Case report

Acute pancreatitis following ingestion of high dose *Garcinia cambogia*

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Abstract

Acute pancreatitis is a systemic inflammatory condition carrying significant morbidity and mortality. *Garcinia cambogia* (GC) has been recently deduced as a rare cause of this condition.

A 22-year-old female presented with epigastric pain, nausea and systemic unrest. Her amylase was elevated and had high inflammatory markers. CECT abdomen revealed necrotizing acute pancreatitis. After receiving in-hospital care for two weeks, she made a complete recovery. She had consumed large doses of GC as a weight loss supplement for several months preceding. After excluding other causes of pancreatitis, GC was identified as the possible cause.

Acute pancreatitis due to GC is a rare entity with only a handful of cases reported. Large doses of GC are thought to induce oxidative damage in the pancreas causing inflammation. Current evidence for its causal nature is based on history and deduction only. Further research is needed to confirm GC as a cause of pancreatitis.

Keywords: pancreatitis, garcinia, case report, Sri Lanka, toxicology

Background

Acute pancreatitis is a condition characterised by diffuse inflammation of the pancreas carrying significant morbidity and mortality.[1,2] Global incidence is 34 per 100,000 person-years and is on the rise.[1] It also carries a large burden on annual healthcare expenditure accounting for almost 10 billion dollars annually in the USA.[1] Sri Lankan statistics on this condition is sparse however an increasing trend in incidence has been observed in our clinical practice.

Acute pancreatitis is diagnosed in accordance with the revised Atlanta classification when two of the following three criteria are met; acute onset severe, persistent epigastric pain often radiating to back; serum amylase/lipase levels more than thrice upper normal; characteristic findings of acute pancreatitis

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on contrast-enhanced computerised tomography (CECT) or magnetic resonance imaging (MRI) /ultrasonography of the abdomen.[2] Treatment is based on resuscitation, analgesia, close monitoring, and treating the causative factors and complications. Secondary prevention plays a major role in long-term management where education, lifestyle modification, pharmacotherapy and surgery are instigated to treat or control the causative agents.[2]

Acute pancreatitis has a plethora of causative factors, of which gallstone disease and alcohol take precedence.[2] A less known cause is *Garcinia cambogia* (GC), natively termed "goraka", which is a fruit native to Southeast Asia used as a condiment.[3,4] In addition, it has been used as an appetite suppressant for eons in herbal medicine which uses greater amounts than what is used in day-to-day cooking. Few case reports on acute pancreatitis caused by GC have been published but none have been reported from Sri Lanka thus far. This is the first such case reported from Sri Lanka.

Case presentation

A 22-year-old female presented to surgical casualty with generalised abdominal pain for three days. The pain had initiated in the epigastrium with radiation to the back but had soon spread throughout the abdomen. This was associated with profuse vomiting and nausea. She had no history of fever, jaundice or melena. Her urine output had been low and was constipated for two days. She denied a history of biliary colic, alcohol ingestion, smoking, trauma to the abdomen, recent viral infection, scorpion or other animal bites. She was not on prescribed medication for any medical condition and had no family history of pancreatic pathology. The only peculiarity in her otherwise insignificant history was the ingestion of a large amount of GC made into juice regularly for several months preceding this event as a herbal remedy to lose weight.

On examination she was tachycardic with a heart rate of 100 beats per minute; blood pressure was 104/67mmHg and had significant epigastric tenderness with generalised abdominal distension and minimal bowel sounds. She was not pale or icteric, nor did she have any ecchymoses. Respiratory and neurological examinations were normal. Initial resuscitation with crystalloids stabilised the patient, and her parameters normalised.

Initial blood investigations revealed neutrophil leukocytosis, elevated serum amylase of more than thrice the normal upper limit and high C-reactive protein. Liver functions were normal and so was her urine full report and serum creatinine. Fasting blood glucose and lipid profile were within normal range. Serum calcium was low and the rest of her electrolytes were normal (Table 1). Chest radiograph excluded gas under the diaphragm and abdominal X-ray revealed mild distension of ileal loops. Computerised tomography of the abdomen showed that the pancreas was swollen with multiple small hypodense areas throughout (Figure 1). Peri-pancreatic retroperitoneal fat stranding was noted however, no collections, abscesses or calcifications were seen. The liver, gall bladder and biliary tract were all normal, with no evidence of gallstones.

A diagnosis of acute necrotizing pancreatitis most likely due to GC was made by deduction and she received intensive care for two days and was sent to the ward for continuation of care. After receiving two weeks of in-ward care, the patient was discharged after making a complete recovery.

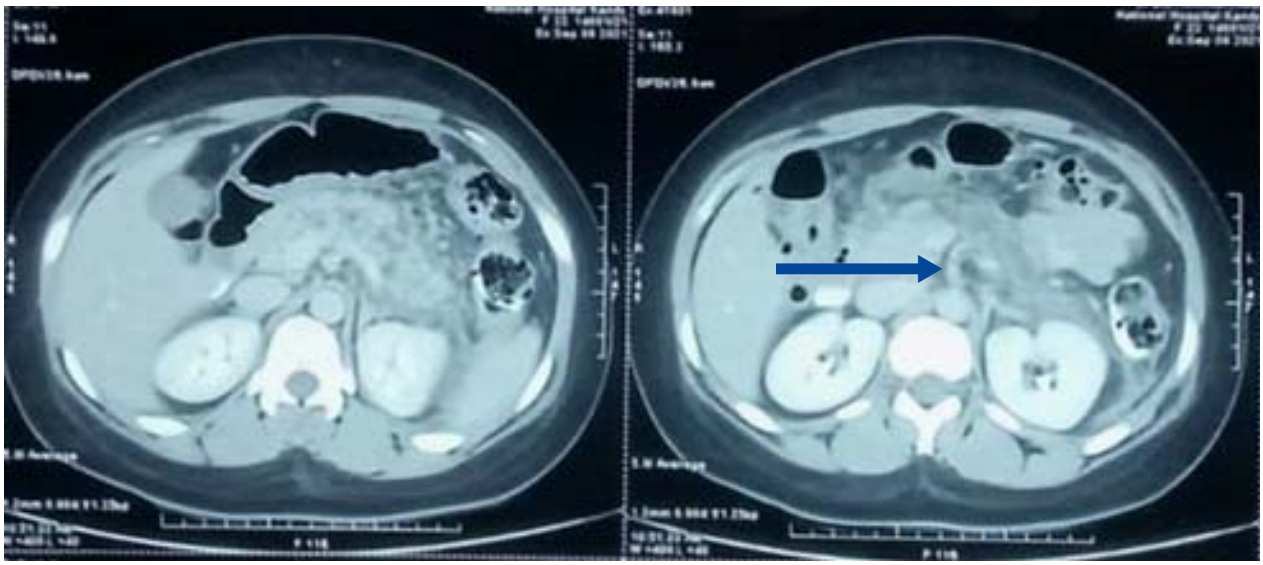

Figure 1. CT images depicting diffuse oedema of the pancreas, peri pancreatic fat stranding and areas of necrosis consistent with acute necrotizing pancreatitis.

Table 1. Blood investigations

Investigation	*Value*	*Normal range*
White blood cells (WBC)	17 ×10^9/L	4-11×10^9/L
Haemoglobin (Hb)	12.5 g/dL	12-15 g/dL
Platelets	234 ×10^9/L	150-450 ×10^9/L
Serum amylase	458 U/L	30-110 U/L
CRP	264 mg/L	<10 mg/L
AST	21 IU/L	10-40 IU/L
ALT	30 IU/L	7-56 IU/L
ALP	150 IU/L	90-300 IU/L
Total bilirubin	8 µmol/L	3-20 µmol/L
PT INR	1.0	< 1.1
Serum creatinine	79 µmol/L	50-110 µmol/L
Serum calcium	1.6 mmol/L	2.2-2.6 µmol/L
Serum Na	141 mEq/L	135-145 mEq/L
Serum K	4.1 mEq/L	3.5-5.5 mEq/L
Fasting blood glucose	85 mg/dL	70-99 mg/dL

Discussion

Pathophysiology of acute pancreatitis is complex as new research sheds light on its vast array of complex intracellular events. However, several of these events are at the center of its pathogenesis irrespective of the cause. These are abnormal calcium signalling, premature trypsinogen activation within acinar cells, mitochondrial dysfunction, endoplasmic reticulum stress, impaired unfolded protein response and impaired autophagy. These events are triggered by toxic damage to acinar cells and by increased ductal pressures. With resulting cell injury, an acute inflammatory response is initiated by the body which results in pancreatitis.[1-5]

GC is derived from the fruit of the Malabar tamarind tree which is native to Southeast Asia including Sri Lanka.[6] Of its many chemical compounds, hydroxy citric acid (HCA) is the most important medically as it is responsible for the weight reduction property of GC. HCA acts by inhibiting ATP citrate lyase which is an enzyme involved in fatty acid synthesis that results in reduced lipogenesis and favour lipid oxidation.[3,6,7] It is known to cause appetite suppression via elevating serotonin levels in the brain.[4,7] Both these effects act synergistically and enhance weight reduction. Taking these into consideration, many herbal products using *Garcinia cambogia* have been manufactured to cater for the increasing obese population as weight reduction supplements.[8]

Several reports have put forward theories as to how GC cause acute pancreatitis. These propose increased oxidative stress to be the culprit in initiating acinar cell damage.[3,4] It had been observed that supplementation with GC up-regulated mRNA expression of hepatic superoxide dismutase and glutathione peroxidase owing to an increase in lipid peroxidation. This suggests that GC induces oxidative stress which then results in the over-expression of antioxidant genes. Several reports have depicted the association of GC with hepatotoxicity. [3,4,8] It is hypothesized that GC increases oxidative stress in the pancreas in a fashion similar to that in the liver which causes acinar cell damage and inflammation resulting in acute pancreatitis.

Conclusion

Based on current literature, acute pancreatitis caused by GC presents like any other case of pancreatitis; classically epigastric pain radiating to the back with nausea/vomiting and systemic unrest.[3,4,9] All reported cases had elevated amylase/lipase levels and radiological changes classically seen in pancreatitis on computerized tomography.[3,4,9] Transaminases were normal in all cases which excluded hepatocellular damage. All reports so far on GC pancreatitis is based on circumstantial and historic evidence and as of yet, no accurate test is available to confirm the causal relationship of GC to pancreatitis. Further research is therefore necessary in this regard to shed light into its pathophysiology and to confirm its causal nature.

Author contributions

SMPV, LSC, SJ, EADU, JW, AS, AD discussed the case. SMPV, JW, AS and AD collected information. LSC, SJ and EADU critically analysed the case. SMPV prepared the initial draft. All did the relevant alterations and after compiling the final draft accepted it.

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Declaration of conflicting interest

No conflicts of interest to declare.

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Data availability statement

Not applicable.

Ethics approval

Ethical clearance is exempted for case reports at our institution.

Consent to participate and publication

Written informed consent was obtained from the patient for the publication of this case report.

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