Introduction

Acute pancreatitis is sudden inflammation of the exocrine pancreatic tissue associated with high mortality, mainly because of systemic inflammatory response and multiple organ failure (Mitchell RM, 2003). Incidence of acute pancreatitis ranging from 5 to 80 cases per 100,000 population, with increasing evidence in western countries (Su KH, 2006). The severity of clinical presentation varies from a mild, self-limiting form to severe disease. About 1-7% of patients are with interstitial pancreatitis, 8-39% with necrotising pancreatitis and 14-62% with infected necrosis. Almost all patients with necrotising pancreatitis without multi organ failure survive, where as those with multi organ failure has a median mortality of 47% (Topazian M, 2003).

Acute pancreatitis has several etiological factors; alcohol and gallstones accounts for 70-80% of cases. Moreover, other etiological factors are viral infections such as mumps and hepatitis, autoimmune disorders such as Systemic Lupus Erythematosus and polyarteritis nodosa, drugs like thiazides, sulfonamides, tetracycline, valproic acid, estrogens, azathioprine as well as hyper calcemia, hyper triglyceridaemia and endoscopic retrograde cholangiopancreatography (Dragnov P, 2005).

Although its pathophysiology is not fully understood, it is mainly characterized by premature activation of digestive enzymes in acinar cells (Frossard JL, 2001), Oxygen and nitrogen derived free radicals (Frossard JL, 2001 and Sweiry JH, 1996), inflammatory cell recruitment (Norman J, 1998), microcirculatory disturbances (Frossard JL, 2001) and imbalance between apoptosis and necrosis (Saluja A, 1996 and Bhatia M, 1998) have been reported to play important role in determining the severity of pancreatitis.

The main features of this condition are pancreatic necrosis and associated sepsis, with both localized and systemic inflammatory syndromes. The most common symptoms are severe epigastric pain radiating to the back, nausea, vomiting, diarrhoea, loss of appetite, fever, chills and shock (Whitcomb DC, 2006).

Repeated attacks of acute pancreatitis have the potential to develop into chronic pancreatitis or pancreatic cancer characterized by fibrosis and loss of acinar cell function. There are no specific therapies for acute pancreatitis. Conflicting or inconclusive data exist regarding
the efficacy of atropine, lexipafant, and low molecular weight dextran, antioxidants such as N-acetyl cystein, indomethacin, interleukin-10 and infliximab (Norton ID, 2001, Fantini L 2006 and Holtz HG, 1995). Several studies and meta-analysis that evaluated the efficacy of somatostatin and octreotide suggest a slight trend toward benefit (Andriulli A, 1998, Paran H, 2000 and Karakoyunlar O, 1999). However the present treatment is mainly aimed at supportive and symptomatic relief. As a result of the limitations of conventional therapy, there is a need to develop novel and safe therapeutic agents to treat acute pancreatitis.

L-Arginine induced pancreatitis is an experimental model of severe necrotizing acute pancreatitis (Mizunuma T, 1984). Depending on the dose and duration of L-Arginine administration different phases of pancreatitis can be studied. This model has advantages like reproducibility and it causes selective, dose dependent acinar cell necrosis (Hegyi P, 2004). Administration of L-Arginine cause damage not only to pancreas but also to lungs, liver and kidney which is very similar to the human disease which may range from a local inflammatory process to a severe pancreas injury associated with extra pancreatic manifestations, such as circulatory, renal or pulmonary complications (O’Brein G, 2005 and Szabolcs A, 2006). As a model of acute pancreatitis, L-Arginine induce it mainly through oxygen, nitrogen derived free radicals generation and inflammatory mediators (Czako L, 2000, Varga IS, 1997, Takacs T, 1996 and Rakonczay Z Jr, 2003). So, the anti oxidant and/or anti-inflammatory drugs could be useful in the management of acute pancreatitis. Previous studies reported that α, β amyrin (Melo MC, 2010), pentoxifylline, alpha lipoic acid (Abdin AA, 2010), N-acetyl cysteine (Bull. Alex. Fac. Med, 2008), Allopurinol (Czako L, 1998), Methyl prednisolone (Melo MC, 2010), Melatonin (Shabir S, 2010) & Selenium (Hardman J, 2005) shown protective effect on L-Arginine induced pancreatitis by virtue of their anti oxidant & anti-inflammatory properties.

Previous study has been reported for anti-oxidant, anti-inflammatory, anti-carcinogenic activity for b-pinene (Abed KF, 2007, Agnihotri S, 2010), lawsone (Chaudhary G, 2010 and Hazra A. 2002), myrcene (Ahmadi-Golsefidi M, 2008) and limonene (Sun J, 2007 and Crowell PL, 1996). However, no study has been reported the protective effect of B-Pinene, Lawsone, Myrcene and Limonene on L-Arginine induced pancreatitis in rats.