

## HYPERTRIGLYCERIDEMIA: A COMMON INCIDENTAL FINDING IN YOUNG INDIAN ADULTS – A CASE SERIES

Afreen Arshad Choudhry<sup>1</sup>, Ketki Khandhadiya<sup>2</sup>, Shailaza Shrestha<sup>3</sup>, Rana Gopal Singh<sup>4</sup>

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Corresponding Author:  
**Dr. Afreen Arshad Choudhry,**  
Email: afreen185@gmail.com

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<sup>1</sup>Assistant Professor, Department of Biochemistry, Autonomous State Medical College, Gonda, Uttar Pradesh, India

<sup>2</sup>Professor and Head, Department of Biochemistry, Baba Kinaram Autonomous State Medical College, Chandauli, Uttar Pradesh, India.

<sup>3</sup>Associate Professor, Department of Biochemistry, Heritage Institute of Medical Sciences, Varanasi, Uttar Pradesh, India.

<sup>4</sup>Former Principal and Professor Emeritus, Department of General Medicine, Heritage Institute of Medical Sciences, Varanasi, Uttar Pradesh- India.

### ABSTRACT

Hypertriglyceridemia (HTG) is a common abnormality that is often seen in the laboratory setting. Chronic hypertriglyceridemia is not an isolated condition and apart from being linked to metabolic syndrome; it is found to have a strong association with Coronary artery disease. Taking into account the increased incidence of acute myocardial infarction in young Indian adults, it becomes important to throw some light onto CVD in young Indians. Here with we have documented 6 cases reported to have an incidental finding of hypertriglyceridemia in the diagnostic laboratory of a tertiary care center over a period of 8 months.

### INTRODUCTION

Hypertriglyceridemia (HTG) is a common abnormality that is often seen in the laboratory setting. Increased cases of Hyperlipidemia along with obesity are documented owing to a spectrum of causes like lifestyle, chronic stress, unhealthy diet, intake of certain medications or presence of metabolic or hormonal disorders like Type 2 Diabetes Mellitus and Hypothyroidism. In a small fraction of individuals; hyperlipidemia can be caused due to genetic factors as well.<sup>[1,2]</sup>

HTG is defined as an abnormally increased concentration of triglycerides in blood. The clinical guidelines like the NCEPATP III, American Heart Association, National Lipid Association, The Endocrine Society and European society of Cardiology have concluded that a triglyceride level >150 mg/dL is defined as Hypertriglyceridemia [Table 1].

Severe hypertriglyceridemia (TG> 1000mg/dl) is documented to be a significant risk factor for recurrent acute pancreatitis. Chronic hypertriglyceridemia is not an isolated condition and apart from being linked to metabolic syndrome; it is found to have a strong association with Coronary Artery Disease.<sup>[3-9]</sup>

Taking into account the increased incidence of acute myocardial infarction in young Indian adults, it becomes important to throw some light onto CVD in

young Indians. Here with we have documented 6 cases reported to have an incidental finding of hypertriglyceridemia in the diagnostic laboratory of a tertiary care center over a period of 8 months. It is important to emphasize that these cases did not attend the Outpatient clinic due to any cardiovascular complaint and had visited for a routine checkup or an unrelated complaint.

### Methods of laboratory analysis

For fasting plasma glucose (FPG), 2ml of blood was collected in grey top (oxalate-fluoride) vacutainers after 12 hours of fasting. For Lipid profile, Liver function tests and Renal function tests analysis, 8 hours fasting sample was used in red top (no additives) vacutainers.

FPG was analyzed enzymatically based on GOD-POD method. Total cholesterol, Triglycerides and HDL were analyzed enzymatically using kits manufactured by Chema. Urea was analyzed using kits based on Urease- GLDH method, creatinine by modified Jaffe's method. SGOT and SGPT were analyzed using enzymatic colorimetric assay. All the tests were performed using kits manufactured by Chema on the Dirui-300 B autoanalyzer. In case of HTG, the serum was diluted to perform the investigations to overcome the lipemic interference.

## CASE DESCRIPTIONS

### Case 1

A 38-year-old male came for routine screening to the hospital and was advised to have his routine blood chemistry investigations done. Laboratory results revealed Hypertriglyceridemia. On enquiry, he stated that he walked 20 minutes daily, was a bank employee by profession. His Fasting Plasma Glucose and BMI was normal. History revealed that his father is diabetic for 15 years, is a heart patient and had suffered first heart attack at the age of 52. Laboratory investigations are shown in Table 2.

### Case 2

A 38-year-old male visited the medicine OPD with a complaint of sore throat, fever and cough. He was advised treatment accordingly but was asked to have his routine investigations done. This was because the patient stated that he had not got any blood investigations done previously. Laboratory investigations revealed HTG. During discussion, he mentioned that he was a smoker and was an occasional drinker. He did not mention any significant family history. He was a school teacher by profession and did not get much time for regular exercise due to his personal and professional commitments. Laboratory investigations are shown in Table 2.

### Case 3

A 34-year-old male visited the medicine OPD with complaints of frequent episodes of headache which exacerbated in the evening. He was referred to the ophthalmology department for examination. History revealed that he did at times feel fatigued and had breathlessness after long walks. He mentioned a family history of heart disease and Diabetes Mellitus. On account of this, the medicine consultant checked

his blood pressure which was found to be raised and therefore advised him to get his routine blood chemistry analysis done. The investigations revealed HTG. The ECG, on the other hand, did not show any significant changes. Laboratory investigations are shown in Table 2.

### Case 4

A 34-year-old male visited the medicine OPD with complaints of frequent episodes of indigestion, vomiting and abdominal pain after having meals. He was advised to get his routine investigations done which revealed HTG. Apart from the routine chemistry, serum amylase and lipase were also analyzed which were found to be within normal limits. Laboratory investigations are shown in Table 2.

### Case 5

A 36-year-old male had his blood sugar, lipid profile, liver function and kidney function analyzed as part of his routine health checkup. The results revealed HTG and he was advised by the Biochemistry consultant to visit the Medicine OPD with the reports. History revealed that he was a chronic smoker. Laboratory investigations revealed HTG and are shown in Table 2.

### Case 6

An elderly diabetic nephropathy patient visited the medicine OPD for her routine checkup accompanied by her son. Upon discussion; the elderly woman mentioned that her 35 years old son was a farmer by profession and had not been in the best of his health in the recent past. He mainly had episodes of breathlessness when he returned from field work. The medicine consultant suggested that he gets his routine biochemistry tests done along with ECG. ECG did not show any significant changes. Laboratory investigations revealed HTG and are shown in Table 2.

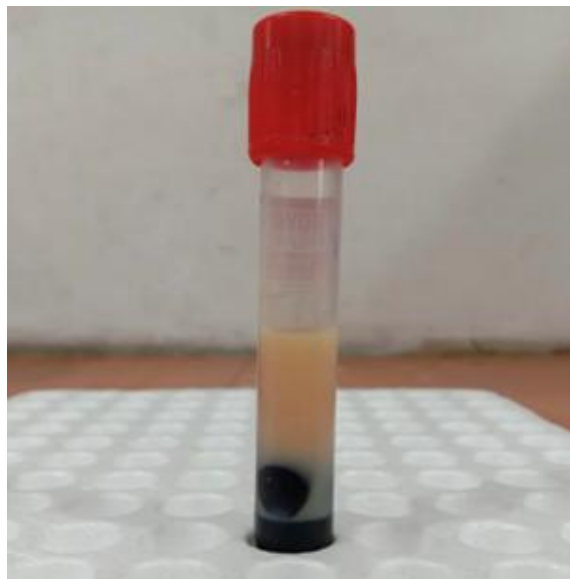
**Table 1: Definition of Hypertriglyceridemia as per Various Clinical Guidelines.**

Guideline	Classification	Serum Triglyceride levels
NCEP/ ATP III. <sup>[3]</sup> American Heart Association. <sup>[4]</sup> National Lipid Association. <sup>[5]</sup>	Normal Borderline-high TGs High TGs Very high TGs	150 mg/dL 150-199 mg/dL 200-499 mg/dL ≥500 mg/dL
The Endocrine Society. <sup>[6]</sup>	Normal Mild HTG Moderate HTG Severe HTG Very severe HTG	<150 mg/dL 150-199 mg/dL 200-999 mg/dL 1000-1999 mg/dL ≥2000 mg/dL
European Society of Cardiology. <sup>[7]</sup>	Normal Mild-moderate HTG Severe HTG	<150mg/dL 1.7<10mmol/L > 10 mmol/L

**Table 2: Demographic characteristics and Laboratory investigations of the cases.**

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
<b>Demographic characteristics</b>						
Age	38	38	34	40	36	35
Gender	M	M	M	M	M	M
F h/o T2DM	Y		Y			Y
Smoker (Y/N)	N	Y	N	N	Y	N
Alcoholic (Y/N)	N	Y	N	N	N	N
<b>Laboratory investigations</b>						
Hemoglobin (g/dl)	12.6	13.8	11.6	13.2	14.7	13.5
FPG (mg/dl)	112	98	120	86	99	120
Total cholesterol (mg/dl)	174.7	168.4	182	152	125	165

HDL (mg/dl)	32.2	34.8	36.9	42	39	31.6
Triglycerides (mg/dl)	446.7	512.7	449	560	420	487
Urea (mg/dl)	20	40	36	43	26	39
Creatinine (mg/dl)	0.7	1.1	0.9	0.8	1.0	0.8
SGOT (U/L)	36	24	32	55	36	28
SGPT (U/L)	50	43	38	62	41	39
Amylase (U/L)	--	--	--	Within normal limits	--	--
Lipase (U/L)	--	--	--	Within normal limits	--	--



**Figure 1: Lipemic sample due to Hypertriglyceridemia.**

## DISCUSSION

HTG is one of the most commonly encountered form of dyslipidemia seen in clinical practice. HTG can be due to causes that have a genetic origin (primary HTG) or can be due to lifestyle choices (secondary HTG). Primary HTG is relatively uncommon and it mainly involves a mutation in the gene encoding for the enzyme lipoprotein lipase (Lpl). Lpl is mainly responsible for the breakdown of lipids rich in triglycerides. Secondary HTG; on the other hand, could be due to a high fat diet, excessive alcohol intake, conditions like type 2 DM, hypothyroidism, obesity, metabolic syndrome and intake of certain medications. Majority of patients with HTG are asymptomatic, while some may experience abdominal pain, acute pancreatitis and cutaneous xanthomas.<sup>[8,9]</sup>

According to the Endocrine society; mild to moderate HTG predisposes the individual towards CVD and severe to very severe HTG increases the risk for acute pancreatitis.<sup>[6]</sup> A study documented in the British Heart Journal -conducted by Bainton D *et al* has concluded that the serum triglyceride level is the main predictor for cardiovascular events when compared to total cholesterol levels.<sup>[10]</sup> Abdel Maksoud *et al* have inferred in their study that TG levels are an independent predictor of CVD.<sup>[11]</sup> Patel A *et al* carried out a meta analysis that included 26 studies and inferred that serum TG level is an independent and important predictor of cardiovascular events and stroke in the Asia – Pacific population.<sup>[12]</sup> Kannel WB *et al* in their review have

explained the role of fasting and non fasting TG as vascular risk factors in subjects that have low LDL Cholesterol.<sup>[13]</sup>

### HTG and pancreatitis

The mechanism of occurrence of acute pancreatitis in patients with HTG is proposed to be probably by the occlusion of pancreatic capillaries by chylomicron-triglyceride rich lipoprotein particles. This stimulates the release of pancreatic lipase which hydrolyzes triglycerides to release free fatty acids that when in excess can cause injury to the cell. This breakdown of the chylomicrons to free fatty acids may also cause release of inflammatory cytokines like IL-1 $\beta$  and IL-6 that initiates a cascade of inflammatory response.<sup>[14,15]</sup>

### HTG and Cardiovascular diseases

Chylomicrons are as such large particles and therefore cannot enter into the arterial intima. However, apo E and cholesterol-rich remnants of TG rich lipoproteins can easily enter and can bind to the vascular proteoglycans. The modification of these vascular proteoglycans due to oxidative damage liberates toxic by-products that cause local injury, activation of cytokine expression and inflammation. Ultimately, the remnants of the TG-rich lipoprotein are taken up by the macrophages leading to the formation of foam cells which initiates the formation of atherosclerotic plaque.<sup>[16,17]</sup>

### The Laboratory perspective

Lipemia is characterized by turbidity of serum/plasma due to the accumulation of lipoprotein particles. Amongst all, chylomicrons have the highest potential of causing turbidity in the sample [Figure 1].

Lipemia causes interference in various laboratory methodologies with spectrophotometry based methods being most affected. In HTG, TG may form a separate layer at the top of patient's serum which can result in a decrease in electrolytes and metabolite concentration.

There are various techniques that are used for resolution of lipemia interference in samples during laboratory analysis. These include:

1. Centrifugation of the samples in an ultracentrifuge. This method has limited application because of the limited availability of ultracentrifuge in regular diagnostic laboratories.
2. Extraction of the samples using polar solvents. This method however, is time consuming and prevents determination of all lipophilic substances.
3. Dilution of sample using normal saline. This is the most commonly used method which has been employed in the afore mentioned cases as well. A

dilution of 1:5 was able to overcome the lipemic interference for all the analytes tested.<sup>[18,19]</sup>

The technological advancement in clinical laboratories has made the analytical phase of laboratory analysis increasingly reliable. However, it is essential that the analyst holds sound knowledge of the technique used and takes steps accordingly to eliminate analytical interferences in the laboratory, which in most cases are simple measures. These solutions make the laboratory tests more reliable for diagnosis and clinical follow up which ultimately allows the laboratory to successfully accomplish its mission to aid in appropriate diagnosis.

Previous studies have shown that the population of Indian subcontinent has a higher predisposition towards CAD even with low cholesterol levels. Another hospital based study conducted on South Asian population has revealed that 75% of AMI patients have a Total cholesterol level <200mg/dl that indicates that the population in question has a lower threshold to TC levels when compared to the western population.<sup>[20,21]</sup> This also re-iterates the additional risk of CAD to the South-Asian population.

## CONCLUSION

This case series demonstrates the incidental finding of HTG in a diagnostic laboratory of a tertiary care centre of India within a span of 8 months. This does not include the other known cases of HTG attending the Medicine outpatient clinic. This case series also highlights that all the HTG case findings were observed to be in males within the age group of 31 to 40 years. This throws light on the increased risk of development of CAD in the young adults and therefore emphasis needs to be given to the screening of this age group for timely diagnosis of these chronic and occasionally lifestyle disorders like Type 2 DM, dyslipidemia and metabolic syndrome on the whole. This would help in timely control of its progression.

## REFERENCES

1. Garg R, Rustagi T. Management of hypertriglyceridemia induced acute pancreatitis. *BioMed Res Int*, 2018; 2018:4721357.
2. Parhofer KG, Laufs U. The Diagnosis and Treatment of Hypertriglyceridemia. *Dtsch Arztebl Int*, 2019; 116(49):825-32.
3. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*, 2001; 285:2486-97.
4. Miller M, Stone NJ, Ballantyne C, Bittner V, Criqui MH, Ginsberg HN et al. American Heart Association Clinical Lipidology T, Prevention Committee of the Council on Nutrition PA, Metabolism, Council on Arteriosclerosis T, Vascular B, Council on Cardiovascular N, Council on the Kidney in Cardiovascular D. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*, 2011; 123:2292-333.

5. Jacobson TA, Ito MK, Maki KC, Orringer CE, Bays HE, Jones PH, et al. National lipid association recommendations for patient- centered management of dyslipidemia: part 1--full report. *J Clin Lipidol*, 2015; 9:129-69.
6. Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, Murad MH, Stalenhoef AF. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97:2969–2989.
7. Authors/Task Force Members. Catapano AL, Graham I, De Backer G, Wiklund O, Chapman MJ, Drexel H, Hoes AW, Jennings CS, Landmesser U, Pedersen TR, Reiner Z, Riccardi G, Taskinen MR, Tokgozoglu L, Verschuren WM, Vlachopoulos C, Wood DA, Zamorano JL. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias: The Task Force for the Management of Dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). *Atherosclerosis*, 2016; 253:281-344.
8. Gomez-Huelgas R, Bernal-Lopez MR, Villalobos A, Villalobos A, Mancera-Romero J, Baca- Osorio AJ et al. Hypertriglyceridemic waist: an alternative to the metabolic syndrome? Results of the IMAP Study (multidisciplinary intervention in primary care). *Int J Obes (Lond)*, 2011; 35(2):292-9.
9. Maric N, Mackovic M, Bakula M, Mucic K, Udiljak N, Marusic M. Hypertriglyceridemia-induced pancreatitis treated with continuous insulin infusion-Case series. *Clin Endocrinol (Oxf)*, 2022; 96(2):139-43.
10. Bainton D, Miller NE, Bolton CH. Plasma triglyceride and high density lipoprotein cholesterol as predictors of ischaemic heart disease in British men: The Caerphilly and Speedwell Collaborative Heart Disease Studies. *Br Heart J*, 1992; 68(1): 60-6.
11. Abdel-Maksoud MF, Hokanson JE. The complex role of triglycerides in cardiovascular disease. *Semin Vasc Med*, 2002; 2(3): 325-333.
12. Patel A, Barzi F, Jamrozik K et al. Serum Triglycerides at al. Risk Factor for Cardiovascular Diseases in the Asia-Pacific Region. *Circulation*, 2008; 110(17): 2678-86.
13. Kannel WB, Vasan RS. Triglycerides as vascular risk factors: New Epidemiologic Insights For Current Opinion in Cardiology. *Curr Opin Cardiol*, 2009; 24(4): 345-350, 2009.
14. Gan SI, Edwards AL, Symonds CJ, Beck PL. Hypertriglyceridemia-induced pancreatitis: A case-based review. *World J Gastroenterol*, 2006; 12(44): 7197-202.
15. Yang F, Wang Y, Sternfeld L et al. The role of free fatty acids, pancreatic lipase and Ca<sup>2+</sup> signalling in injury of isolated acinar cells and pancreatitis model in lipoprotein lipase-deficient mice. *Acta Physiol (Oxf)*, 2009; 195(1): 13-28.
16. Austin MA, Hokanson JE. Epidemiology of triglycerides, small dense low-density lipoprotein, and lipoprotein(a) as risk factors for coronary heart disease. *Med Clin North Am*, 1994; 78:99-115.
17. Goldberg IJ, Eckel RH, McPherson R. Triglycerides and heart disease: still a hypothesis? *Arterioscler Thromb Vasc Biol*, 2011; 31:1716-25.
18. Nikolac N. Lipemia: causes, interference mechanisms, detection and management. *Biochem Med (Zagreb)*, 2014; 24(1): 57-67.
19. Andrade NNN, Oliveira MV, Souza CL. Procedures to minimize interference of hypertriglyceridemia in laboratory exams of lipemic samples in acute pancreatitis: a case report. *J Bras Patol Med Lab*, 2016; 52(2):103-6.
20. Lee J, Heng D, Chia KS, Chew SK, Tan BY, Hughes K. Risk factors and incident coronary heart disease in Chinese, Malay and Asian Indian males: the Singapore Cardiovascular Cohort Study. *Int J Epidemiol*, 2001; 30(5):983-8.
21. Biswas UK, Kumar A. Hypertriglyceridemia: a case report from diagnostic laboratory, Barasat, West Bengal, India. *Asian Pac J Trop Biomed*, 2011; 1(4):328-9.