Sonographic "Comet Tail" Artifacts in the Urinary Bladder in a Case of Type V Hyperlipidemia: A Previously Unreported Association

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Abstract

The case pertains to a 29-year-old male who presented with epigastric pain at the hospital. On evaluation, he was sonographically detected to have "comet tail" reverberation artifacts involving the echogenic floaters in the lumen of the urinary bladder. The patient was subsequently diagnosed with Type V hyperlipidemia and acute pancreatitis. The association between this classic sonographic finding and hyperlipidemia has not been previously found documented in the available literature and hence merits a discussion of the relevant clinical details pertaining to the case.

Keywords: Cholesterol, comet tail artifacts, hypercholesterolemia, hyperlipidemia, reverberation

Introduction

'Comet tail' artifact seen on ultrasound is a reverberation type of artefact produced due to manifest variance in acoustic impedances between a crystal and its adjacent media. It is defined as a condensed elongated tapering trail of sonographic echoes occurring distal to a markedly reflecting particle.[1,2] It has been described in the literature as being relevant in the diagnosis of a score of pathological conditions involving the thyroid, lungs, liver, bile ducts, gallbladder, and pancreas.[1] In cases specific to gall bladder, comet-tail artifacts are considered as being suggestive of the cholesterolic character of the tissue being imaged, diagnosed as cholecystoses of gall bladder (adenomyomatosis or choleterolosis).[3-5] The colour doppler variant of this artefact is the colour 'comet-tail artifact' which is described as a rapidly alternating colour doppler signal occurring posteriorly in relation to the crystal causing it and appearing on doppler as a linear band of colour showing aliasing, seen in calcified lesions, urinary and biliary stones.[6,7]

Case Report

A 29-year-old, moderately built, nonobese male presented to the hospital with chief complaints of epigastric pain for the past 2 days. The initial workup in the form of a routine hemogram did not reveal any abnormality with normal cell counts. Subsequently, the patient was taken up for ultrasound using a 2–5 MHz convex array transducer, which detected hepatomegaly with diffusely increased hepatic echogenicity consistent with hepatic steatosis. The pancreas could not be visualized because of being obscured by bowel gas.

The unique finding that caught attention during the ultrasound was the presence of "comet tail" reverberation artifacts emanating from echogenic floaters in the urinary bladder distended with urine [Figures 1 and 2]. This lead to clinical suspicion of a deranged lipid profile as similar findings of 'comet tail' artifacts are also typically seen in cases of cholesterol crystal deposition in the gall bladder wall in adenomyomatosis & choleterolosis.

A blood lipid profile was done & our patient was detected to have a high triglyceride level of 2090 mg/dl, which is classified as 'very severe' hypertriglyceridemia (>2000 mg/

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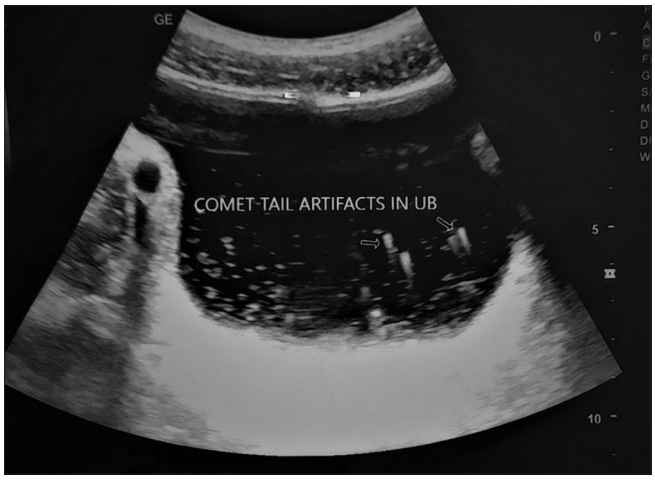

Figure 1: Axial section at the level of urinary bladder showing "comet tail" reverberation artifacts (arrows)

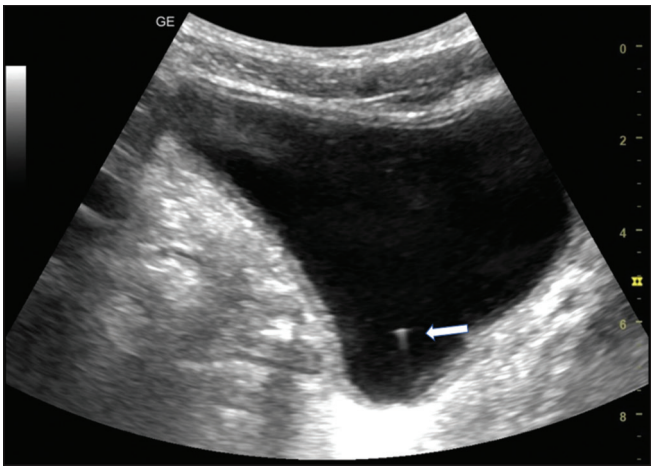

Figure 2: Sagittal section at the level of urinary bladder showing the "comet tail" artifact (arrow)

dl) as per the endocrinology society guidelines provided by Berglund *et al*.[8] Cholesterol level was 227 mg/dL, low-density lipoprotein (LDL) <30 mg/dL, high-density lipoprotein (HDL) 20 mg/dL, and very-low-density lipoprotein 456 mg/dL. Serum amylase levels were 670 IU/L, and serum lipase was 830 IU/L. Subsequently, a contrast-enhanced computer tomography scan of the abdomen was done, which showed acute pancreatitis with a modified computed tomography severity index of 4. He had no family history of dyslipidemia (his mother and sister were tested). All secondary causes of increased triglycerides were ruled out. He had a normal range for thyroid-stimulating hormone (0.85 mIU/L) and normal apolipoprotein-B levels.

The urine was normal in gross appearance with a triglyceride level of 115 mg/dL. No urinary proteins or cholesterol was detected. In contradiction to the supposition that lipid-containing urine would be milky in gross appearance, Peng et al actually found in their study on chyluria patients, that nearly half of the urine-triglyceride-positive patients had clear urine at time of examination.[9] To be more specific, urine triglyceride levels of <100 mg/dL did not give a hazy appearance to urine.[10] Urine triglycerides/cholesterol were not normally found in healthy individuals/control subjects with no "normal" range specified in available literature.[9,10]

Our patient was subsequently diagnosed as Type V hyperlipidemia with acute pancreatitis. He was managed with plasmapheresis, fibrates, statin, and nonstatin agents like ezetimibe.

Discussion

Type V hyperlipoproteinemia is a condition typically seen in adults, characterized by laboratory findings that include fasting triglyceride levels exceeding 1000 mg/dL (>11.3 mmol/L), increased total cholesterol, and low concentrations of both LDL and HDL cholesterol. This disorder stands out among the spectrum of lipid disorders due to its distinctive profile, deviating from the classic classifications of hyperlipoproteinemia outlined by Frederickson's classification, which categorizes these disorders from Type I to V based on specific lipoprotein components in the serum.[11]

Familial hypercholesterolemia is one of the most common inherited disorders in Japan and Western countries, with a frequency of 1 in 500.[12,13] Combined hyperlipidemia (both hypertriglyceridemia and hypercholesterolemia), known as Type V hypercholesterolemia, is occasionally familial and has more acquired environmental aspects as its causative factors. It causes accumulation of lipid droplets in pancreatic acinar cells, underlying that elevation of plasma triglycerides is essential to the development of acute pancreatitis as one of the most common complications in these patients. The metabolism of triglycerides to free fatty acids serves to trigger acute pancreatitis. In contrast, isolated hypercholesterolemia does not lead to massive alterations in the endocrine function of the pancreas as brought out by Csonka *et al*.[14] Our patient had acute pancreatitis, which developed as a complication of Type V hyperlipidemia.

Klahr *et al*. conducted a qualitative and quantitative urine analysis in chronic renal failure and nephrotic syndrome patients. They found increased urinary lipids (free cholesterol, cholesterol esters, triglycerides, free fatty acids, and phospholipids) in these patients with the hypothesis that glomerular damage resulted in loss of lipoprotein in glomerular filtrate, which resulted in detection of urinary lipids.[15] Interestingly, one patient in their study group having hyperlipidemia and not suffering from nephrotic syndrome was found to have increased urinary triglyceride levels, not explained by the glomerular damage theory. However, they could not establish any correlation between urinary excretion of lipids and the blood lipid levels in subjects in the study group.

In our study, no increased urinary protein excretion was seen, so the possibility of lipoprotein in urine leading to detection of urinary lipids is excluded. The exact underlying mechanism for the presence of triglycerides in urine in our case of hyperlipidemia could not be ascertained. One possibility is

that a part of the lipid appearing in the urine might have been sourced from the blood through tubular secretion. Another likelihood is that since 90% of the intestinal chylomicron-lipid composition is triglyceride, and also our patient had only triglyceride in urine with no cholesterol, it is possible that the source of the urine lipid was intestinal chylomicron. Both these theories need advanced research with biochemical tagging of lipids to ascertain the source of fatty acids in urine.

Rokni & Simon demonstrated that cholesterol crystals in a water medium demonstrate higher twinkling artifact as compared to other pathological mineralizations like calcium phosphate or uric acid crystals, because cholesterol is a lipid and thus very hydrophobic. This leads to the formation of microbubbles on its surface thus causing reverberations.[16] The ‘comet tail’ artifacts seen in the present case, and hitherto so far unreported in literature with the present association, make a significant addition to the clinical inferences of this sonographic parameter. This finding can be pivotal in early diagnosis of familial hyperlipidaemia in unsuspecting populations. It will significantly contribute in reducing morbidity & mortality in patients who may otherwise clinically present with acute pancreatitis as the initial presentation secondary to the underlying hypertriglyceridemia.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent form. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Thickman DI, Ziskin MC, Goldenberg NJ, Linder BE. Clinical manifestations of the comet tail artifact. J Ultrasound Med 1983;2:225-30.
2. Oh SH, Han HY, Kim HJ. Comet tail artifact on ultrasonography: Is it a reliable finding of benign gallbladder diseases? Ultrasonography 2019;38:221-30.
3. Cherraqi A, Imrani K, Andour H, Messaoud O, Benelhosni K, Billah NM, *et al.* Low phospholipids associated cholelithiasis syndrome in a young women: A rare case report. Radiol Case Rep 2023;18:11-6.
4. Miyoshi H, Inui K, Katano Y, Tachi Y, Yamamoto S. B-mode ultrasonographic diagnosis in gallbladder wall thickening. J Med Ultrason (2001) 2021;48:175-86.
5. Shapiro RS, Winsberg F. Comet-tail artifact from cholesterol crystals: Observations in the postlithotripsy gallbladder and an *in vitro* model. Radiology 1990;177:153-6.
6. Tchelepi H, Ralls PW. Color comet-tail artifact: Clinical applications. AJR Am J Roentgenol 2009;192:11-8.
7. Kim HC, Yang DM, Jin W, Ryu JK, Shin HC. Color Doppler twinkling artifacts in various conditions during abdominal and pelvic sonography. J Ultrasound Med 2010;29:621-32.
8. Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, Murad MH, *et al.* Evaluation and treatment of hypertriglyceridemia: An endocrine society clinical practice guideline. J Clin Endocrinol Metab 2012;97:2969-89.
9. Peng HW, Chou CF, Shiao MS, Lin E, Zheng HJ, Chen CC, *et al.* Urine lipids in patients with a history of filariasis. Urol Res 1997;25:217-21.
10. Rahman MM. Easy method of detection of chyle in urine. Indian J Nephrol 2012;22:147-8.
11. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. Ann Intern Med 1971;75:471-2.
12. Mabuchi H, Miyamoto S, Ueda K, Oota M, Takegoshi T, Wakasugi T, *et al.* Causes of death in patients with familial hypercholesterolemia. Atherosclerosis 1986;61:1-6.
13. Molnár G, Gyarmathy VA, Zádori N, Hegyi P, Kanizsai P. Severe hypertriglyceridemia-induced acute pancreatitis. Case Rep Gastroenterol 2021;15:218-24.
14. Csonka C, Baranyai T, Tiszlavicz L, Fébel H, Szűcs G, Varga ZV, *et al.* Isolated hypercholesterolemia leads to steatosis in the liver without affecting the pancreas. Lipids Health Dis 2017;16:144.
15. Klahr S, Tripathy K, Bolanos O. Qualitative and quantitative analysis of urinary lipids in the nephrotic syndrome. J Clin Invest 1967;46:1475-81.
16. Rokni E, Simon JC. The effect of crystal composition and environment on the color Doppler ultrasound twinkling artifact. Physics in Medicine & Biology 2023;68:035021.