Prevention Of The Late Complications Of Acute Pancreatitis

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SUMMARY

UCLA researchers in the Department of Medicine and Surgery have developed a novel therapeutic for the prevention of late inflammatory complications in severe acute pancreatitis patients.

BACKGROUND

Acute pancreatitis, or sudden inflammation of the pancreas, a disease generally caused by gallstones or alcohol abuse, is a common reason for hospitalization worldwide. In some cases, life-threatening complications of acute pancreatitis may occur, such as multi-organ failure. Although multi-organ failure is often fatal, current treatment is supportive, with no known specific interventions. Pancreatic inflammation allows endotoxin (lipopolysaccharide, LPS), originating from Gram-negative bacteria in the gut lumen, to escape into the portal vein that drains the gut, activating inflammatory pathways in the liver and also producing systemic inflammation that damages other organs. Widespread inflammation increases gut permeability and further increases LPS uptake into circulation. In acute pancreatitis patients and also those with burns, trauma, and other acute insults, prevention of LPS release from the gut lumen into the portal vein could therefore decrease inflammation, and lessen the severity of late disease complications.

INNOVATION

UCLA researchers have demonstrated that glucagon-like peptide 2 (GLP-2) acutely inhibits LPS uptake from the gut lumen into the portal vein. Administration of the GLP-2 analog in an acute pancreatitis model decreased pulmonary and hepatic inflammation while decreasing LPS entry into the portal vein. This treatment may decrease circulating LPS in severe acute pancreatitis patients and other patients with multi-organ failure following an acute insult, and could therefore reduce morbidity and mortality in critical illness.

APPLICATIONS

- Treatment of inflammatory complications of severe acute pancreatitis and other causes of multi-organ failure
- Strengthen the gut mucosal barrier

ADVANTAGES

- Decreased lipopolysaccharide release into the portal vein
- Decreased activation of hepatic pro-inflammatory pathways
- Decreased severity of extra-pancreatic complications of severe acute pancreatitis
- An analog of GLP-2 is already FDA approved for treatment of short gut syndrome
- GLP-2 is an endogenous hormone that has been successfully infused into humans
- GLP-2 and analogs have no known short-term adverse effects in humans
- GLP-2 or GLP-2 analog infusion in patients with systemic inflammation may substantially decrease morbidity and mortality from multi-organ failure

STATE OF DEVELOPMENT

This treatment has been successfully tested in mouse models of acute pancreatitis.

PATENT STATUS

Country	Type	Number	Dated	Case
United States Of America	Published Application	20220257722	08/18/2022	2019-463

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OTHER INFORMATION

KEYWORDS

pancreatitis, acute pancreatitis, multi-organ failure, glucagon-like peptide-2, lipopolysaccharide, teduglutide, endotoxin, inflammation

CATEGORIZED AS

- **Medical**
  - Disease: Digestive System
  - Disease: Metabolic/Endocrinology
  - Therapeutics

RELATED CASES

2019-463-0

RELATED MATERIALS

▶ Chen X, Zhao HX, Fu XS, Li CP, Zhong XL. Glucagon-like peptide 2 protects intestinal barrier in severe acute pancreatitis through regulating intestinal epithelial cell proliferation and apoptosis. Pancreas. 2012 Oct;41(7):1080-5.

▶ Kouris GJ, Liu Q, Rossi H, Djuricin G, Gattuso P, Nathan C, Weinstein RA, Prinz RA. The effect of glucagon-like peptide 2 on intestinal permeability and bacterial translocation in acute necrotizing pancreatitis. Am J Surg. 2001 Jun;181(6):571-5. PubMed PMID: 11513789.

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