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Drug-interference in clinical chemistry analyses: pseudohypertriglyceridaemia

Knowledge about the drug kinetics and their possible interactions with analytical methods is required to correctly validate results of a patient. Many times these discussions about laboratory related factors affecting the patient values are confined to clinical biochemistry forums. However, there is a need for increasing awareness of clinicians regarding this issue. With this in mind, we present three cases of hypertriglyceridaemia due to drug interference.

In 2013, samples were received from three patients who presented with acute stroke for lipid profile estimation. Physical signs of xanthomas or xanthelasmas were absent in all the patients. They were being treated with antihypertensive medication, antioedema measures and neuroprotective drugs. Oral glycerol was advised as a part of treatment to all of them. None of these samples were lipaemic on inspection. The clinicians were advised to send a fresh fasting sample for lipid profile testing before starting the day's medications and were received from cases 1 and 2. The repeat serum triglyceride values were found to be (90 mg/dL and 100 mg/dL) respectively. The details are shown in Table 1.

Pseudohypertriglyceridaemia is defined as the presence of falsely high levels of serum triglycerides due to excess concentrations of glycerol in blood.[1] Over estimation of triglycerides can be seen because of interference caused with high glycerol levels.[2] High blood levels of glycerol are seen in a genetic disorder resulting from glycerol kinase deficiency[3] or from exogenous sources like blood collection tubes with glycerol coated stoppers or from drugs. The other important exogenous sources of interference is drugs prescribed for the patient.[4] Endogenous interferent includes the effects of hemolysis, bilirubinemia, lipemia and paraproteinemia. Glycerol kinase deficiency is more prevalently seen as a cause for pseudohypertriglyceridaemia. An online *Medline* search revealed two case reports[1,2] due to oral glycerol therapy as part of the patient's management for some other clinical condition.

Repeat testing revealing normal values rules out glycerol kinase deficiency. Also the other

Table 1: Lipid profile levels in the 3 cases

Patient	Age (years), gender	Sample	Serum total cholesterol (mg/dL)	Serum triglycerides (mg/dL)	Serum HDL (mg/dL)
1	46, M	Initial	172	962	47
		Fresh sample before administration of glycerol	152	90	58
2	65, F	Initial	161	1140	34
3	43, F	Initial	128	1588	42
		Fresh sample before administration of glycerol	143	100	38

M = male; F = female; HDL= high density lipoprotein

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possible exogenous source i.e., like blood collection tubes with glycerol coated stoppers are not evident in the present series. Further, other causes like haemolysis, bilirubinemia, lipaemia, paraproteinemia were not evident in our patients. Thus, drugs interfering with laboratory analysis is the likely cause of psuedohypertriglyceridaemia in our patients. On review of medical records of these patients, all the three cases had received oral glycerol 30 mL 8th hourly as a part of their treatment. Orally ingested glycerol is rapidly absorbed via the intestine appears in the blood. Maximum serum glycerol levels have been observed 1-2 hours after ingestion. The terminal elimination half-life has been shown to be 0.61-1.18 hours.[5]

Estimation of triglycerides in the present study was done on Beckman synchron CX5 autoanalyzer, USA using commercial kit from AMS S.p.A, Guidonia (Rome), Italy. The method is linear upto 1000 mg/dL. Samples with levels above 1000mg/dL are diluted 1:1 using normal saline. Estimation of triglycerides involves hydrolysis of triglycerides by lipase. Glycerol concentration is then measured through coupled reactions that finally produce a quinoneimine, whose concentration is proportional to the triglyceride concentration.

Free glycerol present in the blood reacts in the second step along with glycerol released from triglycerides giving false high values. To avoid such interferences, a blood sample preferably before administration of drugs or after the biological half-life of the drugs has elapsed should be collected for analysis. The most important reason of this presentation is to bring out the requirement of exposing the clinicians to the quality control issues of clinical chemistry laboratory during their postgraduate training. As is well known, generating a quality report is a two way process involving both the clinicians and laboratory physicians.[6,7] The knowledge about glycerol administration coming in the way of triglyceride estimation at the level of the clinical departments would have facilitated obtaining the samples at an appropriate time and there by avoiding laboratory error.

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