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Review

Acute pancreatitis following adult liver transplantation: A systematic review

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ABSTRACT

Although uncommon, acute pancreatitis is a well-recognized, but generally serious, complication following liver transplantation. In addition to being more prevalent in patients who underwent liver transplantation than in the general population, it has a more aggressive course and can be responsible for significant morbidity and mortality. The post-liver transplant population has altered anatomy, increased comorbidities, and requires a myriad of drugs. These characteristics make them different from the pre-transplant population. Despite their retrospective nature, prior studies have identified numerous etiological factors that are associated with an increased risk of acute pancreatitis following liver transplantation. These can be broadly classified into the following four categories: surgical and anatomical factors, infections, post-transplant management, and post-transplant complications.

The aim of this systematic review is to assimilate the available information regarding acute pancreatitis following adult liver transplantation to describe the risk factors and natural history of the disease and to highlight possible areas for further investigation.

Keywords: Acute pancreatitis, post liver transplantation, systematic review

INTRODUCTION

Acute pancreatitis (AP) can be a serious complication following liver transplantation. Among patients who underwent liver transplantation, its prevalence is generally higher than that in the general population, with reported incidence ranging from 3% to 8% (1-5). It can also have a more aggressive course, with mortality ranging from 37.5% to 63% (2,3,6,7) and is observed in both adult and pediatric populations (8). AP has been well documented in the setting of solid organ transplantation and has been described following kidney, bone marrow, intestinal, and heart transplantation (9-14).

The purpose of this systematic review is to assimilate the available information regarding AP following adult liver transplantation to describe the risk factors and natural history of the disease as well as highlight the possible areas for further investigation.

MATERIALS AND METHODS

PubMed, ScienceDirect, and OvidSP were searched using the keywords "acute pancreatitis" and "liver transplant," and the relevant original articles and case reports were reviewed. Studies investigating AP in the setting of adult liver transplantation were selected for detailed review (Table 1). Studies investigating pediatric liver transplant, multi visceral transplants, and asymptomatic hyperlipasemia and/or hyperamylasemia were excluded along with review articles, abstracts, editorials, and letters to the editor (Figure 1).

Etiology

The post-liver transplant population has altered anatomy and increased comorbidities as well as requires a myriad of drugs. As a result, they differ from the pre-transplant population. Previous studies have identified numerous etiological factors that are potentially associated with increased risk of AP following liver transplan-

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Table 1. Summary of studies investigating acute pancreatitis post adult liver transplant

Author (Reference) (Year)	Study design	Number of patients	Incidence of acute pancreatitis	Mortality	Cause of death	Identified etiology	Complications	Factors associated with acute pancreatitis post liver
Alexander et al. (1) (1988)	Retrospective case-control	51	8%	Not reported	-	-	-	Pancreatitis associated with acute hepatitis B
Yanaga et al. (6) (1992)	Retrospective	196	4%	63%	Primary graft failure (25%) Multible organ failure (25%) Systemic CMV (13%)	Aorto-Hepatic interposition graft placement (50%) Migration of T-tube (13%) CMV (13%) HBV (13%)	Pseudocyst (25%) Pancreatic abscess (13%) Retroperitoneal abscess (13%)	-
Camargo et al. (2) (1995)	Retrospective	354	2.8%	40%	Sepsis (75%) Intra-abdominal bleeding from acute hemorrhagic necrotic pancreatitis (25%)	ERCP (20%) Unknown (80%)	Percutaneous drainage (60) Multible intra-abdominal abscesses requiring laparotomy (20%)	Significantly more likely if hepatitis B surface antigen positive
Lupo et al. (7) (1997)	Retrospective	1181	1.5%	37.5%	-	Multiple organ failure (19%) Intra-operative pancreatic injury (19%) Use of galibladder conduit (19%) ERCP (13%) Unknown (25%)	-	Early (<1 month) associated with increased mortality. No significant effect of Hepatitis B or retransplant
Verran et al. (4) (2000)	Retrospective	298	3%	11%	Disseminated malignancy (100%)	Biliary tract manipulation (33%) Alcohol ingestion (25%) Hepatic artery thrombosis plus biliary tract problems (25%) Tumor in the region of the pancreas (22%)	Pseudocyst requiring drainage (22%) Pancreatectomy and spleneclomy (11%)	Incidence greater in men. Only studied 'late' pancreatitis (>2 months after liver transplant)
Krokos et al. (3) (2008)	Retrospective	1832	3%	63.6%	-	-	-	Significantly more likely if hepatitis B (30.9% vs. 6.2%). Increasing number of grafts in the same patient increases risk of pancreatitis >90 minutes of veno-venous bypass Roux-en-Y Loop. Quantity of intra-operative IV calcium chloride

CMV: cytomegalovirus; HBV: hepatitis B virus; ERCP: endoscopic retrograde cholangiopancreatography; IV: intravenous

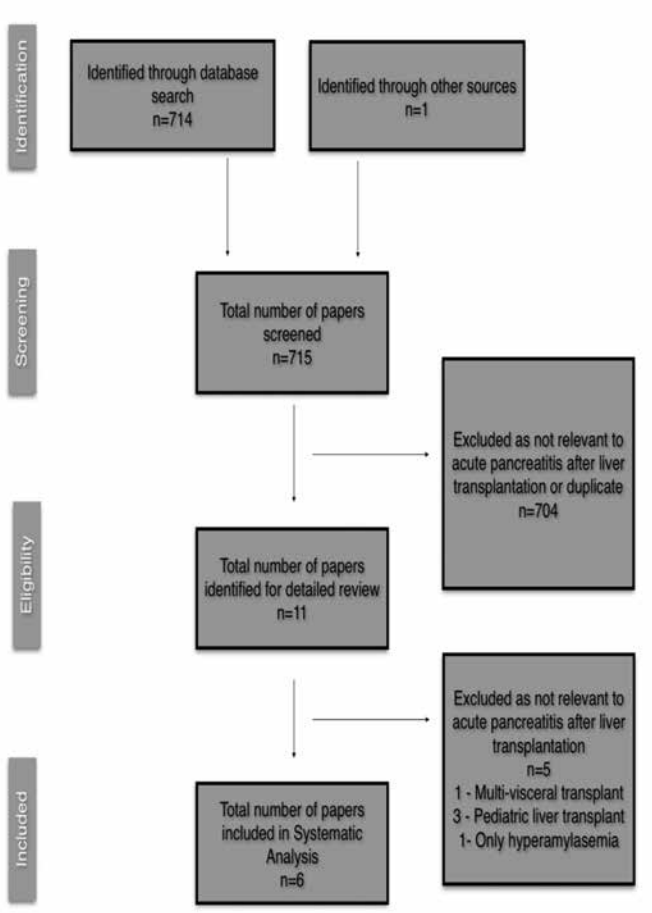

Figure 1. Prisma flow diagram illustrating Precess for identification of papers for inclusion.

tation. These can be broadly classified into the following four categories: surgical and anatomical factors, infections, post-transplant management, and post-transplant complications.

It is important to distinguish AP from asymptomatic hyperamylasemia or hyperlipasemia; the diagnosis of AP is made upon the fulfillment of two of the following three criteria: 1) characteristic abdominal pain; 2) serum amylase and/or lipase level that is >3 times the upper limit of the normal level; and 3) characteristic findings on abdominal computed tomography scan or other abdominal imaging studies (15,16). AP is associated with significant morbidity and mortality in both pre- and post-transplant populations (17).

Isolated hyperamylasemia and hyperlipasemia without abdominal pain are not associated with increased morbidity or mortality (2,3) and are commonly explained by a diverse range of etiologies, including drugs (18), renal insufficiency (18) diabetes (19), surgery (20), and tumors (20). One prospective study of 1,756 acute medical patients showed that 88% of asymptomatic patients with hyperamylasemia or hyperlipasemia had normal pancreas on imaging and 3% had evidence of chronic pancreatitis (20). Asymptomatic hyperlipasemia and amylasemia are not consistent with AP and have not been included in this systematic review.

Surgical factors

Post-operative pancreatitis is a well-known phenomenon and is most commonly seen in biliary and gastric procedures but has also been documented in cardiac and abdominal vascular surgeries. Theories for the etiology of post-operative pancreatitis include intra-operative manipulation of pancreatic and peri-pancreatic tissue and pancreatic ischemia during hypotension or cardiopulmonary bypass (21-25). During liver transplant, some degree of manipulation and injury to the peri-pancreatic tissue is necessary, and both extensive peri-pancreatic dissection and direct pancreatic injury have been shown to cause post-liver transplant AP. Factors that increase operative complexity or the amount of pancreatic dissection include repeat liver transplant, complex visceral transplants, and use of aorto-hepatic interposition grafts; all these further increase the risk of AP (3,7,13). The use of Roux-en-Y loop choledochojejunostomy have been linked to an increased risk of AP in some (3) but not other studies (2).

Cardiopulmonary bypass has been shown to cause post-operative AP in the non-transplant population, presumably because of ischemia (23,26) and increasing durations of veno-venous bypass have been inconsistently linked with the risk of developing AP post-liver transplant (2,3). Hypercalcemia has been linked to AP in the non-transplant population (23,27); Krokos et al. (3) demonstrated a close association between increasing amounts of intra-operative calcium and the development of post-liver transplant AP. A dose-related response was observed, with a rapid increase in the risk after the administration of 1.5 g of intravenous calcium chloride.

Infective causes

Infectious etiologies for AP are well documented but rare in the general population; viral causes such as mumps, cytomegalovirus (CMV), and herpes virus; bacterial infections such as mycoplasma, legionella, and leptospira as well as fungal and parasitic causes have been described (28). Infectious etiologies are also rare in the post-transplant population. There are a few published reports of AP due to infection following liver transplantation. Yanaga et al. (6) implicated systemic CMV as the cause of acute pancreatitis in one patient, although the diagnostic criteria was not provided, and Coelho et al. (29) has described one patient with liver transplant who died from AP caused by varicella-zoster virus and another patient who died from tuberculosis of the pancreas (30).

Several studies have linked hepatitis B virus (HBV) infection with the development of AP following liver transplantation (1-3). This association was first described by Alexander et al. (1) who noted that AP presented in 67% of patients who developed acute recurrent HBV infection. A large-scale retrospective review by Krokos et al. (3) demonstrated a significant association

between HBV-induced liver disease as a reason for transplantation and the development of AP. Yanaga et al. (6) also implicates HBV infection in the development of AP in one patient, but fails to give specifications on how the conclusion was reached. Unfortunately, neither of the abovementioned latter two studies indicates anything regarding the viral load, treatment status, or liver function.

Cavallari et al. (31) described a patient who developed a fatal necrotizing pancreatitis with concurrent acute HBV recurrence after liver transplantation. Importantly, they also demonstrated the presence of hepatitis B surface antigen and HBV DNA in the acinar cells of the pancreas accompanied by a necrotizing inflammation. The presence of HBV in the pancreatic tissue and HBV surface antigen in pancreatic and biliary secretions has been demonstrated in non-transplanted patients with HBV (32), and there are multiple reports of AP in acute exacerbations of HBV (33). Although there certainly is evidence linking HBV infection to AP in the immunocompromised post-transplant population, more research is required to further clarify the pathophysiology of this relationship.

Post-transplant complications

Biliary complications including biliary leaks and strictures can occur following liver transplantation (34). As a result, patients frequently require therapeutic endoscopic retrograde cholangiography (ERCP), which can result in post-ERCP pancreatitis (PEP). A recent large systematic review of the placebo or no stent arms of PEP prophylaxis trials reported PEP rates at 9.7% and 14.7% in average-risk and high-risk patients, respectively (35-37). A number of studies in the post-liver transplant population have demonstrated lower rates of PEP varying between 1% and 6.4% (38), with 2 studies showing that PEP is responsible for 11% of post-liver transplant pancreatitis (4,7). Other procedures that involve direct manipulation of the biliary tract have been shown to cause AP after liver transplantation, including T-tube manipulation or removal, percutaneous trans-hepatic cholangiography, and placement of biliary drains (4). As with the non-transplant population, biliary sludge and stone have been associated with the development of AP following liver transplantation, accounting for 22% of late-onset AP in one series (4).

Immunocompromised patients have long been known to be at an increased risk of malignancy, and liver transplant recipients have been shown to be at an increased risk of malignancies such as cholangiocarcinoma and lymphoma (39, 40). Malignancies that involve the biliary tract can present with AP in both the pre- and the post-transplant population, causing up to 22% of post-liver transplant pancreatitis (4,41).

Drugs

Many of the immunosuppressive drugs used in solid organ transplant have been identified as potential causes for drug-induced pancreatitis; however, whether they cause AP after liver transplantation is more controversial (3,7). Because patients are rarely rechallenged with drugs that may have been implicated in causing AP, the strength of evidence surrounding the association of most drugs and AP is weak. Steroids are commonly used to treat acute cellular rejection, and both dexamethasone and prednisone have the ability to cause drug-induced pancreatitis (42). Other drugs commonly used in the post-operative period that could cause pancreatitis in the post-liver transplant population include azathioprine/6-mercaptopurine and furosemide (42-45). Despite the range of drugs that could potentially cause drug-induced pancreatitis in the liver transplant population, only one paper identified a drug (adefovir) as a potential cause for drug-induced pancreatitis following liver transplantation (46). In fact, one study identified corticosteroids as a protective factor against PEP following liver transplantation (38).

Alcoholic liver disease is a leading indication for liver transplantation in the western counties (47), and alcohol is also the second most common cause of AP in the general population. Although a majority of cases of patients with alcoholic cirrhosis must demonstrate 6 months of sobriety prior to being listed for a liver transplantation, recidivism is not uncommon (48). Verran identified alcohol intake as the reason for 25% of cases of post-liver transplant pancreatitis, which is similar to the rate seen in the non-transplant population (4,49).

Management

There are no published guidelines specific for the management of post-transplant pancreatitis. As with AP in the non-transplant population, the mainstay of management in the reviewed studies consisted of conservative treatment with intravenous fluids and bowel rest with further surgical management as required (2-4,6,7,50). Although specifications of therapy are not discussed, this seems to be in line with the recommended treatment of AP in the general population (16).

Outcomes

AP following liver transplantation is a cause of morbidity and mortality. When outcomes are analyzed, there seems to be a marked difference between "early" (<1–2 months after liver transplantation) and "late" (>1–2 months after liver transplantation) AP. Early pancreatitis is typically associated with a poor prognosis with mortality rates as high as 63% (7), which are considerably higher than those in the general population, whereas late pancreatitis seems to have better outcomes with mortality from 0% to 11% (4,7), which is more comparable to that of AP in the general population (17).

Where documented in the reviewed papers, conservative management was successful in 58%–65% of cases, with 34%–42% requiring further surgical input, and 22–25% of patients developing pseudocysts (3,4,6,7). Better outcomes were seen in patients who were treated medically than those who required surgical intervention (mortality of 50% and 89%, respectively), although this probably represents more severe disease in the

patients who required surgery. Retransplantation was required in up to 25% of cases of AP (3,6).

CONCLUSION

Although uncommon, post-liver transplant AP carries significant morbidity and mortality, and studies to date have identified a number of risk factors. However, the majority of these studies are small and retrospective. There is a need for high quality prospective studies to further clarify the risk factors for the development of AP following liver transplantation.

Although there are no specific guidelines for the treatment of post-transplant pancreatitis, clinicians should be aware of the increased severity of this disease as well as of the increased risk of allograft loss and of retransplantation.

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REFERENCES

1. Alexander JA, Demetrius AJ, Gavaler JS, Makowka L, Starzl TE, Van Thiel DH. Pancreatitis following liver transplantation. Transplantation 1988; 45: 1062-5. [CrossRef]
2. Camargo CA Jr, Greig PD, Levy GA, Clavien PA. Acute pancreatitis following liver transplantation. J Am Coll Surg 1995; 181: 249-56.
3. Krokos NV, Karavias D, Tzakis A, et al. Acute pancreatitis after liver transplantation: incidence and contributing factors. Transpl Int 1995; 8: 1-7. [CrossRef]
4. Verran DJ, Gurkan A, Chui AK, et al. Pancreatitis in adult orthotopic liver allograft recipients: risk factors and outcome. Liver Transpl 2000; 6: 362-6. [CrossRef]
5. Sanroman Budino B, Vazquez Martul E, Pertega Diaz S, Veiga Barreiro A, Carro Rey E, Mosquera Reboredo J. Autopsy-determined causes of death in solid organ transplant recipients. Transplant Proc 2004; 36: 787-9. [CrossRef]
6. Yanaga K, Shimada M, Gordon RD, et al. Pancreatic complications following orthotopic liver transplantation. Clin Transplant 1992; 6: 126-30.
7. Lupo L, Pirenne J, Gunson B, et al. Acute-pancreatitis after orthotopic liver transplantation. Transplant Proc 1997; 29: 473. [CrossRef]
8. Tissieres P, Simon L, Debray D, et al. Acute pancreatitis after orthotopic liver transplantation in children: incidence, contributing factors, and outcome. J Pediatr Gastroenterol Nutr 1998; 26: 315-20. [CrossRef]
9. Fernandez-Cruz L, Targarona EM, Cugat E, Alcaraz A, Oppenheimer F. Acute pancreatitis after liver transplantation. Br J Surg 1989; 76: 1132-5. [CrossRef]
10. Ko CW, Gooley T, Schoch HG, et al. Acute pancreatitis in marrow transplant patients: prevalence at autopsy and risk factors. Bone Marrow Transplant 1997; 20: 1081-6. [CrossRef]
11. Slakey DP, Johnson CP, Cziperle DJ, et al. Management of severe pancreatitis in renal transplant recipients. Ann Surg 1997; 225: 217-22. [CrossRef]
12. Herline AJ, Pinson CW, Wright JK, et al. Acute pancreatitis after cardiac transplantation and other cardiac procedures: case-control analysis in 24,631 patients. Am Surg 1999; 65: 819-25; discussion 826.
13. Papachristou GI, Abu-Elmagd KM, Bond G, et al. Pancreaticobiliary complications after composite visceral transplantation: incidence, risk, and management strategies. Gastrointest Endosc 2011; 73: 1165-73. [CrossRef]
14. Tabakovic M, Salkic NN, Bosnjic J, Alibegovic E. Acute pancreatitis after kidney transplantation. Case Rep Transplant 2012; 2012: 768193. [CrossRef]
15. Kiriyama S, Gabata T, Takada T, et al. New diagnostic criteria of acute pancreatitis. J Hepatobiliary Pancreat Sci 2010; 17: 24-36. [CrossRef]
16. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology 2013; 13: e1-15. [CrossRef]
17. Fagenholz PJ, Castillo CF, Harris NS, Pelletier AJ, Camargo CA Jr. Increasing United States hospital admissions for acute pancreatitis, 1988-2003. Ann Epidemiol 2007; 17: 491-7. [CrossRef]
18. Rattner DW, Gu ZY, Vlahakes GJ, Warshaw AL. Hyperamylasemia after cardiac surgery. Incidence, significance, and management. Ann Surg 1989; 209: 279-83. [CrossRef]
19. Semakula C, Vandewalle CL, Van Schravendijk CF, et al. Abnormal circulating pancreatic enzyme activities in more than twenty-five percent of recent-onset insulin-dependent diabetic patients: association of hyperlipasemia with high-titer islet cell antibodies. Belgian Diabetes Registry. Pancreas 1996; 12: 321-33. [CrossRef]
20. Lankisch PG, Doobe C, Finger T, Lubbers H, Mahlke R, Brinkmann G, et al. Asymptomatic Hyperamylasemia and/or Hyperlipasemia: Their Incidence, Causes and Clinical Impact. Pancreas 2005; 31: 451. [CrossRef]
21. Ponka JL, Landrum SE, Chaikof L. Acute pancreatitis in the postoperative patient. Arch Surg 1961; 83: 475-90. [CrossRef]
22. White MT, Morgan A, Hopton D. Postoperative pancreatitis. A study of seventy cases. Am J Surg 1970; 120: 132-7. [CrossRef]
23. Fernandez-del Castillo C, Harringer W, Warshaw AL, et al. Risk factors for pancreatic cellular injury after cardiopulmonary bypass. N Engl J Med 1991; 325: 382-7. [CrossRef]
24. Z'graggen K, Aronsky D, Maurer CA, Klaiber C, Baer HU. Acute postoperative pancreatitis after laparoscopic cholecystectomy. Results of the Prospective Swiss Association of Laparoscopic and Thoracoscopic Surgery Study. Arch Surg 1997; 132: 1026-30; discussion 1031. [CrossRef]
25. Burkey SH, Valentine RJ, Jackson MR, Modrall JG, Clagett GP. Acute pancreatitis after abdominal vascular surgery. J Am Coll Surg 2000; 191: 373-80. [CrossRef]
26. Ohri SK, Desai JB, Gaer JA, et al. Intraabdominal complications after cardiopulmonary bypass. Ann Thorac Surg 1991; 52: 826-31. [CrossRef]
27. Carnaille B, Oudar C, Pattou F, Combemale F, Rocha J, Proye C. Pancreatitis and primary hyperparathyroidism: forty cases. Aust N Z J Surg 1998; 68: 117-9. [CrossRef]

28. Parenti DM, Steinberg W, Kang P. Infectious causes of acute pancreatitis. Pancreas 1996; 13: 356-71. **[CrossRef]**
29. Coelho JC, Wiederkehr JC, Campos AC, Zeni Neto C, Oliva V. [Acute pancreatitis caused by varicella-zoster virus after liver transplantation]. J Chir (Paris) 1994; 131: 96-8.
30. Coelho JC, Wiederkehr JC, Parolin MB, Balbi E, Nassif AE. Isolated tuberculosis of the pancreas after orthotopic liver transplantation. Liver Transpl Surg 1999; 5: 153-5. **[CrossRef]**
31. Cavallari A, Vivarelli M, D'Errico A, et al. Fatal necrotizing pancreatitis caused by hepatitis B virus infection in a liver transplant recipient. J Hepatol 1995; 22: 685-90. **[CrossRef]**
32. Yoffe B, Burns DK, Bhatt HS, Combes B. Extrahepatic hepatitis B virus DNA sequences in patients with acute hepatitis B infection. Hepatology 1990; 12: 187-92. **[CrossRef]**
33. Yuen MF, Chan TM, Hui CK, Chan AO, Ng IO, Lai CL. Acute pancreatitis complicating acute exacerbation of chronic hepatitis B infection carries a poor prognosis. J Viral Hepat 2001; 8: 459-64. **[CrossRef]**
34. Shah SR, Dooley J, Agarwal R, et al. Routine endoscopic retrograde cholangiography in the detection of early biliary complications after liver transplantation. Liver Transpl 2002; 8: 491-4. **[CrossRef]**
35. Cotton PB, Garrow DA, Gallagher J, Romagnuolo J. Risk factors for complications after ERCP: a multivariate analysis of 11,497 procedures over 12 years. Gastrointest Endosc 2009; 70: 80-8. **[CrossRef]**
36. Kochar B, Akshintala VS, Afghani E, et al. Incidence, severity, and mortality of post-ERCP pancreatitis: a systematic review by using randomized, controlled trials. Gastrointest Endosc 2015; 81: 143-9. **[CrossRef]**
37. Sanna C, Giordanino C, Giono I, et al. Safety and efficacy of endoscopic retrograde cholangiopancreatography in patients with post-liver transplant biliary complications: results of a cohort study with long-term follow-up. Gut Liver 2011; 5: 328-34. **[CrossRef]**
38. Law R, Leal C, Dayyeh BA, et al. Role of immunosuppression in post-endoscopic retrograde cholangiopancreatography pancreatitis after liver transplantation: a retrospective analysis. Liver Transpl 2013; 19: 1354-60. **[CrossRef]**
39. Penn I. Posttransplantation de novo tumors in liver allograft recipients. Liver Transpl Surg 1996; 2: 52-9. **[CrossRef]**
40. Koshiol J, Pawlish K, Goodman MT, McGlynn KA, Engels EA. Risk of hepatobiliary cancer after solid organ transplant in the United States. Clin Gastroenterol Hepatol 2014; 12: 1541-9. **[CrossRef]**
41. Corfield, AP, Cooper MJ, Williamson RC. Acute pancreatitis: a lethal disease of increasing incidence. Gut 1985; 26: 724-9. **[CrossRef]**
42. Badalov, N, Baradarian R, Iswara K, Li J, Steinberg W, Tenner S. Drug-induced acute pancreatitis: an evidence-based review. Clin Gastroenterol Hepatol 2007; 5: 648-61; quiz 644. **[CrossRef]**
43. Jones PE, Oelbaum MH. Frusemide-induced pancreatitis. Br Med J 1975; 1: 133-4. **[CrossRef]**
44. Lankisch PG, Droge M, Gottesleben F. Drug induced acute pancreatitis: incidence and severity. Gut 1995; 37: 565-7. **[CrossRef]**
45. Trivedi CD, Pitchumoni CS. Drug-induced pancreatitis: an update. J Clin Gastroenterol 2005; 39: 709-16. **[CrossRef]**
46. Weber A, Carbonnel F, Simon N, et al. Severe acute pancreatitis related to the use of adefovir in a liver transplant recipient. Gastroenterol Clin Biol 2008; 32: 247-9. **[CrossRef]**
47. Roberts MS, Angus DC, Bryce CL, Valenta Z, Weissfeld L. Survival after liver transplantation in the United States: a disease-specific analysis of the UNOS database. Liver Transpl 2004; 10: 886-97. **[CrossRef]**
48. Cuadrado A, Fabrega E, Casafont F, Pons-Romero F. Alcohol recidivism impairs long-term patient survival after orthotopic liver transplantation for alcoholic liver disease. Liver Transpl 2005; 11: 420-6. **[CrossRef]**
49. Toh, SK, Phillips S, Johnson CD. A prospective audit against national standards of the presentation and management of acute pancreatitis in the South of England. Gut 2000; 46: 239-43. **[CrossRef]**
50. Ince AT, Senturk H, Singh VK, et al. A randomized controlled trial of home monitoring versus hospitalization for mild non-alcoholic acute interstitial pancreatitis: a pilot study. Pancreatology 2014; 14: 174-8. **[CrossRef]**