# Octreotide in management of acute pancreatitis

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# List of abbreviations

| Abbreviation | Text |
|---|---|
| RR | Relative risk (statistic) |
| OR | Odd ratio(statistic) |
| Et al | Et Alia ( Latin) And others ( English) |
| ARDS | Adult respiratory distress syndrome |

# Abstract

**Background:** Acute pancreatitis is a common cause of hospitalization and a major source of morbidity worldwide. To date ,there exist no specific treatment for this disease.The idea that the pharmacological inhibition of exocrine pancreatic secretion might serves as a specific treatment for acute pancreatitis has been a subject of controversy for more than three decades. Activated pancreatic enzymes seem to be involved in the generation and progression of this "autodigestive" acute inflammatory disease, and it was therefore hypothesized that the administration of somatostatin or its analogue octreotide might help prevent progression of the disease and its complications.

**Patients and method:** A prospective study conducted at Baghdad teaching hospital / Medical city from November 2015 to November 2017 , seventy eight patients were randomly assigned into two groups: control group (n=40)who received conservative treatment only and treatment group(n=38) the same conservative treatment to control group plus octreotide 0.1 s.c injection every 6 hours for maximum 7 consecutive days. The patients were assessed for hospital stay duration, development of complications and mortality.

**Aim of the study**: To investigate the curative effect of octreotide in the treatment of acute pancreatitis.

**Results:** The two groups matched with regard to age, gender, duration between onset of symptoms and admission ,severity and etiology of AP. Mortality and the development of complications were lower in treatment group than in control group (ARDS 7.89% vs. 17.5% ,septicemia 18.42% vs. 32.5% , renal failure 5.26% vs.10% , Abscess 2.63% vs. 7.5% ,pseudocyst 7.89% vs.15% , death 10.5% vs. 17.5% ), but statistically were not significant (P-value >0.05).

**Conclusion:** Although it was statistically not significant due to small sample size of the study ,it seems that octreotide may be effective in lowering morbidity and mortality in acute pancreatitis.



Key words: Octreotide , acute pancreatitis.

# Introduction

Acute pancreatitis is acommon cause of hospitalization and amajor source of morbidity worldwide[1-2].To date ,there exist no specific treatment for this disease[3-4].

Worldwide, the incidence of acute pancreatitis is between 4.9 and 73.4 cases per 100000.[5,6,7]

Acute pancreatitis is clinically classified into mild and severe forms[1] .

The majority of patients suffer from mild acute pancreatitis,aself limiting disease which responds well to conservative treatment. In up to 20% of pateints with acute pancreatitis ,however the disease progress to asevere form involving both pancreatic and extrapancreatic necrosis.while the morbidity and mortality of mild acute pancreatitis are low ,patients with severe acute pancreatitis commonly develop systemic complication and have a mortality of (10%-50%) despite all currently available treatment strategies.[9,10,11,12,13,14,15]

Although the initial steps in the pathogenesis of acute pancreatitis are not fully understood ,the term"autodigestion",introduced by chiari in 1896,summarises prevailing concept that the acute inflammation is triggered by the activation of digestive enzymes.[16,17]

## Causes of acute pancreatitis

Gallstones are the most common cause.[18,19] Migrating gallstones cause transient obstruction of the pancreatic duct, a mechanism shared by other recognized causes (e.g., endoscopic retrograde cholangiopancreatography [ERCP]), as well as purported causes (i.e., pancreas divisum and sphincter of Oddi dysfunction). [20] Alcohol is the second most common cause of acute pancreatitis.
Prolonged alcohol use (four to five drinks daily over a period of more than 5 years) is required for alcohol-associated pancreatitis.[21]

Drugs appear to cause less than 5% of all cases of acute pancreatitis, although hundreds of drugs have been implicated.[22] The drugs most strongly associated with the disorder are azathioprine, 6-mercaptopurine, didanosine, valproic acid, angiotensin-converting–enzyme inhibitors, and mesalamine.

pancreatitis caused by drugs is usually mild. Recent data do not support a role for glucagon-like peptide 1 mimetics in causing pancreatitis. [23] It is common for patients to be taking one of the many drugs associated with ncreatitis when they are admitted to the hospital with acute pancreatitis,[24] but it is exceedingly difficult to determine whether the drug is responsible.

Mutations and polymorphisms in a number of genes are associated with acute (and chronic) pancreatitis[25]

The cause of acute pancreatitis often cannot be established, and the proportion of persons who are considered to have idiopathic acute pancreatitis increases with age. [26] Morbid obesity is a risk factor for acute pancreatitis [19,27] and for severe acute pancreatitis.[28] Type 2 diabetes increases the risk of acute pancreatitis by a factor of 2 or 3.[19]

| **Cause** | **Approximate Frequency** |
|---|---|
| Gallstones | 40% |
| Alcohol | 30% |
| Hypertriglyceridemia | 2–5% |
| Genetic causes | Not known |
| Drugs | <5% |
| Autoimmune cause | <1% |
| ERCP | 5-10% among patients under-going ERCP |
| Trauma | <1% |
| infection | <1% |
| Surgical complication | 5-10% among patients under-going cardiopulmonary bypass |
| Celiac disease and Crohn's disease, pancreas divisum (controversial), and sphincter of Oddi dysfunction (very controversial) | Rare |
| Diabetes, obesity, and smoking | Common |

## Diagnosis and Classification

Accurate diagnosis of acute pancreatitis requires at least two of the following three diagnostic features : abdominal pain consistent with acute pancreatitis, serum lipase or amylase levels that are at least 3 times the upper limit of the normal range, and findings of acute pancreatitis on cross-sectional imaging (computed tomography [CT] or magnetic resonance imaging. [29]

## Prediction of Severity

Several scoring systems have been developed to incorporate clinical, radiographic, and laboratory findings in various combinations: Acute Physiology and Chronic Health Evaluation II (APACHE II), APACHE combined with scoring for obesity (APACHE-O), the Glasgow scoring system, the Harmless Acute Pancreatitis Score (HAPS), PANC 3, the Japanese Severity Score(JSS), Pancreatitis Outcome Prediction (POP), and the Bedside Index for Severity in Acute Pancreatitis (BISAP). [30] These scoring systems all have a high false positive rate (i.e., in many patients with high scores, severe pancreatitis does not develop), which is an unavoidable consequence of the fact that in most patients, severe disease does not develop. The scoring systems are complex and cumbersome and not routinely used.

These scoring systems cannot replace ongoing evaluation by an experienced clinician. The presence of the systemic inflammatory response syndrome (SIRS) is usually obvious, although it may not be recognized.

SIRS can be diagnosed on the basis of four routine clinical measurements, with findings of two or more of the following values: temperature, below 36°C or above 38°C; pulse, greater than 90 beats per minute; respiratory rate, greater than 20 breaths per minute (or partial pressure of arterial carbon dioxide, <32 mm Hg); and white-cell count, lower than 4000 or higher than 12,000 per cubic millimeter. SIRS that persists for 48 hours or more after the onset of symptoms is indicative of a poor prognosis.

Recent guidelines [31,32] recommend using demographic and clinical factors at admission (advanced age, high body-mass index, and coexisting conditions), simple laboratory values at admission and during the next 24 to 48 hours (hematocrit, >44%; blood urea nitrogen level, >20 mg per deciliter [7 mmol per liter]; or creatinine level, >1.8 mg per deciliter [159 µmol per liter]), and the presence of SIRS to identify patients who are at greatest risk for severe disease

and most likely to benefit from a high-intensity nursing unit. During the first 48 to 72 hours, a rising hematocrit or blood urea nitrogen or creatinine level, persistent SIRS after adequate fluid resuscitation, or the presence of pancreatic or peripancreatic necrosis on cross-sectional imaging constitutes evidence of evolving severe pancreatitis.[33]

## Management of Acute Pancreatitis

### Fluid Resuscitation

Aggressive fluid administration during the first 24 hours reduces morbidity and mortality,[34,35] current guidelines provide directions for early and vigorous fluid administration. Vigorous fluid therapy is most important during the first 12 to 24 hours after the onset of symptoms and is of little value after 24 hours. Administration of a balanced crystalloid solution has been recommended at a rate of 200 to 500 ml per hour, or 5 to 10 ml per kilogram o fbody weight per hour, which usually amounts to 2500 to 4000 ml within the first 24 hours.[31,32] Clinical cardiopulmonary monitoring for fluid status, hourly measurement of urine output, and monitoring of the blood urea nitrogen level and hematocrit are practical ways to gauge the adequacy of fluid therapy.[36,37]

### Feeding

In patients with mild acute pancreatitis who do not have organ failure or necrosis, there is no need for complete resolution of pain or normalization of pancreatic enzyme levels before oral feeding is started.[38]

A lowfat soft or solid diet is safe and associated with shorter hospital stays than is a clear-liquid diet with slow advancement to solid foods.[39,40] Most patients with mild acute pancreatitis can be started on a low-fat diet soon after admission. A need for artificial enteral feeding may be predicted by day 5, on the basis of symptoms that continue to be severe or an inability to tolerate attempts at oral feeding.Total parenteral nutrition should be reserved for the rare cases in which enteral nutrition is not tolerated or nutritional goals are not met.[41]

## Antibiotic therapy

Although the development of infected pancreatic necrosis confers a significant risk of death, well designed trials[42,43] and meta-analyses[44,45] have shown no benefit of prophylactic antibiotics. Prophylaxis with antibiotic therapy is not recommended for any type of acute pancreatitis unless infection is suspected or has been confirmed .[31,32] Nonetheless, many patients continue to receive prophylactic antibiotics despite guidelines to the contrary.[46,47]

## 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[31,32,48]

## Treatment of Fluid Collections and Necrosis

Acute peripancreatic fluid collection do not require therapy. Symptomatic pseudocysts are managed primarily with the use of endoscopic techniques.[49,50] 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. 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 infection of the necrotic tissue. A CT scan may reveal evidence of air bubbles in the necrotic cavity. Therapy begins with the initiation of broads pectrum antibiotics that penetrate the necrotic tissue. Aspiration and culture of the collection are not required.[51]A number of minimally invasive techniques (e.g., percutaneous, endoscopic, laparoscopic, and retroperitoneal approaches [52,53] are available to debride infected necrotic tissue in patients with walled-off pancreatic necrosis. A small proportion of patients with infected necrosis can be treated with antibiotics alone.[53,54]

## Octreotide

Octreotide , along acting octapeptide analogue of the native hormone somatostatin, became clinically available in 1982.[55] It has proved to be a powerful inhibitor of basal and stimulated exocrine pancreatic secretion in healthy volunteers and unlike somatostatin, can be given subcutaneously [56]. Duration of action is 6-12 hr, Absorption is rapid and complete , bioavailability is 100% after subcutaneous injection ,extensively hepatic metabolism and excreted in urine.[57]

The earliest trial of somatostatin , a hormone which inhibits exocrine pancreatic secretion, as a rational therapeutic approach in human acute pancreatitis, yielded "an impressive clinical improvement".[58]

The findings of later ,randomized trials of somatostatin in acute pancreatitis were controversial.[59,60,61,62,63,64]

The idea that the pharmacological inhibition of exocrine pancreatic secretion might serves as a specific treatment for acute pancreatitis has been a subject of controversy for more than three decades. Activated pancreatic enzymes seem to be involved in the generation and progression of this "autodigestive" acute inflammatory disease, and it was therefore hypothesized that the administration of somatostatin or its analogue octreotide might help prevent progression of the disease and its complications.[9,10,11,12]

## Aim of the study

To investigate the curative effect of octreotide in the treatment of acute pancreatitis.

# Patients and method

A prospective study conducted at Baghdad teaching hospital/ Medical City from November 2015 to November 2017.

Inclusion criteria

1) Patients with moderate and severe acute pancreatitis .
2) The diagnosis based on:
   a. Symptoms of upper abdominal pain radiating to back.
   b. Serum amylase level greater than three times the upper limit of normal.
   c. Edema of the pancreas on ultra sound examination.
   d. Severity of pancreatitis confirmed by CT scan performed in all patients within the first 72 hours of admission.

Exclusion criteria

1) Patients with mild acute pancreatitis were excluded.

2) when the diagnosis of acute pancreatitis is not confirmed by the above mentioned criteria.

The patients were randomly assigned into two groups:

1) Control group: conservative treatment only:

a. Aggressive fluid resuscitation.

b. Nasogastric drainage.

c. Broad spectrum iv antibiotics( third generation cephalosporin and metronidazole).

d. Intravenous proton pump inhibitor.

2) Treatment group : the same conservative treatment to control group plus octreotide (Sandostatin® ,Novartis Pharma) 0.1 mg subcutaneous injection every 6 hours for maximum 7 consecutive days.

The randomization of the patients depends on the availability of octreotide in the hospital and on the physician who admit the patient (some physician don't believe in the use of octreotide in acute pancreatitis).

Octretide was stored at 2-8 C.

The patients were assessed for :

1) Hospital stay duration.

2) Development of complications (ARDS,septicemia,renal failure,abcess formation,pancreatic psuedocyst).

3) Mortality.

The diagnosis of ARDS was made based on tachypnea , hypoxia (arterial $PO_2$ less than 60 mmHg in room air) and diffuse interstitial pattern on chest x-ray.

Sepsis was defined as fever >38.5 , leukocytosis>15000 and positive blood culture.

Renal failure was defined by increase in serum creatinine >2mg/dl.

Pseudocyst and abscess were diagnosed by CT scan examination.

## Statistical Analyses

Statistical analyses were performed using SPSS statistical package for Social Sciences (version 17.0 for windows, SPSS, Chicago, IL, USA) and Graph Pad Prism 7.00 software Inc. Data are presented as mean ± SD, and range for quantitative variables and as number and percentage for qualitative variables.

Quantitative variable differences between two groups were evaluated with independent t-test; qualitative relations were evaluated using Chi-square test. Binary logistic regression was used to calculate the odd ratio, while the relative risk and its 95% confidence interval calculated using Koopman asymptotic score

P value of <0.05 was considered statistically significant.

# Results

Between November 2015 and November 2017, 78 patients entered the study , 40 patients were treated by traditional conservative treatment ( control group) and in 38 patients octreotide ( 0.1 mg every 6 hr s.c for max 7 consecutive days ) added to conservative treatment (treatment group).

The two groups matched with regard to age ,gender, etiology , severity of acute pancreatitis and the duration between onset of symptoms and hospital admission.There was no significant difference between the two groups.( Table 1)

Table 1: Clinical characteristics of the patients in both groups.

| | | **Control group(n=40)** | **Treatment group(n=38)** | **P- value** |
|---|---|---|---|---|
| **Mean age** (yr) | | 40.32±10.42 | 44.42±11.22 | 0.099 |
| **Gender** ( F/M) | | 24/16 | 27/11 | 0.305 |
| **Etiology** no(%) | Gallstone<br>Alcohol<br>PostERCP<br>Drugs<br>unknown | 28 (70%)<br>5 (12.5%)<br>5 (12.5%)<br>1 (2.5%)<br>1 (2.5%) | 29 (76.31%)<br>5 (13.15%)<br>3 (7.89%)<br>0 (0%)<br>1 (2.63%) | 0.832 |
| **Severity** no(%) | Moderate<br>Severe | 20 (50%)<br>20 (50%) | 23 (53.5%)<br>15 (42.9 | 0.372 |
| **Mean Duration between onset of symptoms and admission** (days) | | 2.48± 1.26 | 2.11± 1.31 | 0.208 |

The complications was lower in treatment group as compared to control group, only 3 patients developed ARDS in treatment group while 7 patients in control group. Renal failure occurred in only 2 patients in treatment group while 4 patients in control group.

One patient was complicated by abscess in treatment group while 3 in control group and 3 patients developed pseudocyst in treatment group in contrast to 6 patients in control group.

Although the number of patients was lower in treatment group ,but statistically was not significant (P- value > 0.05). (Table 2)

Table 2: showing the relation of complications to treatment.

| Complications | Control group(n=40) No. (%) | Treatment group(n=38) No. (%) | P-value |
|---|---|---|---|
| ARDS | 7 (17.5%) | 3 (7.89%) | 0.312 |
| Septicemia | 13 (32.5%) | 7 (18.42%) | 0.198 |
| Renal failure | 4 (10%) | 2 (5.26%) | 0.676 |
| Abscess | 3 (7.5%) | 1 (2.63%) | 0.616 |
| Upper GIT bleeding | 0 (0%) | 1 (2.63%) | 0.487 |
| Pseudocyst | 6 (15%) | 3 (7.89%) | 0.482 |

When comparing the risk of developing complications between the two groups , patients in the control group have increased risk of developing complications ( ARDS, septicemia, renal failure, abscess and pseudocyst) in comparison to treatment group, however, all these complications was not statistically significance (i.e. modest effect) as illustrated in( Table 3 ).

Table 3: risk assessment of complication according to type of therapy.

| Complications | RR | 95%CI of RR |
|---|---|---|
| ARDS | 2.217 | 0.6771 to 7.491 |
| Septicemia | 1.764 | 0.8165 to 3.934 |
| Renal failure | 1.9 | 0.4312 to 8.557 |
| Abscess | 2.85 | 0.428 to 19.49 |
| Pseudo cyst | 1.333 | 0.3545 to 5.063 |

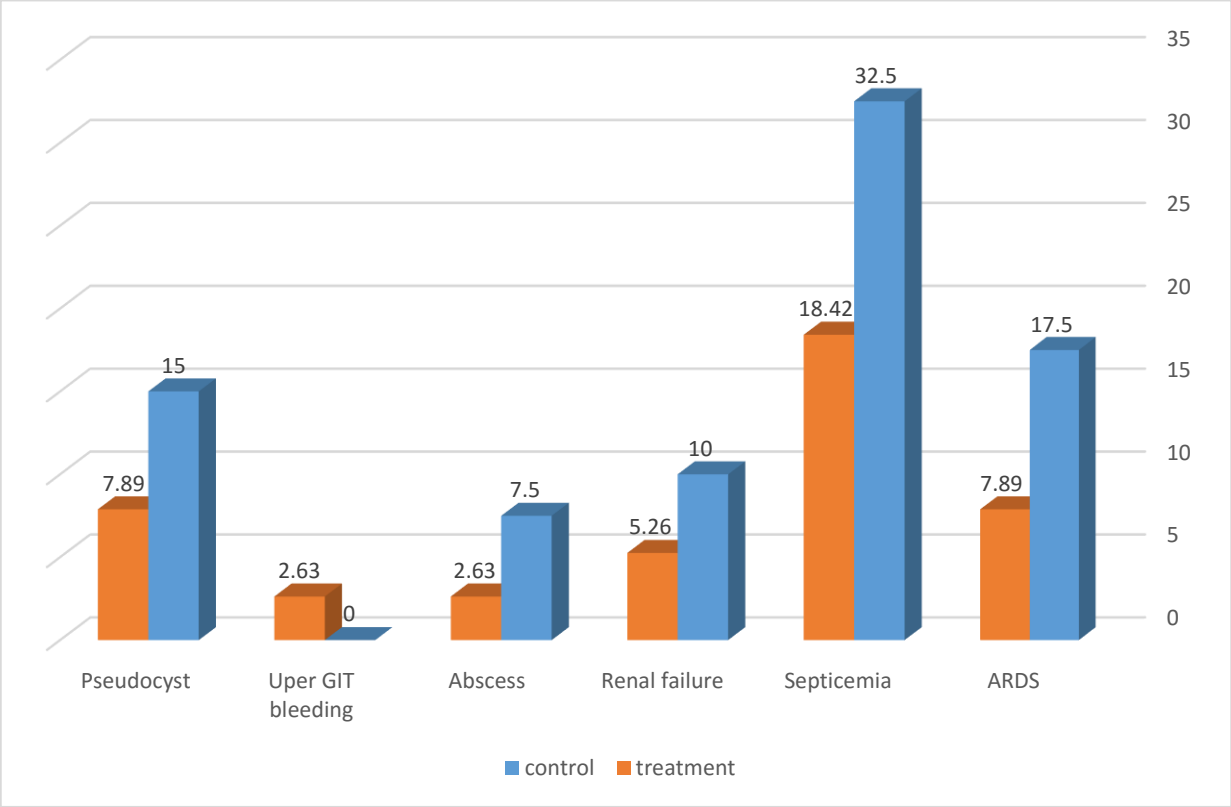


Figure 1: Illustrating the development of complications in the studied groups.

Mortality in treatment group was (10.5%) while in control group was (17.5%). Patients who were cured and discharged (65%) in the control group while, (81.57%) patients cured and discharged in the treatment group.One patient in the treatment group referred to another hospital.The number of patients who discharged with complication was (17.5%) in the control group but, only (5.26%) in the treatment group discharged with complication.

Mean hospital stay was 11.05 days in the control group and 12.95 days in the treatment group.

Statistically there was no significant difference in the outcome between the two groups P- value > 0.05 ( Table 4)

Table 4 : The relation of outcome to treatment.

| outcome | Control group (n=40) | Treatment group (n=38) | p- value |
|---|---|---|---|
| Cured and discharged no.(%) | 26 (65%) | 31 (81.57%) | 0.259 |
| Discharged with complication no. (%) | 7 (17.5%) | 2 (5.26%) | 0.496 |
| Reffered no.(%) | 0 (%) | 1(2.63%) | - |
| Death no.(%) | 7 (17.5%) | 4 (10.5%) | 0.173 |
| Mean hospital stay (day) | 11.05±5.31 | 12.95±5.74 | 0.133 |

Patients received treatment had 2 folds increased probability of achieving cure and discharge compared to those on control, also patients on treatment had 50% reduction in the probability of having complications compared to those on control.

Table 5: Risk assessment of outcome.

| | OR | 95%CI of OR |
|---|---|---|
| Cured & discharged | 2.154 | 0.568 – 8.168 |
| | | |
| Discharged & complications | 0.500 | 0.068 – 3.675 |
| Death | Refrence | - |

$R^2$ = 0.054, p (model) = 0.821

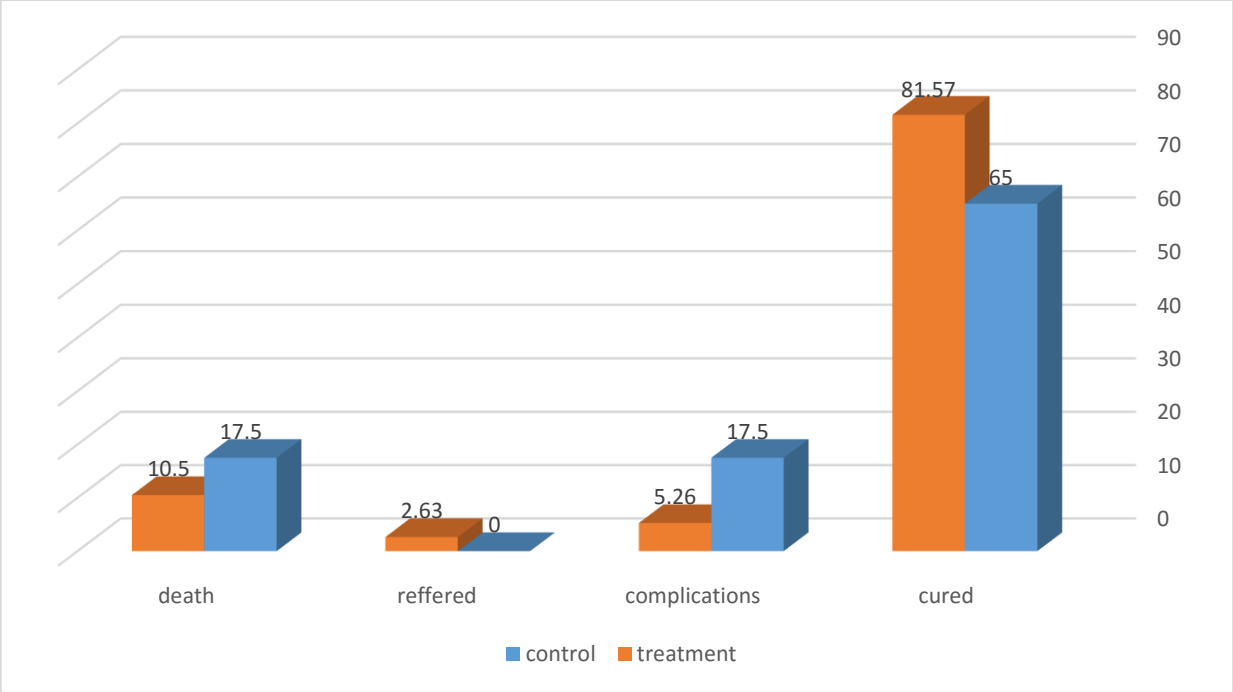


Figure 2: showing the relation of outcome to treatment.

# Discussion

The morbidity and mortality of severe acute pancreatitis are still distressingly high.[9,10,11.12]

Its natural course has two phases. The first phase ( during the first two weeks after onset) is characterized by a systemic inflammatory response (SIRS) due to the liberation of vasoactive and toxic mediators by the necrotizing process.[64,65]

In the second phase (during the third week and beyond) septic complications occur as a result of infection of pancreatic necrosis.[66] although the initiating pathophysiological process are not known ,the disease is thought to result from destruction of the pancreas and peripancreatic fatty tissues by digestive enzymes which are produced in the acinar cells and activated intracellularly or extracellularly.[16,17]

In the absence of any available test to determine severity, close examination to assess early fluid losses, hypovolemic shock, and symptoms suggestive of organ dysfunction is crucial Rather than depending on a scoring system to predict severity of AP, clinicians need to be aware of intrinsic patient-related risk factors, including laboratory and imaging risk factors, for the development of severe disease. These include: a patient ' s age, comorbid health problems, body mass index , the presence of SIRS , signs of hypovolemia such as an elevated BUN and an elevated hematocrit , presence of pleural effusions and/ or infiltrates altered mental status , and other factors.[31] Two treatment options might be of value to limit the "autodigestion" local damage and to interfere with sirs in the early phase acute pancreatitis inhibition of the activated enzymes with protease inhibitors ;or inhibition of exocrine secretion to" put the gland to rest " and prevent the further release and activation of digestive enzymes.protease inhibitors( such as aprotinin and gabexate mesilate), hormones(such as calcitonin and glucagon) and atropine have all been clinically tested aspossible specific treatment for acute pancreatitis, sofar without success[13,14,15].

Somatostatin and its analogue octreotide have been tested in several experimental models of acute pancreatitis , with contradictory results, sometimes negative[68,69,70,71,72]and sometimes positive[73,74,75,76,77,78]. The first clinical study of somatostatin treatment ,published in 1980, revealed"an impressive improvement

" in all 14 patients treated with a continuous intravenous infusion of somatostatin. [57]

This encouraging result provided the motivation for further trials, none of which showed a significant benefit on the prognosis of patients with acute pancreatitis [58,59,60,61,62,63].

In 1991 , Caraballo and colleagues[79] published a meta- analysis of six randomized clinical trials involving somatostatin. The individual trials had not revealed any statistically significant benefit ,in the meta-analysis , however, the 207 patients in the somatostatin group had a significant lower mortality than the 207 patients in the placebo (6.2% versus 14% , $p<0.01$).

In our study gallstones was the most common cause of acute pancreatitis 73% ( 70% in control group & 76.3% in treatment group).the same result with Paran et al [80],Osman et al [81], Karakoyular et al [82]and Talaiezadeh[83]. All studies shows gallstones commonest cause of acute pancreatitis ,while in W.Uhl[84] et al study the commonest etiology was alcohol 42% then gallstones 37%.

In our study ARDS ,septicemia ,pseudocyst ,renal failure and abscess were lower in treatment group than in control group,but statistically was not significant due to small sample of the study ,the same result with Karakoyuler[82] and Talaiezadeh [83] while Paran[80] in his study shows a significant decrease in ARDS and septicemia in patients received octreotide with no significant decrease in other complications (abscess , pseudocyst and renal failure).

Hospital stay duration in our study didn't have a significant difference between the two groups ( control 11.05 vs treatment 12.95), while Paran[80] had a significant decrease in hospital stay when adding octreotide to the treatment of acute pancreatitis (33% control vs 20.6% treatment).

The overall mortality in our study was 14.1% (17.5% in control group vs 10.5% in treatment group ) but ,statistically was not significant . W.Uhl [84]had 14% mortality (16% control vs 15% low dose octreotide and 12% high dose octreotide ) while the mortality in Osman et al[81] study was 12.5% (10% control vs 15% treatment ). Paran et al [80] had a significant decrease in mortality (8% treatment vs 32% control )and overall mortality 20%.

Talaiezadeh AH et al [83] (astudy of 25 patients 6 in control group vs. 19 received octreotide 0.05 mg q 12 hr.),Karakoyunlar et al [82] ( a study of 43 patients 21 in control group vs. 22 received octreotide 0.5 Mcg/Kg/ hr iv infution ) and Paran H[80] ( astudy of 50 patients 25 in control group vs. 25 received octreotide 0.1 mg q 8hr s.c ) concluded that octreotide may have a beneficial role in management of pateints with acute pancreatitis and could be partially effective in lowering morbidity and mortality.

Osman et al [81] (a study of 40 patients 20 in control group vs. 20 received octreotide 0.2 mg q 8hr s.c ) concluded that no benefit was added to the clinical improvement after treatment of acute pancreatitis with octreotide and he don't recommend octreotide as aroutine treatment for patients with acute pancreatitis.

W.Uhl et al [84] (a study of 302 patients 103 in control group vs. 98 received low dose octreotide 0.1 Mcg q 8hr s.c and 101 received high dose octreotide 0.2 Mcg q 8hr s.c) concluded that octreotide treatment for acute pancreatitis can not be recommended on the basis of his study.

In our study the results were not significant statistically due to small sample of the study because of limited time of study and unavailability of octreotide and most physicians don't believe in the use of it in acute pancreatitis inspite of that it seems that octreotide has some effect in lowering morbidity and mortality.

## Conclusion

Although it was statistically not significant due to small sample size of the study ,it seems that octreotide may be effective in lowering morbidity and mortality in severe acute pancreatitis.

## Recommendation

A larger sample size study or a meta-analysis study is recommended to prove the curative effect of octreotide in acute pancreatitis.

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# الخلاصه

**المقدمه:** التهاب البنكرياس الحاد من الاسباب الشائعه لدخول المستشفى ومصدر رئيسي لاعتلال المرضى في العالم. الى يومنا هذا، لا يوجد علاج معين لهذا المرض. استغلال فكرة تثبيط الافرازات الخارجيه للبنكرياس كعلاج لالتهاب البنكرياس الحاد كانت موضع خلاف لاكثر من ثلاثة عقود. قد تتسبب انزيمات البنكرياس في احداث وتقدم عملية الهضم الذاتي والالتهاب الحاد للبنكرياس، لذلك من المفترض ان لاستخدام السوماتوستاتين او نظيره الاوكتريوتايد قد يساعد في منع تقدم المرض وظهور مضاعفاته.

**الهدف من الدراسة :** تحري التاثير العلاجي للاوكتريوتايد في حالات التهاب البنكرياس الحاد.

**الطريقة:** اجريت دراسه استباقيه في مستشفى بغداد التعليمي / مدينة الطب للفتره من تشرين الثاني ٢٠١٥ الى تشرين الثاني ٢٠١٧، تم تقسيم ثمانيه وسبعون مريض بصوره عشوائيه الى مجموعتين: المجموعه الاولى والمكونه من اربعين مريض تلقوا كافة العلاجات الداعمه والمجموعه الثانيه المكونه من ثمانية وثلاثين مريض تلقوا بالاضافة الى العلاجات الداعمه ٠,١ ملغم من دواء الاكتريوتايد كل ٦ ساعات عن طريق الحقن تحت الجلد لمدة اقصاها ٧ ايام. تم تقييم المرضى من حيث مدة الرقود في المستشفى ،ظهور مضاعفات البنكرياس الحاد والوفيات.

**النتائج:** المجموعتين كانت متوافقه من حيث العمر، الجنس، الفترة ما بين ظهور الاعراض ودخول المستشفى، شدة المرض والعامل المسبب لالتهاب البنكرياس الحاد. كانت نسبة الوفيات وظهور المضاعفات اقل في المجموعة الثانية من المجموعة الاولى ( متلازمة الضائقة التنفسية للبالغين ٧,٨٩% مقابل ١٧,٥% ، تسمم الدم الجرثومي ١٨,٤٢% مقابل ٣٢,٥% ، فشل كلوي ٥,٢٦% مقابل ١٠% ، خراج البنكرياس ٢,٦٣% مقابل ٧,٥% ، الكيسة البنكرياسية الكاذبة ٧,٨٩% مقابل ١٥% والوفيات ١٠,٥% مقابل ١٧,٥% ).

على الرغم من تلك الفروقات ،احصائياً لم تكن ذات اهمية.

**الاستنتاج:** قد يكون دواء الاوكتريوتايد ذو فائدة في تقليل نسبه الوفيات وظهور الاعراض الجانبية لمرضى التهاب البنكرياس على الرغم من ان النتائج الاحصائية لم تكن ذات اهمية لقلة عدد المرضى المشمولين بالدراسة.