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Acute Pancreatitis and Alcohol Abuse: A Case Report

Case Report

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Introduction

Damage affects normal digestion and absorption (1). Gallstone, alcohol abuse, auto immune, drug abuse, abnormal trauma, post surgery, infections, metabolic changes like hypercalcemia, hyperparathyroidism, hypertriglyceridemia and genetics are risk factors causing pancreatitis (2,3-7). Risk associated with seen in developing countries like India and China have reported increased consumption of alcohol (2,11). We present an interesting case of a man with a history of hypertension, two attacks of pancreatitis, Hepatomegaly with grade 1 fatty liver (due to alcohol, overweight, hypertension) is admitted in the hospital with a relapse of acute pancreatitis.

Case Presentation

A 47 year old male with a known case of acute pancreatitis came to the emergency ward of the hospital with clinical features presenting epigastric pain, giddiness and nausea. The laboratory results revealed hence a relapse of acute pancreatitis was diagnosed. The patient was immediately put on treatment by the doctor after screening patient's health status and clinical severity of the disease. The dietician performed a detailed assessment of his dietary intake by taking 24-hour recall studying his food pattern, habits, likes and dislikes which helped in understanding his overall nutritional status of poor lifestyle, erratic meal pattern and high consumptions of zero calorie foods. The anthropometry measurements revealed abdominal adiposity with a waist circumference of 111cms and body mass index were 29kg/m^2 of pre-obese stage, a population based study revealed an increase risk for acute pancreatitis with an increased body mass index status (10) and a 2 fold increase in risk of acute pancreatitis with waist circumference of > 105cm (14). The relapse of his third attack was due to chronic alcoholism and poor lifestyle with other complications. To avoid stimulation of the pancreas, he was nil by mouth (NBM) for 3 tube) was inserted for initiating feeds to balance the nutritional needs. Oral feeds were initiated on absence of abdominal pain, tenderness, absence of other complications, reduction in levels of CRP and pancreatic enzymes. NJ-tube feeds of clear liquids were initiated on the 4th day 30cc/hourly which was well tolerated hence on the following 5th day 60cc/hourly of full liquids was given, 6th day with 70cc/hourly of full liquids, 7th day with 150cc/ 3 hours of full liquids + 2hourly Oral liquids, 8th day with 200cc/ 3 hours of full liquids + 2hourly Oral liquids. On the 9th day soft diet was given to the patient on seeing the improvement in tolerance level. The patient showed quick improvements and hence was discharged on the 10th day with medical and nutritional support. Overall nutritional status was taken care by following a systemic approach a suitable diet was planned which helped in early recovery of the patient with an overall improvement in moderate protein, starting with 1 tsp of fat (coconut oil or ghee), small portion frequent meals, early dinner. A population studies reported the largest increase in incidence of acute pancreatitis compared to chronic (2). One such independent risk factor leading in increasing the risk factors of pancreatitis is alcohol. In an Indian

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study, nationwide prospective survey of chronic pancreatitis concluded that idiopathic pancreatitis was the most common form, followed by alcoholic pancreatitis in India. Alcoholism is the most common cause of chronic pancreatitis worldwide (12). Autodigestion of the pancreas is seen due to the toxic effect of alcohol (13). Our case presented similar observations of pancreatitis induced by chronic alcoholism and the relapse clearly states the involvement of alcohol and no improvement in lifestyle. An improved health status of the patient was noted after one month follow up and a normal nutritionally balanced diet comprising approximately 1800 to 2000kcal was followed by the patient.

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