International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Role of Homoeopathy in Management of Pancreatitis

Dr. Puja Kumari[1], Dr. Sanjay Kumar Singh[2]

[1]PGT, Department of Organon of Medicine, R. B. T. S Govt. Homoeopathic Medical College & Hospital

[2]HOD, Department of Organon of Medicine, R. B. T. S Govt. Homoeopathic Medical College & Hospital

Abstract: ***Inflammation of the pancreas is a defining feature of pancreatitis. The pancreas, a sizable organ located behind the stomach, is responsible for producing several hormones and digesting enzymes. Acute pancreatitis and chronic pancreatitis are the two primary forms. Homoeopathy can be a good alternative in management on pancreatitis. There are many medicines which can be used on the basis of individualization.***

Keywords: Homoeopathy, Inflammation, Pancreatitis, Individualization

1. Introduction

The pancreas is an organ of the digestive system and endocrine system of vertebrates. In humans, it is located in the abdomen behind the stomach and functions as a gland. The development of the human pancreas begins approximately on gestational day 26. It begins as three endodermal buds on the caudal portion of the foregut. The dorsal bud gives rise to the majority of pancreatic tissues, namely, the upper portion of the head, isthmus, and tail. The right ventral bud develops into the inferior portion of the head. In most cases, the left ventral bud will gradually regress, if it does not, it can lead to the congenital malformation known as an annular pancreas. The pancreas is a mixed or heterocrine gland, i. e., it has both an endocrine and a digestive exocrine function.99% of the pancreas is exocrine and 1% is endocrine. As an endocrine gland, it functions mostly to regulate blood sugar levels, secreting the hormones insulin, glucagon, somatostatin and pancreatic polypeptide. As a part of the digestive system, it functions as an exocrine gland secreting pancreatic juice into the duodenum through the pancreatic duct. This juice contains bicarbonate, which neutralizes acid entering the duodenum from the stomach; and digestive enzymes, which break down carbohydrates, proteins and fats in food entering the duodenum from the stomach.

Types
Pancreatitis is inflammation of the pancreases. Pancreatitis occurs when the enzymes damage the pancreas, which causes inflammation. Pancreatitis can be acute or chronic. Either form is serious and can lead to complications.

Acute pancreatitis
Acute pancreatitis is the sudden development of symptoms related to inflammation of the pancreas. The diagnosis of acute pancreatitis is made by a combination of symptoms, physical exam findings, and laboratory tests including amylase and lipase. If the diagnosis is uncertain, abdominal imaging studies such as a computed tomography (CT) scan may also be necessary. Some symptoms are -

- Gradual or sudden onset of severe pain in the upper abdomen that may radiate to the back and usually persists for several days.
- Nausea and vomiting
- Fever

Chronic pancreatitis
Chronic pancreatitis is a disease characterized by persistent symptoms and dysfunction related to irreversible damage of the pancreas. Symptoms include chronic abdominal pain, diarrhoea and blood sugar elevation. The pancreas can no longer produce the necessary enzymes of digestion and sugar control. Fat cannot be broken down properly and will pass through the body creating greasy stools. Food products cannot be broken down properly resulting in nutrient deficiency. Insulin is no longer produced at the level needed resulting in elevated blood sugars and either a new diagnosis of diabetes or worsening diabetes if it is already is present.

Pathophysiology of acute pancreatitis -
Acute pancreatitis is a common clinical condition resulting from an acute injury to the pancreas usually causing self - limiting pancreatic inflammation. A severe multi - system inflammatory response can occur in up to 25% of patients diagnosed with pancreatitis, in which 30% to 50% will expire. There are multiple etiologies responsible for AP, with the two most common being gallstones, which account for up to 40% of cases, and alcohol, which is responsible for approximately 30% of cases. Other causes of AP include the following: medications such as angiotensin - converting enzyme (ACE) inhibitors, sulfa - based drugs, furosemide, azathioprine, 6 - mercaptopurine, and valproate; infections such as coxsackievirus B, cytomegalovirus, and hepatitis A and E; inherited mutations in cationic trypsinogen (PRSS1) or cystic fibrosis; mechanical etiologies such endoscopic retrograde cholangiopancreatography (ERCP), abdominal trauma, pancreatic cancer, sphincter of Oddi stenosis, and pancreatic divisum; and metabolic causes such as hypertriglyceridemia and hypercalcemia.

The pathophysiology of gallstone pancreatitis is a result of mechanical obstruction of the ampulla from a stone or edema caused by the passage of the stone through the duct inducing pancreatic ductal hypertension and acinar cellular injury. The metabolization of alcohol into toxic metabolites increases enzymatic content and destabilizes lysosomal and zymogen granules, with sustained increases in calcium overload, and

activated pancreatic stellate cells potentiate acinar cell autodigestion and cell death. Irrespective of the inciting injury, AP is a consequence of acinar cell disruption and enzymatic release triggering intra - acinar zymogen activation and cellular autodigestion. The effector enzymes trypsin, chymotrypsin, elastase, phospholipase A2, and lipase break down tissue membranes, causing apoptosis, necrosis, edema, vascular damage, haemorrhage, and a subsequent localized and systemic inflammatory response. The severity of AP is thought to be correlated with the degree of necrosis related to apoptosis. A higher necrosis - to - apoptosis ratio correlates to the increasing severity of illness.

Diagnosis of acute pancreatitis -

The diagnostic algorithm for AP encompasses laboratory markers and radiographic imaging to support the clinical presentation of a patient presenting with severe epigastric abdominal pain with or without radiation, who has a history of alcohol use or gallstones. The diagnosis can be made if at least two of the following criteria are met: abdominal pain consistent with the disease process, serum amylase and/or lipase greater than three times the upper limit of normal, and characteristic findings on CECT. Serum pancreatic enzyme levels peak on the first day and normalize around three to seven days, although lipase has greater sensitivity and specificity than amylase both early and later in the disease course. Urine trypsinogen - 2 will also help support the diagnosis as the sensitivity and specificity are greater than 90% for AP. Other supporting laboratory values are elevated white blood cell (WBC) count, hematocrit, and blood urea nitrogen (BUN) due to the third - spacing of fluids. Hyperglycemia may result from pancreatic insufficiency and hypocalcemia due to saponification of peripancreatic fatty tissue.

Pathophysiology of chronic pancreatitis -

Chronic pancreatitis (CP) is persistent long - standing inflammation of the pancreas that results in permanent structural damage marked by fibrosis and ductal strictures leading to an irreversible decrease in exocrine and endocrine pancreatic function. The etiology of CP is multifactorial, although the most common inciting factors include chronic alcohol consumption, which accounts for over 50% of cases, and tobacco smoking. Studies show an independent dose - response relationship between both alcohol and smoking in the development of CP and it is likely that both risk factors exhibit a synergistic effect. However, not all patients with these risk factors develop AP or CP; suggesting other cofactors are involved. Other etiologic factors include genetic mutations in the cationic trypsinogen gene (PRSS1), serine peptidase inhibitor Kazal type 1 (SPINK1), and the cystic fibrosis transmembrane regulator (CFTR). Chronic obstructive causes of CP include pancreatic ductal strictures, tumor mass effect, pancreatic divisum, and sphincter of Oddi dysfunction. There are several autoimmune predisposing factors including systemic Immunoglobulin G4 (IgG4) disease (type 1) and idiopathic (type 2). Tropical pancreatitis is an idiopathic cause in areas such as India, Indonesia, and Nigeria, marked by an early age of onset, large ductal calculi, and accelerated disease course. Additional risk factors include chronic hypercalcemia and hyperlipidemia.

Chronic damage and remodelling of pancreatic parenchyma led to exocrine and endocrine pancreatic insufficiency. When protease and lipase secretions are reduced to less than 10% of normal, the patient develops malabsorption characterized by steatorrhea malnutrition and weight loss. Glucose intolerance may ensue at any time due to insulin deficiency, although overt insulin - dependent diabetes mellitus usually occurs late in the disease course. Patients suffering from CP are at a significantly higher risk of developing hypoglycemia due to the resultant damage and reduction in alpha cells. Additional complications associated with CP include the formation of pseudocysts, bile duct or duodenal obstruction, pancreatic duct disruption resulting in ascites or pleural effusion, splenic vein thrombosis, which can cause gastric varices, pseudoaneurysms of arteries near the pancreas or pseudocyst, and an increased risk of pancreatic adenocarcinoma with the risk being greatest in hereditary and tropical pancreatitis. Signs and symptoms of CP include constant or intermittent epigastric abdominal pain, which is usually postprandial and relieved by sitting upright or leaning forward. Glucose intolerance, hypoglycemia, weight loss, fatigue, abdominal distention, and steatorrhea are all classical signs of CP. About 10% to 15% of patients report no pain and present only with symptoms of malabsorption.

Diagnosis of chronic pancreatitis -

The diagnosis of CP relies on clinical assessment, imaging, and pancreatic function tests. MRI coupled with magnetic resonance cholangiopancreatography (MRCP) is the preferred imaging modality as it can reveal pancreatic masses and provide optimal visualization of ductal abnormalities consistent with CP. The use of IV secretin during MRCP increases sensitivity for detecting ductal abnormalities and allows for a functional assessment. Pancreatic function tests are useful when imaging studies are non - diagnostic. Direct tests involve IV infusion of cholecystokinin (CCK) or secretin to measure the production of digestive enzymes or bicarbonate, respectively. The diagnostic accuracy is highest with these tests when conducted early in the disease course. However, these interventions are invasive, time - consuming, and not well standardized. Indirect tests involve analysis of blood or stool samples. Serum levels of trypsinogen less than 20 ng/mL are highly specific for CP. A 72 - hour fecal fat test in patients on a high - fat diet is diagnostic for steatorrhea. Decreased levels of fecal chymotrypsin and elastase suggest pancreatic insufficiency. The indirect tests are readily available, more convenient, less invasive, and inexpensive, although they are less accurate in diagnosing the disease in its earlier stages.

Homoeopathic Management

The aim of homeopathy is not only to treat pancreatitis but to address its underlying cause and individual susceptibility. As far as therapeutic medication is concerned, several well - proved medicines are available for pancreatitis treatment that can be selected on the basis of cause, sensations, and modalities of the complaints. For individualized remedy selection and treatment.

On the basis of sign & symptom following homoeopathic medicine can be used therapeutically in pancreatitis -

- ***Carbolic Acid*** - Appetite lost. desire for stimulants and tobacco. Constant belching, nausea, vomiting, dark olive green. Heat rises up oesophagus. Flatulent distention of

stomach and abdomen. Painful flatulence often marked in one part of the bowel. [Sulpho - Carbonate of Soda.] fermentative dyspepsia with bad taste and breath.

- ***Atropinum*** - It has shown an affinity for the pancreas, which relates it to Iod., Kali. iod., and Iris. Vomiting of food, after hot drinks, with severe sticking pains in umbilical region., better after vomiting. Region of stomach very sensitive, swelling in pyloric region.
- ***Bar. Mur***. - There is great indigestion. Below stomach to left a hardness from which paroxysms of dyspnoea come. Induration of pancreas. Distressing throbbing in abdomen (abdominal aneurism). Vomiting of a small quantity of water, with nausea. Sickness. Pressure on the stomach, with spasm.
- ***Kali iod.*** **-** Cutting in right side, cutting in attacks in afternoon, with burning and nausea, inclination to eructation's, which afterwards occur, itching externally about umbilicus and inclination to emission of flatus. Griping and burning.
- ***Iris.*** **-** Thyroid, Pancrease, salivary, intestinal glands, and gastro - intestinal mucous membrane, are especially affected. Increases the flow of bile. Sick headaches and cholera morbus are a special therapeutic field for its action.

Some rubrics related to Pancreatitis -

Complete Repertory -
Abdomen - Inflammation - Pancreas
Abdomen - Pancreas - Affection
Abdomen - pain - Pancreas
Abdomen - Sensitive, tenderness - Pancreas

Boericke Repertory
Abdomen - Pancreas affection
Clarke Repertory
Clinical – Pancreatitis

Gentry Repertory
Hypochondrium – Pancreatitis

2. Conclusion

In conclusion, a comprehensive understanding of pancreatitis is crucial for both healthcare professionals and the general population. Continued research, early detection strategies and advancements in treatment modalities are essential in addressing the challenges posed by pancreatitis and improving the overall prognosis for individuals affected by this condition. Homoeopathy also plays an important role in the treatment of pancreatitis. It also eliminates the chances of recurrence of pancreatitis in the most simple and harmless way.

References

[1] https: //www.ncbi. nlm. nih. gov/pmc/articles/PMC9454414/ (22 - 2 - 24)
[2] Penman D. Ian, et al, Davidson's Principles and Practice of Medicine.22nd Edition. China; Elsevier; 2014
[3] https: //pubmed. ncbi. nlm. nih. gov/33496779/ (22 - 2 - 24)
[4] https: //www.ncbi. nlm. nih. gov/pmc/articles/PMC2886461/ (23 - 2 - 24)
[5] Boericke W. Pocket Manual of Homoeopathic Materia Medica & Repertory, B. Jain Publishers P (ltd); 2002.
[6] Clarke Henery John, A Dictionary of Practical Materia Medica, B Jain Publishers Pvt Ltd, 2005