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# Polyarteritis Nodosa Presenting as acute Pancreatitis

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**Abstract:- Acute pancreatitis is a common presentation to the critical care uniti, common causes are gallstone, alcohol etcii. The pancreas is a major organ in the body that regulates major hormonal functions.It essentially carries out two roles an exocrine capability that assists in digestion and an endocrine capability that controls with blood sugar levelsiii.This case report features the significance of monitoring the autoimmune reason of acute pancreatitisPolyarteritis nodosa (PAN) is one of the uncommon reasons for acute necrotizing pancreatitis. PAN can result in inflammation of arterioles causing arteriolar ectasia, aneurysm formation, and thrombosis, resulting in organ ischemiaiv.**

***Keywords:-*** *Acute pancreatitis, Polyarteritis nodosa.*

## I. INTRODUCTION

Acute pancreatitis can deteriorate patient general condition and may even lead to death[v]. Polyarteritis nodosa **s** a systemic necrotizing inflammation of blood vessels (vasculitis) affecting medium-sized muscular arteries, can affect the arteries of the kidneys and other major organs expect lungs' circulation[vi]. But in this case patient developed acute pancreatitis due to the reduce blood supply to the organ, this is a rare presentation of polyarteritis nodosa. In this case report we discuss in detail about the polyarteritis nodosa and acute pancreatitis.

## II. CASE REPORT

The case is of a 32-year-old male patient know instance of hypertension on irregular medication, presented to the emergency department with c/o with severe abdominal pain, vomiting, reduced urine output and confusion, abdominal pain was very severe, patient was bending forward to get relief from the pain. He drinks alcohol occasionally. No history of any insect bite (scorpion bite)

On examination, he is poorly built and nourished, dehydrated,rash on his right leg.Vitals BP- 160/100 mmHg, PR- 98 beats/minute sinus rhythm, normal volume character, and condition of the vessel wall, oxygen saturation-94%, respiratory rate – 25 cycles/epigastric tenderness was present.The patient was admitted to critical care. Blood investigations showed: HB- 9.8 (13.0 - 18.0 gm/dl ), TC-11020,N-90% (4000.0 - 10000.0 / µl ),CRP-96 (<6),ESR-81, URE-pus cells 6-8, albumin- 1+(foley's catheter was not inserted), sugar 70 mg/dl, urea – 192(8.0 - 49.0 mg/dl), serum creatinine- 10.6 (0.7 - 1.2 mg/dl) serum sodium- 129 mmol/l (135.0 - 148.0 (mmol/l), serum potassium- 4.8 mmol/l (3.5 - 5.1 mmol/l), urine spot sodium- 46, fractional excretion of urea – >1%, S. lipase- 2198 (13.0 - 60.0 IU/L), S. amylase- (2135 25.0 - 86.0 IU/L), S. Total bilirubin- 1.2 (0.0 - 1.2 mg/dL), SGPT- 230 (0.0 - 40.0 IU/L ), SGOT -183 (0.0 - 50.0 IU/L ),ALP- 164 (35.0 - 129.0 IU/L ) Total protein 5.1 (6.4 - 8.3 g/dl ), S. albumin - 2.8 (3.5 - 5.2 g/dl ) S. globulin- 2.3 (2.0 - 3.5 g/dl ), S.calcium- 8.3 (8.5 - 10.1 mg/dL ), S. triglycerides – 287 (50.0 - 150.0 mg/dL ) reset of the lipid profile are within normal in limits. Chest x-ray within normal limits. USG abdomen and pelvis -Mildly bulky pancreatic head – suggested serum amylase/lipase correlation, Minimal perinephric fluid in bilateral kidneys and Grade I fatty liver. MRI abdomen and pelvis-Pancreas appears enlarged with increased signal on T2WI and abnormally low signal on T1WI due to edema fat suppression very important in highlighting edema and fluid around pancreas on T2WI**.**2D ECHO- normal LV function, IVC- plethoric.The patient was kept on nil per oral. A minimal amount of IV fluids were given according to IVC and cardiac status.Given reduced urine output and altered kidney function test, the patient was started on hemodialysis after inserting a temporary dialysis catheter into the right IJV. After the first dialysis patient started to have urine started coming out. The patient required 5 dialyses to normalize the kidney function test and to void urine normally.The patient was symptomatically improved and shifted to the ward. From there patient told us that he had a history of arthralgia, weight loss, testicular pain and weakness of his right ankle with a rash on his right leg.He suffered a right-side foot drop and sensory impairment across the right dorsum of the foot, but his deep tendon reflexes were still intact. He felt a rash on his right leg and a weakness in his right ankle two weeks prior to the admission. He tested negative for rheumatoid factor, ANA, ANCA (including MPO and PR3), and other autoimmune antibodies. His HbA1c and homocysteine levels were within normal ranges, and he tested HIV-negative. Additionally negative were his hepatitis B and C statuses. Peroneal nerve palsy on the right side was discovered by a nerve conduction investigation. A deep punch biopsy from a vasculitic cutaneous lesion revealed fibrinoid necrosis and segmental transmural inflammation of the muscle arteries as well as a polymorphonuclear infiltration that was suggestive of a medium artery vasculitis. The patient was diagnosed to have polyarteritis nodosa. Digital subtraction angiography of aorta was not done. The patient was referred to a rheumatologist for better care.

## III. DISCUSSION

### *A. POLYARTERITIS NODOSA*

a) DEFINITION

The renal and visceral arteries are frequently involved in the multisystem, necrotizing vasculitis of small- and medium-sized muscle arteries known as polyarteritis nodosa[vii].

b) PATHOLOGY AND PATHOGENESIS

Small- and medium-sized muscle arteries exhibit necrotizing inflammation as the vascular lesion in polyarteritis nodosa. The segmental lesions frequently affect arteries' bifurcations and

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branches. They might enlarge circumferentially to affect nearby veins. The extent and location of vascular involvement and the consequent ischemia alterations are reflected in the clinicopathologic findings, which also show the involvement of many organ systems. Polymorphonuclear neutrophils infiltrate all layers of the vessel wall and perivascular regions during the acute stages of the disease, which causes intimal proliferation and vessel wall degradation. As the lesions advance to the subacute and chronic stages, mononuclear cells invade the region.

c) CLINICAL AND LABORATORY MANIFESTATIONS

Polyarteritis nodosa is characterized by nonspecific signs and symptoms. Over half of cases involve fever, weight loss, and malaise. Patients typically exhibit nebulous symptoms at first, such as weakness, malaise, headaches, abdominal pain, and myalgias, which can develop into a fulminant sickness very quickly. The presenting clinical picture and the entire course of the illness may also be dominated by complaints specific to vascular involvement within a given organ system. Renal involvement in polyarteritis nodosa typically appears as hypertension, renal insufficiency, or haemorrhage from microaneurysms.

For polyarteritis nodosa, there are no diagnostic serologic assays available. The leukocyte count is increased with a preponderance of neutrophils in more than 75% of patients. A high ESR is nearly always present along with the anemia of chronic illness. The specific organ implicated is reflected in other typical test findings. The possibility of hypergammaglobulinemia should prompt a screening for hepatitis B and C in all patients. Patients with polyarteritis seldom have antibodies to proteinase-3 (ANCA) or myeloperoxidase[viii].

d) DIAGNOSIS

The presentation of typical vasculitis features on biopsy material of the affected organs is the basis for the diagnosis of polyarteritis nodosa. The arteriographic demonstration of implicated vessels, particularly in the form of aneurysms of small- and medium-sized arteries in the renal, hepatic, and visceral vasculature, is adequate to make the diagnosis in the absence of readily accessible tissue for biopsy[ix].

e) TREATMENT

Nodular polyarteritis Untreated polyarteritis nodosa has a very bad prognosis, with a reported 10-to-20% 5-year survival rate. Death is typically caused by cardiovascular reasons and gastrointestinal problems, including bowel infarcts and perforations. With polyarteritis nodosa, intractable hypertension frequently exacerbates failure in other organ systems, such as the kidneys, heart, and CNS, increasing late morbidity and death. Since the development of treatment, the survival rate has significantly increased. Prednisone and cyclophosphamide together have been found to have positive treatment outcomes in polyarteritis nodosa[x].

*B. ACUTE PANCREATITIS*

The pancreas is inflamed by pancreatitis. Pancreatic inflammation can range from a minor, self-limiting condition to the total necrosis of the organ. Acute pancreatitis by definition develops in the backdrop of a healthy pancreas and can go away and return to normal after. Contrary to chronic pancreatitis, which results in permanent alterations[xi].

a) CAUSES OF ACUTE PANCREATITIS

A most common cause of pancreatitis is gall stones, other causes are Ethanol, Trauma, Steroids, Mumps (other viruses include Coxsackie B), Autoimmune (e.g. polyarteritis nodosa), Ascaris infection, Scorpion venom, Hypertriglyceridaemia, Hyperchylomicronaemia, Hypercalcaemia, Hypothermia, ERCP, Drugs (azathioprine, mesalazine, didanosine, bendroflumethiazide, furosemide, pentamidine, steroids, sodium valproate)[xii].

b) DIAGNOSIS

Serum lipase or amylase levels that are at least three times the upper limit of the normal range, abdominal pain consistent with acute pancreatitis, and acute pancreatitis findings on cross-sectional imaging (computed tomography [CT] or magnetic resonance imaging) are all necessary for the diagnosis of acute pancreatitis)[xiii].

*C. TREATMENT*

a) FLUID RESUSCITATION

Numerous adverse predictive aspects of acute pancreatitis are caused by significant third-space loss and intravascular volume depletion (hemoconcentration and azotemia). Aggressive fluid therapy was initially thought to be the best course of treatment. Without improving clinical outcomes, early aggressive fluid resuscitation led to a higher incidence of fluid overload. A bolus of 10 ml per kg for patients with hypovolemia or no bolus for those with normovolemia was followed by 1.5 ml per kg of body weight per hour for moderate fluid resuscitation. One study found that Ringer's lactate reduced inflammatory markers more effectively than regular saline. It is practicable to monitor the blood urea nitrogen level, hematocrit, and hourly urine output in addition to clinical cardiac monitoring for fluid status[xiv].

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b) FEEDING

In patients with acute pancreatitis, whole parenteral feeding is now recognised to be more costly, hazardous, and ineffective than enteral nutrition. Before starting oral feeding, there is no need for the pain to completely subside or for the pancreatic enzyme levels to return to normal in patients with moderate acute pancreatitis who do not have organ failure or necrosis.In comparison to a clear-liquid diet with a gradual transition to solid meals, a low-fat soft or solid diet is secure and linked to shorter hospital stays. In the absence of significant pain, nausea, vomiting, and ileus, the majority of patients with moderate acute pancreatitis can begin eating low-fat foods shortly after being admitted (all of which are unusual in mild cases of acute pancreatitis).By day 5, if symptoms are still severe or the patient is unable to tolerate attempts at oral feeding, artificial enteral feeding may be necessary. The least amount of pancreatic secretion is achieved with nasojejunal tube feeding, although randomised trials and a meta-analysis have demonstrated that nasogastric or nasoduodenal feeding are clinically similar. Total parenteral nutrition and feeding using intricate, deeply positioned intestinal tubes have been replaced by straightforward tube feeding.[xv].

c) ANTIBIOTIC THERAPY

Although the development of infected pancreatic necrosis confers a significant risk of death, well-designed trials and meta-analyses have shown no benefit of prophylactic antibiotics. Prophylaxis with antibiotic therapy is not recommended for any type of acute pancreatitis unless the infection is suspected or has been confirmed[xvi].

d) ENDOSCOPIC THERAPY

ERCP is used primarily in patients with gallstone pancreatitis and is indicated in those who have evidence of cholangitis superimposed on gallstone pancreatitis. This procedure is also a reasonable treatment in patients with documented choledocholithiasis on imaging or findings strongly suggestive of a persistent bile duct stone (e.g., jaundice, a progressive rise in the results of liver biochemical studies, or a persistently dilated bile duct). ERCP is not beneficial in the absence of these features, in mild cases of acute gallstone pancreatitis, or as a diagnostic test before cholecystectomyEndoscopic ultrasonography is used as a platform for minimally invasive treatment of a pancreatic pseudocyst or walled-off pancreatic necrosis[xvii].

e) TREATMENT OF FLUID COLLECTIONS AND NECROSIS

Acute peripancreatic fluid collections do not require therapy. Symptomatic pseudocysts are managed primarily with the use of endoscopic techniques, depending on local expertise.Necrotizing pancreatitis includes pancreatic gland necrosis and peripancreatic fat necrosis. In the initial phases, the necrotic collection is a mix of semisolid and solid tissue. Over a period of 4 weeks or longer, the collection becomes more liquid and becomes encapsulated by a visible wall. At this point, the process is termed walled-off pancreatic necrosis[xviii].Sterile necrosis does not require therapy except in the rare case of a collection that obstructs a nearby viscus (e.g., duodenal, bile duct, or gastric obstruction).The development of infection in the necrotic collection is the main indication for therapy[xix]. Such infections are rare in the first 2 weeks of the illness. The infection is usually monomicrobial and can involve gram-negative rods, enterobacter species, or gram-positive organisms, including Staphylococcus. Drug-resistant organisms are increasingly prevalent. The development of fever, leukocytosis and increasing abdominal pain suggests an infection of the necrotic tissue. A CT scan may reveal evidence of air bubbles in the necrotic cavity.Therapy begins with the initiation of broad-spectrum antibiotics that penetrate the necrotic tissue. Aspiration and culture of the collection are not required. In current practice, efforts are made to delay any invasive intervention for at least 4 weeks to allow for walling off of the necrotic collection that is, demarcation of the boundary between necrotic and healthy tissue, softening and liquefaction of the contents, and formation of a mature wall around the collection. This delay makes drainage and debridement easier and reduces the risk of complications or death.Delayed intervention is possible in most patients whose condition remains reasonably stable, without the development of a progressive sepsis syndrome. In patients whose condition is not stable, the initial placement of a percutaneous drain in the collection is often enough to reduce sepsis and allow the 4-week delay to be continued[xx].

f) COMPLICATIONS

The classifications of moderately severe pancreatitis and severe pancreatitis are defined by the presence of complications that are systemic, local, or both. Systemic complications include failure of an organ system (respiratory, cardiovascular, or renal) and exacerbation of a preexisting disorder (e.g., chronic obstructive pulmonary disease, heart failure, or chronic liver disease). Local complications comprise peripancreatic fluid collections or pseudocysts and pancreatic or peripancreatic necrosis, whether

sterile or infected. In this classification system, the persistent failure of an organ system (i.e., lasting more than 48 hours) is the prime determinant of a poor outcome[xxi].

g) LONG-TERM CONSEQUENCES OF ACUTE PANCREATITIS

After acute pancreatitis, pancreatic exocrine and endocrine dysfunction develops in approximately 20 to 30% of patients and clear-cut chronic pancreatitis develops in one-third to one-half of those patients. Risk factors for the transition to recurrent attacks and chronic pancreatitis include the severity of the initial attack, the degree of pancreatic necrosis, and the cause of acute pancreatitis. In particular, long-term, heavy alcohol use as the cause and smoking as a cofactor dramatically increase the risk of a transition to chronic pancreatitis and reinforce the need for strong efforts to encourage abstinence[xxii].

## IV. CONCLUSION

It may be uncommon for PAN, a potentially fatal vasculitis, to appear with acute pancreatitis. A diseased artery biopsy and angiography may be helpful in making an early diagnosis, but waiting too long to make the right diagnosis could be fatal.

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