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Wet-Cupping's Impact on Pancreatitis Induced by Hypertriglyceridemia

A case study and brief literature review

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Abstract

Familial hypertriglyceridemia is a genetic disorder marked by excessive production of very low-density lipoproteins, resulting in elevated serum triglyceride levels. This can lead to various medical conditions, including acute pancreatitis. In cases of recurrence, it may progress to chronic pancreatitis. Cupping therapy, a traditional treatment practiced in numerous cultures worldwide, is utilized to address various medical conditions. This case report presents a 34-year-old male diagnosed with familial hypertriglyceridemia, subsequently developing chronic pancreatitis. During his last presentation with acute-on-chronic pancreatitis, his lipid profile revealed a notable reduction in serum triglycerides. Interestingly, this reduction coincided with the introduction of cupping therapy into his treatment regimen. Remarkably, following the initiation of cupping therapy, his hospital admissions for acute pancreatitis notably decreased.

This case report highlights the potential impact of cupping therapy on familial hypertriglyceridemia, potentially mitigating the risk of acute pancreatitis.

Keywords: Hyperlipidemia; Hypertriglyceridemia; Pancreatitis.

Introduction

Familial hypertriglyceridemia, an autosomal dominant genetic disorder, predominantly results from lipoprotein lipase (LPL) dysfunction. LPL regulates triglyceride metabolism in very low-density lipoproteins (VLDL), so its inactivity elevates serum triglycerides. This condition often coexists with hypertension, obesity, diabetes mellitus, and cardiovascular diseases.[1]

Acute pancreatitis, often linked to elevated serum triglycerides, is the most common consequence of hypertriglyceridemia. It's an inflammatory condition of the pancreas caused by the premature activation of pancreatic enzymes and cytokines, inducing autodigestion and inflammation. If acute episodes persist, chronic pancreatitis ensues. This ongoing inflammation activates stellate cells, leading to a fibro-inflammatory response that damages tissue structure and depletes normal parenchyma. Consequently, some patients experience exocrine and endocrine insufficiencies due to this tissue loss.[2]

Cupping is a method of treatment and healing that dates to 1550 BC when it was first described in the ancient Egyptian scripts. It is part of the traditional medicine of many other cultures, including Southeast Asia, Greece, Italy, and Arabic countries. Cupping is a process of using heated or depressurized cups that is applied to the skin to exert suction effects. One of the first comprehensive descriptions of the process was documented by Al-Zahrawi (AD 936-1036), an Arabic scientist, physician, surgeon, and chemist.[3] There have been many types of cupping described. Wet-cupping or bleeding cupping, the most common type involves piercing the skin with a sharp object to release minimal blood.[4] Another type includes only using the pressure generated from the suction apparatus or using heat, known as dry cupping (Figure 1). The most common use of cupping includes pain conditions and acne, as described in a review by Cao et al.[5] The use of cupping as a treatment for hypertriglyceridemia has been rarely described in the literature.[4,5]

Here we report a case that highlights a possible positive effect of cupping on the level of triglycerides and the rate of the episodes of acute pancreatitis.

Case Report

A 34-year-old man, previously healthy, not a smoker nor an ethanol consumer, obese with a history of recurrent episodes of pancreatitis every two months; they were frequent and very painful. He was followed in the Lipid Clinic for a raised triglyceride level. Initial lipid profiles at the time of the diagnosis showed a total cholesterol of 6.1 mmol/L, triglycerides of 27.4 mmol/L, very low-density lipoprotein (VLDL) of 1.8 mmol/L, and high-density lipoprotein (HDL) of 0.59 mmol/L. In view of this lipid profile, genetic testing confirmed lipoprotein lipase deficiency (LPL), and a family history of familial hypertriglyceridemia,[6] he was diagnosed with familial hypertriglyceridemia in 2015. Workup of all other acute pancreatitis causes, including biliary, autoimmune, hypercalcemia, and other causes, was negative. Two years after being diagnosed with familial hypertriglyceridemia, he developed type II diabetes in 2017, with HBA1C of 7.9%, thus was started on metformin 1 gram twice daily initially, then was shifted to a combined glargine-actrapid regimen of insulin that has achieved acceptable blood sugar readings. The patient was recommended for bariatric surgery and was subsequently referred to the surgical team.However, his BMI didn't fit the criteria for eligibility for bariatric surgery, thus deferred.The patient continued to have one epsidode of pancreatititis every one to two months.

At this admission (in February 2022), he presented after 6 months of pancreatitis-free durations , having episodes of abdominal pain, nausea, and vomiting for 2 days. There was no history of fever, jaundice, or any other systemic symptoms. The patient began wet cupping 10 months prior to the current presentation at the hospital. Initially, he practiced it once every two weeks for three months, after which the frequency was reduced to once a month. In each session, approximately 500ml of blood was extracted. Throughout that period, he experienced no episodes of abdominal pain at home and required no hospitalizations (Figure 2). However, he ceased wet cupping in the last 2-3 weeks leading up to the current presentation.

On examination was found to be conscious, alert oriented, and not in distress on the pain 8/10 scale. A general examination revealed an obese young man with a 37.7 Kg/m2 BMI, and he has no corneal arcus, xanthoma, or xanthelasma. The abdomen was soft, tender epigastrium. All other systematic exam was not remarkable.

During the admission, his lipid profile showed triglycerides of 24.2 mmol/L, hemoglobin of 15.7 g/dL, a white blood count of 7.4 x 10^9/L, and a platelet count of 252 x 10^9/L. Lipase on presentation was 858 U/L, amylase 72 U/L, and corrected calcium 2.43 mmol/L.

The patient was managed with intravenous fluids, normal saline 250ml/h alternating with ringer lactate, and pain management and was kept on glargine insulin 36 units at night with aspart insulin pre-each meal 18 units. Clinically, he improved, and his triglyceride level was 13.4 mmol/L on discharge.The patient consented to share the case for publication.

Discussion

This case report describes a case of a patient with familial hypertriglyceridemia who experienced recurrent pancreatitis, showing potential improvement after starting wet cupping.

The exact physiological effects of cupping were not yet fully defined; however, many previous reports theorized some mechanical effects, including activation of the immune and neuroendocrine systems signals resulting from skin stimulation, a similar physiological effect of acupuncture.[7] The pressure generated by the suction results in blood leaking into the tissue, which in turn causes an inflammatory response. The haemoglobin leaked to the tissue is degraded by the macrophages that produce hem-oxidase-I (OH-I), producing three main byproducts: iron, carbon monoxide, and bilirubin. The hem-oxidase-I has an ant-oxidant effect, anti-inflammatory, and immunomodulatory effect by activating Interleukin-10 (IL-10) production, production of catalase, and superoxide dismutase, all of which have ant-oxidant properties. In small amounts, carbon monoxide has a vasodilatory action by stimulating the production of cyclic guanosine monophosphate and an anti-inflammatory effect by down-regulating the production of Tumor Necrosis Factor Alpha (TNF-a) and IL-1b.[8] Hence, this could play a role in reducing or alleviating the inflammatory cascade involved in the

pathogenesis of pancreatitis. Additionally, increased blood flow to the cupped part of the skin and underlying muscle due to the stretch in the capillaries and the vasodilation generated from the local pressure was suggested by Wei et al. to relieve muscular pain and soreness.[9]

In a randomized controlled trial in Saudi Arabia, 80 participants were studied to test the outcome of wet cupping on reducing blood pressure. It has been found that wet cupping has resulted in a significant reduction in blood pressure in the first 4 weeks after the intervention (P=0.046); however, at 8 weeks of follow-up, there was no difference between the groups.[10] This raises the question regarding the potential effects of cupping on hypertensive patients if used in conjunction with pharmacological therapies. Studying the effects of cupping in blinded, randomized, controlled settings might be complex due to the difficulty of comparing it to a placebo. However, A review with metanalysis of a total of 135 randomized control trials done to evaluate the efficacy of cupping done by Huijuan Cao et al. showed that cupping combined with pharmacological therapy in patients with herpes zoster showed better cure outcomes (RR 1.93, 95% CI 1.23 to 3.04, p=0.005) and significantly reduced the rate of postherpetic neuralgia. Similar results were found regarding the effects of wet cupping on improving the symptoms of ankylosing spondylitis (RR 3.84, 95% CI 2.19 to 6.75, p,0.00001) and acne cure rate (RR 1.93, 95%CI 1.40 to 2.65, p,0.0001).[11]

The evidence of cupping efficacy in reducing cholesterol levels is still controversial. One of few articles published, a paper done to evaluate the level of lipids before and 10 days after the cupping among 40 participants selected randomly showed a significant reduction in the total cholesterol ($p = 0.002$), LDL ($P = 0.001$) and a significant increase in the HDL level ($p=0.027$). However, there were no comments on the triglyceride levels.[12] Another study published in 2012 which also tested the lipid profile of 31 hyperlipidemic men who were not on treatment, treated only with wet cupping, showed similar results; however, there was no significant difference in the triglycerides and HDL cholesterol levels found.[13] In contrast, another report showed a decrease in the triglyceride level after wet-cupping ($p \leq 0.05$), which agreed with another comparative study on 18 individuals by Saeed et al. ($p<0.001$).[14] Still, larger randomized controlled trials are needed to evaluate the hypothesis.

This case showed a potential effect of wet cupping in reducing triglyceride levels in the blood, thereby decreasing pancreatic inflammation and reducing the frequency of pancreatitis. However, in view of the lack of robust evidence, we have to acknowledge that this recovery might be spontaneous and coincide with wet cupping

Conclusion

Familial hypertriglyceridemia is a relatively rare genetic disease. Persistent elevation in the levels of triglycerides can lead to hypertriglyceridemia-induced pancreatitis. Recurrent episodes of acute pancreatitis are associated with significant mortality and morbidity. This challenging case showed resistance to the conventional options in treating hypertriglyceridemia resulting in recurrent episodes. Although the evidence to support wet-cupping has not been fully established, our patient showed favourable outcomes after wet-cupping, resulting in fewer admissions with acute pancreatitis in the observed duration. This case report calls for further structured studies to study the efficacy of cupping in reducing the frequency of acute pancreatitis among patients with hypertriglyceridemia.

Authors' Contribution

WS obtained patient consent and contributed to the literature review. AB and AM contributed to the case presentation. WS, AB and AM drafted the manuscript. HF and AA reviewed and edited the manuscript. All authors approved the final version of the manuscript.

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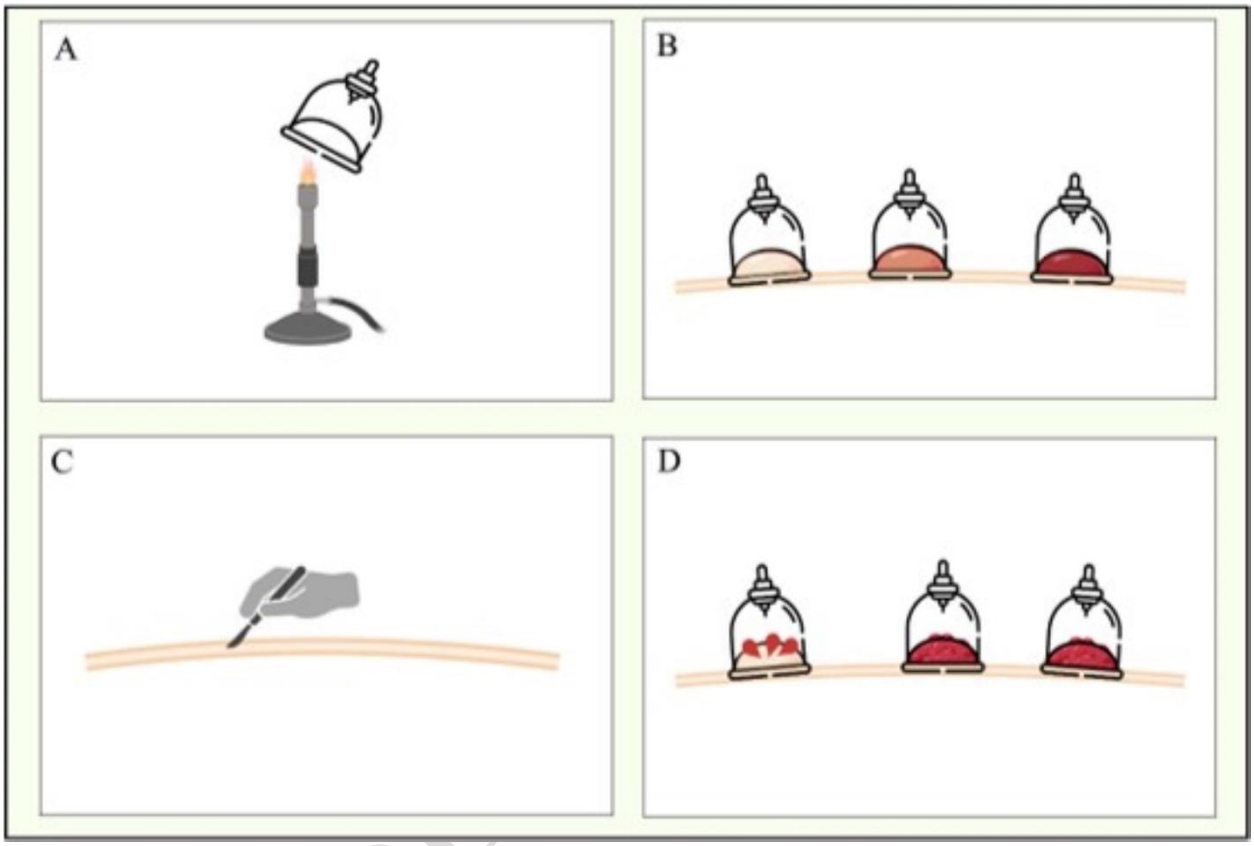

Figure 1: The process of dry and wet cupping. **A:** The cup is heated before the dry and wet cupping. **B:** The heated cups are applied to the skin in dry-cupping, and the blood accumulates underneath the skin. **C:** In wet-cupping, the skin first wounds before applying the heated cups. **D:** The heat generates withdrawing pressure that extracts the blood from the skin.

Figure 2: Wet-cupping with a lipemic blood sample