patients with PELD score of 11 – 18, 3 (3%) of them were have normal growth and 36 (36%) of them have growth failure. For patients with PELD score of 19 – 24, 1 (1%) of them have normal growth and 5 (5%) of them have growth failure. For patients with PELD score of ≥25, there were 3 (3%) patients have normal growth and 3 (3%) patients have growth failure. There was a significant association between high score of PELD and growth failure. Table 7.

Table 7. The distribution of PELD score across growth failure

		PELD Score				Total
		< 11	11 - 18	19- 24	≥ 25	
Growth	Normal	31 (31%)	3 (3%)	1 (1%)	3 (3%)	38 (38%)
	Failure	18 (18%)	36 (36%)	5 (5%)	3 (3%)	62 (62%)
Total		49 (49%)	39 (39%)	6 (6%)	6 (6%)	100

P value< 0.0001

Lipid Profile

The mean of total cholesterol for diseased group was 144.1 ± 83 mg/dl and for control groups it was 158.4 ± 29 mg/dl. There was no significant difference between both groups (p value = 0.21).
The mean of TG for diseased group was 151 ± 167 mg/dl and for control groups it was 131 ± 22 mg/dl. There was no significant difference between both groups (p value = 0.39).
The mean of LDL for diseased group was 90 ± 50 mg/dl and for control groups it was 114 ± 22 mg/dl. There was statistical significant lower difference between diseased and control groups (p value = 0.002).
The mean of HDL for diseased group was 52 ± 42 mg/dl and for control groups it was 71 ± 22 mg/dl. There was statistical significant lower difference between diseased and control groups (p value = 0.002).

Table 8: Mean of lipid profile distribution across disease and control

Group		Mean	SD	Maximum	Minimum	P value
Cholesterol	Disease	144	83	533	5	0.21
	Control	158	30	241	100	
TG	Disease	151	167	1471	18	0.39
	Control	131	22	178	99	
LDL	Disease	90	50	281	5	0.002
	Control	114	22	189	78	
HDL	Disease	52	42	189	4	0.002
	Control	71	22	112	30	

Association of lipid profile and growth.

The association between lipid profile and growth was given in table 12. Both TG and LDL were have significantly lower mean in under growth group in comparison to normal growth group of patients, while no significant difference in HDL between normal and under growth group.

Table 11: The association of lipid profile with growth category

	Normal Growth		Under Growth		P value
	Mean	SD	Mean	SD	
Cholesterol	152	88	139	80	0.35
TG	181	240	133	99	0.03
LDL	97	67	86	36	0.001
HDL	41	37	59	44	0.19

Discussion

Assessment of lipid profile in children with chronic liver disease is important to demonstrate the effect of chronic liver disease on children's lipid profile in our country and evaluate the factors that emerge from the change of lipid profile. The mean of age of diseased group was 3.5 ± 3.7 years with maximum age of 12 years and minimum age of 2 months. The mean of age was comparable to other study conducted by Hala and Rabab in Iraq, [23] and was lower in comparison

to global studies that showed the mean of age was ranging from 5 – 11 years [24,25]. This disparity might relate to the severity of chronic liver disease among Iraqi children. [23,24]

In this study, the growth failure was observed in 62 (62%) of patients, which was higher than a study conducted in Indonesia that showed only 38% of patients have growth failure, [26] This difference between our study and the Indonesian one may be related to the difference in sample size that was low in Indonesian (21 patients). While the Incidence for growth failure was higher in chronic liver disease among children who prepared for orthotropic liver transplant, [27] and this high prevalence may related in fact to pool of patients that investigated in that study that focused on patients who prepared for orthotropic liver transplant only and those patients have severe liver disease that need liver transplant.
Progressive familial intrahepatic cholestasis was the most frequent diagnosis of chronic liver disease in our study 27 (27%) and this was followed by metabolic liver disease 17 (17%), while in Hala and Rabab
Studies [23], the PFIC was the most common in children below 2 years of age and the most common diagnosis in their study was glycogen storage disease.

A study from Egypt have relatively different etiology for chronic liver disease, they demonstrated that the chronic hepatitis responsible for more than third of cases of chronic liver disease in Egypt, in turn, the PFIC was responsible for only 12% of their chronic liver failure children and it was in third place after glycogen storage disease,[28]In our study, the chronic hepatitis was responsible in nearly 19% of our patients, as hepatitis is more common in Egypt than Iraq

A study from East Asia concluded, viral hepatitis is the leading cause of chronic liver disease in children, with the highest incidence of chronic Hepatitis C followed by metabolic disorders and biliary atresia [29].
An epidemiology study from USA showed that biliary atresia is the most common of pediatric liver disease and intrahepatic cholestasis came in third place after metabolic causes [30]. Also, this was relatively and old study and current diagnostic procedure plan an important in the increase of PFIC diagnosis.

Lipid Profile

The lipid metabolism is profoundly disturbed in chronic liver disease. Normally, the lipid profile is at lowest level at birth and increased significantly during first year of life, there after showing a much slower rise in levels. [31]
In our study, the mean of total cholesterol for diseased group was 144.1 ± 83 mg/dl and for control groups it was 158.4 ± 29 mg/dl. There was no significant difference between both groups (p value = 0.21). Which were comparable with study conducting in Turkey demonstrated same results of no difference in cholesterol level between diseased and healthy group. [32].

Both LDL and HDL were have significant level difference between disease and healthy group, and also this demonstrated in study mentioned above.American College of Cardiology/American Heart Association emphasis that, the lower HDL level have no harmful effect on child health and 50% children with low HDL have normal level as adult[33].

The distribution of gender was equal across total cholesterol, TG, LDL, and HDL dyslipidemia and the American College oCardiology/American Heart Association demonstrated no effect of gender on dyslipidemia [33].
The assessment of coagulation status across the level of lipid profile showed that there was no significant association between lipid profile and coagulation status for patients with chronic liver.

Both TG and LDL were have significantly lower mean in under growth group in comparison to normal growth group of patients. Also, in turn, the liver failure led to disturbance in its normal production of lipids and liver failure have direct effect on growth, so with advance liver disease the growth, lipid, coagulation state be affected [37,38,39].

The PELD Score

The mean of PELD score for disease group was 9.85 ± 8.92, with highest value of 33 and lowest value -8. Our results was in comparable to a study conducted in USA. [40]
Categorizations of PELD score showed 49(49%) patients have PELD score of < 11, 39(39%) patients have PELD score of 11 – 18, 6(6%) patients have PELD score of 19 – 24, and same 6 patients (6%) have PELD score of ≥ 25,

There was a significant association between high score of PELD and growth failure, also the high PELD score could be predictor for mortality of patients with chronic liver diseases. [41].

In this study, the relationship between the PELD score and lipid profile has not been demonstrated. This relation was demonstrated in adult patients in many studies, [42,43] So, the severity of chronic liver disease in pediatric did not have an effect on lipid profile in our patients.

Conclusions

From this study, it was found:

1. decrease in lipid parameter (low-density lipoprotein, high-density lipoprotein) in disease group, And There is no significant difference in (serum triglyceride and total cholesterol serum) between disease and control group.
2.There was significant association between younger age and high level of total cholesterol, TG, and HDL
3.The distribution of cholesterol, TG, LDL, and HDL across PELD score were equal and there was no significant difference in distribution

References

1.Ahmad M, Afzal S, Roshan E et al. Use fulness of needle biopsy in the diagnosis of pediatric liver disorders. J Pak Med Assoc 2005;24-8.
2.Mehboob F, Ranjha FA, Masud S. Changes in Serum Lipid Profile Among Patients Suffering From Chronic Liver Disease. Annals of King Edward Medical University. 2007;13(3):209-.
3.Bassendine MF, Sheridan DA, Bridge SH, Felmlee DJ, Neely RD. Lipids and HCV. Semin Immunopathol. 2013;35(1):87-100
4. Ghadir MR, Riahin AA, Havaspour A, Nooranipour M, Habibinejad AA. The relationship between lipid profile and severity of liver damage in cirrhotic patients. Hepat Mon. 2010;10(4):285-8.
5. Selcuk H, Uruc I, Temel MA, Ocal S, Huddam B, Korkmaz M, et al. Factors prognostic of survival in patients awaiting liver transplantation for end-stage liver disease. Dig Dis Sci.2007;52(11):3217-23
6. Jiang M, Liu F, Xiong WJ, Zhong L, Xu W, Xu F, et al. Combined MELD and blood lipid level in evaluating the prognosis of decompensated cirrhosis. World J Gastroenterol. 2010;16(11):1397-401.
7. Nashaat EH. Comparative study of serum lipid profile between chronic hepatitis C Egyptian patients and normal controls and the effect of viral eradication on lipids profile. Report and Opinion. 2010;2:14-20
8. Tsai MH, Peng YS, Chen YC, Lien JM, Tian YC, Fang JT, et al. Low serum concentration of apolipoprotein A-I is an indicator of poor prognosis in cirrhotic patients with severe sepsis. J Hepatol.2009;50(5):906-15
9. Wisniewska-Ligier M, Wozniakowska-Gesicka T, Kups J, Sulat- Syncerek D. Lipid metabolism in children with chronic hepatitis C, A preliminary report. Hepato-gastroenterology. 2006;53(72):887.
10. Stacey S. Huppert, William F. Balistreri. Morphogenesis of the Liver and Biliary System. In: Kliegman, ST geme, Blum, Shah, Tasker,Wilson ed. *Nelson textbook of pediatrics. ed 21.* Philadelphia, Elsevier health sciences;2020. p8240-8253
11. Naresh P. Shanmugam and Anil Dhawan. Complications of Cirrhosis in Children.In: Stefano, Guandalini, David Branski ed. *Textbook of pediatric gastroenterology, hepatology and nutrition. 1st ed. New York, Springer; 2016. P 821-838*.
12. Powell D.W., Approach to the patient with liver disease, Arisz, Arend WP editors. Cecil text book of medicine, 22 ed., Philadelphia: Saunders 2004; p1435.
13. Noelle H. Ebel and Simon P. Horslen.Complications and Management of Chronic Liver Disease. In: Deirdre A. kelly ed. *Diseases of the Liver and Biliary System in Children*. Ed 4th. West Sussex, Wiley Blackwell; 2017. P 344-366.
14. James E. Squires, William F. Balistreri. Manifestations of Liver Disease. In: Kliegman, ST geme, Blum, Shah, Tasker, Wilson ed. *Nelson textbook of pediatrics. ed 21.* Philadelphia, Elsevier health sciences;2020. P 8254-8266.
15.Yang CH, Perumpail BJ, Yoo ER, Ahmed A, Kerner Jr JA. Nutritional needs and support for children with chronic liver disease. Nutrients. 2017 Oct;9(10):1127.
16. Chang CC, Bryce CL, Shneider BL, Yabes JG, Ren Y, Zenarosa GL, Tomko H, Donnell DM, Squires RH, Roberts MS. Accuracy of the pediatric end-stage liver disease score in estimating pretransplant mortality among pediatric liver transplant candidates. JAMA pediatrics. 2018 Nov 1;172(11):1070-7.
17. HUI TW. Anesthesia for Pediatric Liver Transplantation. Living Donor Liver Transplantation. 2011 Mar 8:181.

18. Deirdre Kelly. Chronic liver disease: itching. In: Deirdre Kelly, Ronald Bremner, Jane Hartley, Diana Flynn ed. *Practical Approach to Paediatric Gastroenterology, Hepatology and Nutrition*. 1st ed. West Sussex, Wiley-Blackwell; 2014. P 151-154.
19. (http://healthy-ojas.com/cholesterol/fat-metabolism.html) Thiruvelan on Wed, 2010-06-23 18:52.
20. Lee A. Pyles, William A. Neal. Disorders of Lipoprotein Metabolism and Transport. In: Kliegman, ST geme, Blum, Shah, Tasker, Wilson ed. *Nelson textbook of pediatrics. ed21.Philadelphia*, Elsevier health sciences;2020. P3359-3401
21.Noelle H. Ebel and Simon P. Horslen. Complications and Management of Chronic Liver Disease. In: Deirdre Kelly.*Diseases of the Liver and Biliary System in Children*.4th ed. West Sussex, Wiley Blackwell; 2017. P 343-366.
22.Yang CH, Perumpail BJ, Yoo ER, Ahmed A, Kerner Jr JA. Nutritional needs and support for children with chronic liver disease. Nutrients. 2017 Oct;9(10):1127.
23.Arif HS, Thejeal RF. Etiology of chronic liver disease in Iraqi children, with special emphasis on the role of liver biopsy. Pak J Med Sci. 2011 Jul 1;27(4):870-73.
24.Dhole SD, Kher AS, Ghildiyal RG, Tambse MP. Chronic Liver Diseases in Children: Clinical Profile and Histology. J ClinDiagn Res. 2015 Jul;9(7):SC04-7.
25. A Kansu, AKalayci, B Altuntas, T Doganci, N Girginn. Autoimmune Hepatitis in Children, a Report of Ten Cases. Turkey J Med Sci. 2000;30:55–61.
26.Widodo AD, Soelaeman EJ, Dwinanda N, Narendraswari PP, Purnomo B. Chronic liver disease is a risk factor for malnutrition and growth retardation in children. Asia Pacific Journal of Clinical Nutrition. 2017;26(Supplement): S57.
27. Wolfgang Högler PD, Baumann U, Kelly D. Growth and bone health in chronic liver disease and following liver transplantation in children. Pediatric Endocrinology Reviews. 2010 Mar;7(3):266.
28. Barakat SH, El-Gendy W, Metwaly M*, et al*. Chronic liver diseases in children:an aetiological study. *Archives of Disease in Childhood* 2008;93: pw565.
29. Tahir A, Malik FR, Ahmad I, Krishin J, Akhtar P. Aetiological factors of chronic liver disease in children. Journal of Ayub Medical College Abbottabad. 2011 Jun 1;23(2):12-4.
30. Arya G, Balistreri WF. Pediatric liver disease in the United States: epidemiology and impact. Journal of gastroenterology and hepatology. 2002 May;17(5):521-5.
31.Chandar V, Gidvani CH, Gupta AK, Wilson CG, Sharma YV. Lipid profile in normal healthy children. Med J Armed Forces India. 1994 Apr;50(2):101-104.
32.SelimoĞlu MA, AydoĞdu S, YaĞci RV. Lipid parameters in childhood cirrhosis and chronic liver disease. Pediatrics international. 2002 Aug;44(4):400-3.
33.Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, Braun LT, de Ferranti S, Faiella-Tommasino J, Forman DE, Goldberg R. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2019 Jun 17;73(24):e285-350.
34.Dowla S, Aslibekyan S, Goss A, Fontaine K, Ashraf AP. Dyslipidemia is associated with pediatric nonalcoholic fatty liver disease. Journal of clinical lipidology. 2018 Jul 1;12(4):981-7.
35.Flisiak-Jackiewicz M, Lebensztejn DM. Update on pathogenesis, diagnostics and therapy of nonalcoholic fatty liver disease in children. Clinical and Experimental Hepatology. 2019 Mar;5(1):11.
36.Fang YL, Chen H, Wang CL, Liang L. Pathogenesis of non-alcoholic fatty liver disease in children and adolescence: From "two hit theory" to "multiple hit model". World journal of gastroenterology. 2018 Jul 21;24(27):2974.
37.Nandivada P, Carlson SJ, Chang MI, Cowan E, Gura KM, Puder M. Treatment of parenteral nutrition-associated liver disease: the role of lipid emulsions. AdvNutr. 2013 Nov 6;4(6):711-7.
38.Larson-Nath C, Goday P. Malnutrition in children with chronic disease. Nutrition in Clinical Practice. 2019 Jun;34(3):349-58.
39.Courtney CM, Warner BW. Pediatric intestinal failure-associated liver disease. Current opinion in pediatrics. 2017 Jun 1;29(3):363-70.
40.Chang CC, Bryce CL, Shneider BL, Yabes JG, Ren Y, Zenarosa GL, Tomko H, Donnell DM, Squires RH, Roberts MS. Accuracy of the pediatric end-stage liver disease score in estimating pretransplant mortality among pediatric liver transplant candidates. JAMA pediatrics. 2018 Nov 1;172(11):1070-7.
41.Bolia R, Srivastava A, Yachha SK, Poddar U. Pediatric CLIF-SOFA score is the best predictor of 28-day mortality in children with decompensated chronic liver disease. Journal of hepatology. 2018 Mar 1;68(3):449-55.
42.Ghadir MR, Riahin AA, Havaspour A, Nooranipour M, Habibinejad AA. The relationship between lipid profile and severity of liver damage in cirrhotic patients. Hepat Mon. 2010;10(4):285-8.
43.Muhammed HP, Jayaraj K. Correlation of lipid profile in patients with severity of liver disease: A cross-sectional study in a tertiary care hospital. Int J Res Med Sci. 2017 Jan;5:326-9.