CLINICAL VIGNETTE

OCP Triggered Hypertriglyceridemia: An Uncommon Cause of Pancreatitis

Diana Sarkisyan, MD

A 25-year-old female presented to her primary care physician for an annual exam. She was asymptomatic, but frustrated that despite diet and treatment with fenofibrate, she had not been able to decrease her triglyceride level. She denied alcohol and drug use. She did have a family history of dyslipidemia. She had been hospitalized two months prior with acute pancreatitis where it was first discovered that she had elevated triglycerides (TG).

Physical exam was normal with BMI of 25.3. Fasting glucose, LFTs, CBC, and TSH were normal. She had been screened for primary hypertriglyceridemia during her hospitalization and outside labs showed no mutations in the LPL, apo CII and apo AV genes. Upon discharge from her prior hospitalization, she had started fenofibrate and she had been taking norgestimate-ethinyl estradiol oral contraceptive (OCP) for three years. TG level prior to fenofibrate initiation was 870 and after 682. She was taken off OCP and her subsequent TG level reduced to normal.

Discussion

Hypertriglyceridemia accounts for only 4-10% of acute pancreatitis.[1] Primary causes of hypertriglyceridemia are genetic vs secondary causes which include obesity, diabetes, pregnancy, and medications. Of these medications, oral contraceptives are well known to induce increases in triglyceride levels.[2] However, case reports of OCP induced hypertriglyceridemia leading to acute pancreatitis are rare, and reported cases have significantly worse clinical outcomes.[3] Given the morbidity associated with TG induced pancreatitis, OCP as a common cause of hyper TG should be recognized early by clinicians.

Contraceptive induced hypertriglyceridemia occurs due to estrogen's action in decreasing hepatic triglyceride lipase and lipoprotein lipase activity. This causes an overall increase in low and intermediate-density triglycerides and cholesterol which then can increase free fatty acids to a toxic level.

A study conducted by Naz et al showed statistically significant differences in TG levels in users of OCPs compared to non-users with TG levels of 105 vs 83 respectively.[4]

The exact mechanism of acute pancreatitis secondary to hyper TG is not well established. It is thought that excess TG is hydrolyzed by pancreatic lipase forming free fatty acids which then induce inflammation.[5]

Most of the studies conclude that acute pancreatitis due to hypertriglyceridemia confers worse prognosis than acute pancreatitis due to other etiologies.[6] Recent studies report elevated TG levels are independently associated with a more severe course of AP.[6]

Conclusion

Among the causes that led to secondary hypertriglyceridemia, the use of contraceptive agents is the main reason to be evaluated in young females. As such, it may be wise to check baseline TG prior to initiating oral contraceptives.

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