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CASE REPORT

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 utilised to address various medical conditions. We report a 34-year-old male patient who presented to a tertiary hospital, Muscat, Oman, in 2022. He was diagnosed with familial hypertriglyceridemia and subsequently developing chronic pancreatitis. During his last presentation with acute-on-chronic pancreatitis, his lipid profile revealed a notable reduction in serum triglycerides. 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; Case Report; Oman.

Familial hypertriglyceridemia, an autosomal dominant genetic disorder, predominantly results from lipoprotein lipase (LPL) dysfunction. LPL regulates triglyceride metabolism in very low-density lipoproteins (VLDL); 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 ancient Egyptian scripts. It is part of the traditional medicine of many other cultures, including Southeast Asia, Greece, Italy and Arabic countries. Cupping is the process of using heated or depressurised cups that are applied to the skin to exert suction effects. One of the first comprehensive descriptions of the process was documented by Al-Zahrawi (936–1036 AD), an Arabic scientist, physician, surgeon and chemist.[3] Many types of cupping have been described; wet-cupping or bleeding cupping is the most common type and involves piercing the skin with a sharp object to release minimal blood.[4] Another type is dry cupping which includes only using the pressure generated from the suction apparatus or using heat [Figure 1]. Cupping is most commonly used for 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]

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 male patient presented to a tertiary hospital, Muscat, Oman, in 2022. He was previously healthy, not a smoker nor an alcohol drinker and was obese with a history of recurrent episodes of pancreatitis every 2 months; these episodes were frequent and very painful. He was followed-up in the Lipid Clinic for 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, 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 LPL deficiency and a family history of familial hypertriglyceridemia (he was diagnosed with familial hypertriglyceridemia in 2015).[6] Workup of all other acute pancreatitis causes, including biliary, autoimmune, hypercalcaemia and

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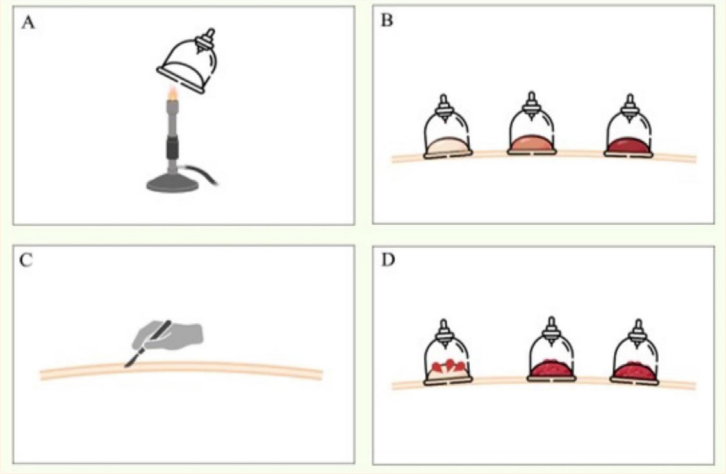

Figure 1: Images showing the process of dry and wet cupping. **A:** The cup is heated before the dry or wet cupping. **B:** In dry-cupping, the heated cups are applied to the skin and the blood accumulates underneath the skin. **C:** In wet-cupping, the skin is first wounded before applying the heated cups. **D:** The heat generates suction pressure that extracts the blood via the skin.

other causes, was negative. The patient developed type II diabetes in 2017, with an HbA1C of 7.9% and was therefore started on metformin 1 g twice daily initially; his prescription was subsequently changed to a combined glargine-actrapid regimen of insulin that achieved acceptable blood sugar levels. The patient was recommended for bariatric surgery and was subsequently referred to the surgical team. However, due to his body mass index (BMI), the patient was not eligibile for bariatric surgery and it was deferred. The patient continued to have one episode of pancreatititis every 1 to 2 months.

At admission, in 2022, he presented after 6 months of not having pancreatitis but with 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. Initially, he underwent cupping once every 2 weeks for 3 months, after which the frequency was reduced to once a month. In each session, approximately 500 mL of blood was extracted. Throughout that period, he experienced no episodes of abdominal pain at home and required no hospitalisations [Figure 2]. The patient ceased wet cupping 2–3 weeks before the current admission.

Figure 2: Photographs of wet-cupping with a lipemic blood sample

On examination, he was found to be conscious, alert, oriented, not in distress and an 8/10 on the pain scale. The patient's BMI was 37.7 kg/m^2 and he had no corneal arcus, xanthoma or xanthelasma. The abdomen was soft, tender epigastrium and all other systematic exam were unremarkable.

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

The patient was managed with intravenous fluids, normal saline (250 mL/h alternating with Ringer's lactate and pain management), and was kept on 36 units of glargine insulin at night and 18 units of aspart insulin before each meal. Clinically, he improved and his triglyceride level was 13.4 mmol/L on discharge.

Patient consent was obtained for publication purposes.

Discussion

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

The exact physiological effects of cupping have not yet been fully defined; however, many previous reports theorise some mechanical effects, including activation of the immune and neuroendocrine systems signals resulting from skin stimulation; a similar physiological effect in seen in acupuncture.[7] The pressure generated by the suction results in blood leaking into the tissue, which in turn causes an

inflammatory response. The haemoglobin is degraded by the macrophages that produce heme-oxidase-1, producing 3 main byproducts: iron, carbon monoxide and bilirubin. The heme-oxidase-1 has an anti-oxidant, anti-inflammatory and immunomodulatory effect by activating interleukin (IL)-10 production and production of catalase and superoxide dismutase, all of which have anti-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 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 Wang *et al*. to relieve muscular pain and soreness.[9]

In a randomised controlled trial in Saudi Arabia, 80 participants were studied to test the outcome of wet cupping on reducing blood pressure. The trial found that wet cupping 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 of potential effects of cupping on hypertensive patients if used in conjunction with pharmacological therapies. Studying the effects of cupping in blinded, randomised, controlled settings might be complex due to the difficulty of comparing it to a placebo. However, Cao *et al*.'s review and meta-analysis of 135 randomised control trials, which aimed to evaluate the efficacy of cupping, by showed that cupping combined with pharmacological therapy in patients with herpes zoster showed better cure outcomes (relative risk [RR] = 1.93, 95% confidence interval [CI]: 1.23–3.04; $P = 0.005$) 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: 2.19–6.75; $P < 0.00001$) and acne cure rate (RR = 1.93, 95% CI: 1.40–2.65; $P < 0.0001$).[11]

The evidence of cupping efficacy in reducing cholesterol levels is still controversial and few studies have been done. Niasari *et al*.'s study, which evaluated the level of lipids before and 10 days after the cupping among 40 randomly selected participants, 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] Mustafa *et al*.'s study was published in 2012 and also tested the lipid profile of 31 hyperlipidemic men who were not on treatment and were treated only with wet cupping; their study showed similar results, however, there was no significant difference in the triglycerides and HDL cholesterol levels.[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*. ($P < 0.001$).[12,14] Still, larger randomised 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, the current authors 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. The current case showed resistance to the conventional methods of treating hypertriglyceridemia resulting in recurrent episodes. Although the evidence to support wet-cupping has not been fully established, the current patient showed favourable outcomes after wet-cupping, resulting in fewer admissions with acute pancreatitis in the observed duration. Further structured studies should be conducted 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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