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Diagnosis and Management of Biliary Strictures after Deceased-Donor Liver Transplantation Based on Clinical Practice

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Abstract

Biliary complications are still the main complications for liver transplantation recipients. Biliary strictures comprise the major part of all biliary complications after deceased-donor liver transplantation (LT). Biliary strictures following LT are divided into anastomotic strictures (AS) and non-anastomotic strictures (NAS). A Limitation of current published researches is that most studies aren't based on clinical practice. The aim of this review is to summarize risk factors, clinical presentation, diagnosis and management in post-LT biliary strictures.

Keywords

Biliary Strictures, Liver Transplantation, Clinical Practice, Anastomotic Strictures, Non-Anastomotic Strictures

1. Introduction

Liver transplantation (LT) is the only curative treatment of most types of liver failure and hepatocellular carcinoma (HCC). According to the scientific registry of transplant recipients (SRTR), the 3-year patient survival after liver transplantation is as high as 80% in the United States, and a 10-year survival rate is approximately 50% [1]. However, biliary complications are still the main threat to patients after LT, with an incidence varying between 5% and 25% [2]. Biliary strictures are one of the most common biliary complications following LT, which appear in 5% - 15% of patients following deceased donor liver transplantation and 28% - 32% of recipients after right-lobe liver donor liver transplantation [3]. Biliary strictures are defined as bile ducts including cystic duct, common bile duct, common hepatic bile duct and intrahepatic bile duct are narrow

in one or more locations of bile ducts.

Biliary strictures after LT are divided into two categories, including anastomotic strictures (AS) and non-anastomotic strictures (NAS). The most significant difference of AS and NAS is the incidence place of biliary strictures. Biliary strictures will be defined as AS when strictures happen in the anastomotic stoma, and be defined as NAS when strictures happen out of the anastomotic strictures. AS is distinct from NAS in pathogenesis, diagnosis, treatment and prognosis. A limitation of current published literatures doesn't focus on clinical practice. Although these published literatures such reviews about biliary strictures written by Kaveh *et al.* [4] and Margaret *et al.* [5] elaborate diagnosis and management of biliary strictures, a surgeon can't have a command of the literature rapidly and apply it to clinical practice because of its lengthiness and complexity. The aim of this review is to summarize risk factors, clinical presentation, diagnosis, management and prognosis AS and NAS after LT in a brief and logical way, which is used to guide clinical practice.

2. Anastomotic Strictures (AS)

2.1. Definition and Characteristics

AS are defined as isolated narrowing at the location of surgical anastomosis which is between the bile duct of donor and recipient, and are generally short, localized, single [6]. Incidence of AS is 4% - 9% in LT recipients [7]. AS can occur at any time after LT, but the majority appears ranging from 7 days to 11 years after LT [8].

2.2. Risk Factors

2.2.1. Surgical Risk Factors

Because surgical techniques are associated with local ischemia and fibrotic healing, the inappropriate operation skill is a significant risk factor for AS [9]. The detailed surgical risk factors are listed in **Table 1**.

Table 1. Risk factors for anastomotic strictures and non-anastomotic strictures.

Anastomotic strictures	Non-anastomotic strictures
Surgical risk factors	Hepatic artery thrombosis/stenosis
duct size mismatch	Primary sclerosing cholangitis
inappropriate suturing	Prolonged ischemic time
hepaticojejunostomy	Autoimmune hepatitis
non-surgical risk factors	Cytomegalovirus
donor age	
prior bile leak	
female donor/male recipient	

1) Duct size mismatch: according to a review, duct size mismatch caused by small caliber of bile ducts can lead to AS [9]. Although this theory does make sense logically, there is still lack of clinical studies to prove it.

2) Inappropriate suturing: too many sutures, uneven tension and distribution of sutures, and too few sutures may result in AS.

3) Hepaticojejunostomy: duct-to-duct anastomosis has been accepted as the standard biliary anastomotic method in many medical centers when recipients don't be diagnosed with primary sclerosing cholangitis. Hepaticojejunostomy without a T-tube is implicated as an independent risk factors for AS, so hepaticojejunostomy shouldn't be taken as a routine anastomotic approach for recipients [4].

2.2.2. Non-Surgical Risk Factors

1) Donor age: according to a retrospective study including a total of 1798 post-DDLT recipients, donor age ($p = 0.04$) is a significant risk factor [10].

2) Prior bile leak: prior bile leak (OR = 2.24, $p = 0.03$) is significantly associated with the formation of AS according to the result of a multivariate logistic regression in a retrospective study [8].

3) Female donor/male recipient: a sex mismatch with a female donor/male recipient is proved as a risk factor for AS in a retrospective study of containing 531 patients [7].

2.3. Clinical Manifestations

1) Symptoms: recipients with AS usually present with nonspecific symptoms, which is associated with biliary obstruction, for example, abdominal pain, jaundice, fever, indigestion, pruritus, anorexia [11].

2) Signs: epigastric tenderness, scleral icterus, subcostal hepatomegaly, etc. signs are also lack of specificity.

2.4. Laboratory Findings

1) Elevated serum total bilirubin, the ratio of conjugated bilirubin to total bilirubin is generally greater than 0.5.

2) Elevations of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase, gamma-glutamyl transferase.

3) Increase of leucocytes and neutrophils in recipients with infection.

2.5. Imaging Studies

1) Abdominal ultrasound (US) is usually the first radiological test. The US Characteristics of biliary strictures include biliary dilation, irregularity, discontinuity. Typical US appearance of AS is continuous, regular smooth dilation in the intrahepatic biliary duct, which is distinct from NAS [12]. Abdominal ultrasound has an approximately 100% of sensitivity in testing dilatation and obstruction of intrahepatic bile duct, however it's capacity to detect the masses and strictures is very low [13]. The abnormality of US usually needs further exami-

nations to diagnose AS.

2) Computed tomography (CT) sometimes is the first imaging test of discovering abnormality of bile duct. CT biliary characteristics of AS is similar to US. However, the sensitivity of CT scan in detecting AS is only 58.8% according to a respective study of 75 recipients [14].

3) Magnetic resonance cholangiopancreatography (MRCP) has been an optimal non-invasive imaging test for confirming whether recipients exist biliary obstruction or not. In a meta-analysis of including 4711 patients, MRCP is proved having a 95% of sensitivity and 97% of specificity [15]. In clinical practice, when suspecting recipients have AS, MRCP is the first non-invasive imaging examination.

4) Endoscopic retrograde cholangiopancreatography (ERCP) is still one of the gold standards for evaluating bile tract, but it isn't optimal imaging test considering it has invasive characteristics. Nowadays, diagnostic ERCP will be considered only when result of MRCP is normal but AS is still suspected [16]. In clinical practice, because complications of ERCP isn't rare and examination fee of ERCP isn't low-cost, patients who are with symptoms of AS or suspected AS by US can choose to follow up for a period of time when the result of MRCP is normal. It's reported that the incidence of pancreatitis after ERCP is 5.9%, the severe complications of ERCP including pancreatitis, infections, hemorrhage, perforations can even cause death. Choosing to undergo ERCP or follow up when non-invasive test can't diagnose AS is usually depended on patients [17].

2.6. Diagnosis

Up to now, there isn't diagnostic standard for AS following LT. when patients with obstructive symptoms or signs after LT, a AS will be suspected. A liver function test including total bilirubin, conjugated bilirubin, ALT, AST, alkaline phosphatase and gamma-glutamyl transferase should be performed, besides abdominal US and MRCP are arranged. If the ratio of conjugated bilirubin to total bilirubin is greater than 0.5 and both result of abdominal US and MRCP indicated a single biliary stricture at the site of anastomosis (the exact place of anastomosis need the surgeon of LT and radiologists to identify together), the diagnosis of AS is generally accurate. Sometimes, when even MRCP can't identify whether AS exists or not, ERCP can help to diagnose AS at last. The detailed diagnostic procedure is shown in **Figure 1**.

2.7. Management

2.7.1. Endoscopic Therapy

Endoscopic therapy has been regarded as the first-line regimen for AS currently because of its minimal invasion [18]. Traditional operation will be considered when endoscopy therapy failed.

1) ERCP-based Balloon dilation and stent placement (E-BDSP) are usually the optimal therapeutic method for recipients with AS, but no standard protocol

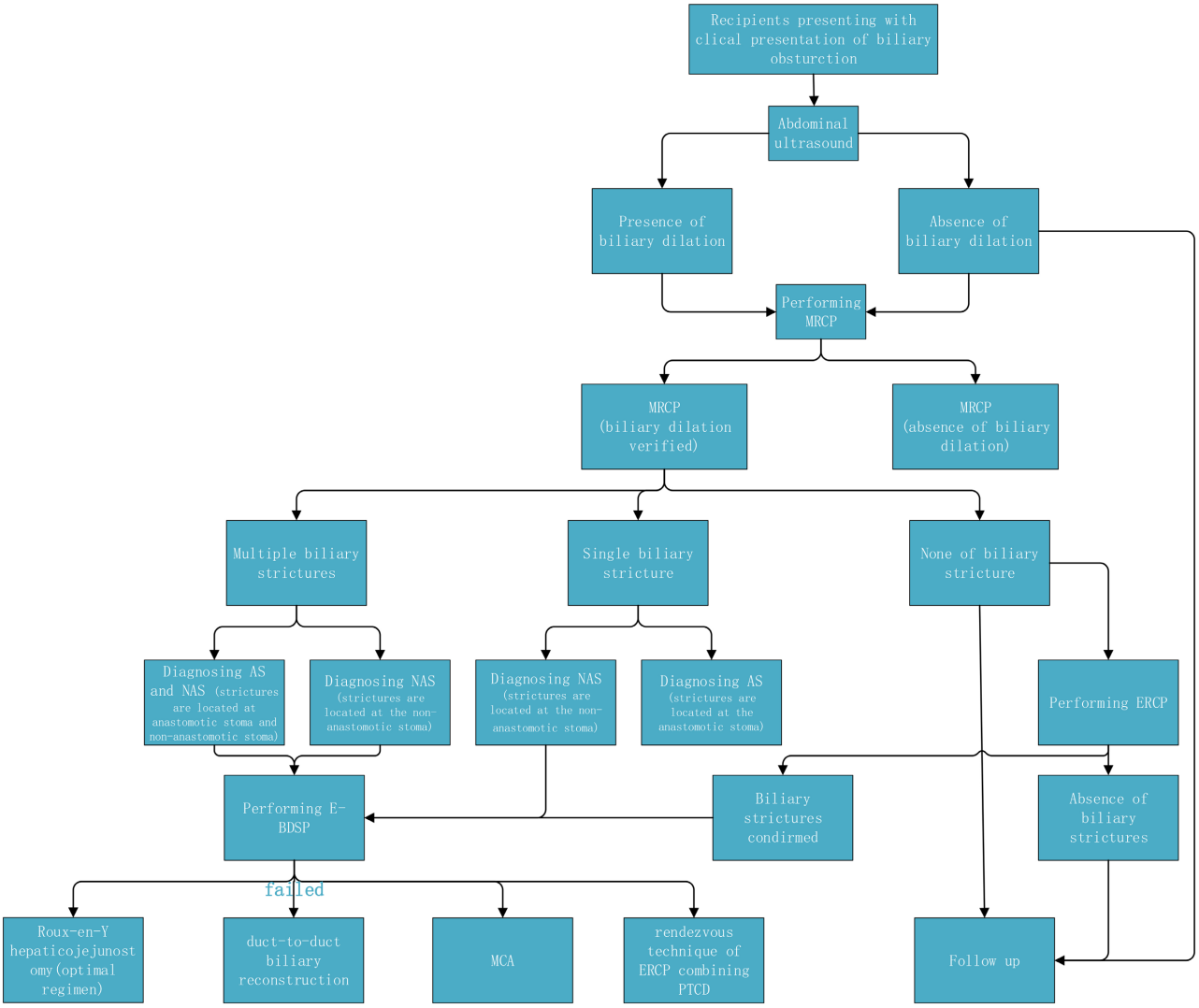

Figure 1. Diagnosis and management algorithm used in our medical center for biliary strictures patients after LT. MRCP = magnetic resonance cholangiopancreatography; AS = anastomotic strictures; NAS = Non-anastomotic strictures; ERCP = endoscopic retrograde cholangiopancreatography; E-BDSP = ERCP-based Balloon dilation and stent placement; MCA = magnetic compression anastomosis; PTCD = percutaneous transhepatic cholangial drainage.

has appeared for the endoscopic management of AS up to now. Multiple plastic stents (MPS) are often placed until AS resolution or for 12 months [19]. It's reported that in order to reduce the likelihood of stent blockage, plastic stents are exchanged every 2 - 3 months [20]. Self-expandable metal stents (SEMS) have been popular in recent years because of the advantages of longer stents patency and a larger diameter. SEMS should be placed in the biliary strictures for a minimum of 3 months and s higher stent migration rate have been noticed in the SEMS compared with MPS [21]. A randomized trial compares SEMS and MPS in patients with AS and found a similar resolution rate between them [22]. Considering the low cost of MPS, MPS are generally recommended to patients.

2) Balloon dilation (BD) was used alone originally, but the recurrence rate of AS is high. Later on, stent placement was added and achieved a satisfactory re-

sponse rate [23]. Up to now, balloon dilation alone isn't generally considered as a first-line treatment regimen when we meet AS clinically.

2.7.2. Surgical Treatment

In the setting of endoscopic therapy failure, traditional surgical treatment is indicated, including duct-to-duct biliary reconstruction and bilioenteric reconstruction.

1) Duct-to-duct biliary reconstruction: when endoscopic treatment failed in AS recipients, the location of isolated anastomotic stricture has been demonstrated in the cholangiogram. Besides, an intraoperative cholangiogram was also obtained to locate the AS before duct-to-duct anastomosis. after resecting stricture as little as possible, proximal bile duct close to liver and distal biliary tract close to duodenum are sutured. A retrospective study containing 20 patients supports the opinion that duct-to-duct reconstruction is feasible and safe [24]. Because the risk factors of AS still exist in the recipients who failed in the management of E-BDSP, duct-to-duct biliary reconstruction may cause AS again in those recipients. Duct-to-duct biliary reconstruction won't be chosen as a second-line therapy for those patients.

2) Bilioenteric reconstruction (Roux-en-Y hepaticojejunostomy): Roux-en-Y hepaticojejunostomy is considered the second-line treatment in patients with AS following failed endoscopic therapy [25].

2.7.3. Magnetic Compression Anastomosis (MCA)

MCA has become a promising treatment for AS patients failing in endoscopic therapy. MCA is a non-surgical treatment. In the technique, a magnet will be placed percutaneously at the proximal site and another magnet is placed to the distal site by endoscopy. Necrosis of stenotic place and fistula formation between the two magnets can achieve repetency of AS, then the stricture can be dilated by means of endoscopic therapy. The safety and feasibility of MCA have been proved clinically and experimentally, but a long-term follow-up study is still expected [26].

2.7.4. Rendezvous Technique of ERCP Combining PTCD

Both endoscopic retrograde cholangiopancreatography (ERCP) and percutaneous transhepatic cholangial drainage (PTCD) are the first-line therapy for AS recipients. In the technique, we inject a contrast agent through cannula of PTCD, and ERCP are also performed to locate the AS according to the bilateral cholangiography. Subsequently, we insert guidewires from both the side of PTCD and ERCP to break through the stricture [27]. Although study about the rendezvous technique is rare and efficacy and safety of the rendezvous are remain to be proved, the rendezvous technique is still a promising new technique when ERCP or PTCD failed in AS recipients.

2.8. Prognosis

Because the majority of AS recipients are amenable to endoscopic therapy,

prognosis of AS is usually satisfactory. According to a retrospective literature, the AS doesn't reduce recipients survival since survival is 98% and 81% in the AS patients at 1 and 5 years, respectively [18].

3. Non-Anastomotic Strictures (NAS)

3.1. Definition and Characteristics

NAS are defined as strictures which happen out of the surgical anastomosis site. NAS are located over 5 mm proximal to the surgical anastomosis [4]. Incidence of NAS varies in different studies from 0.5% to 10% [6]. The mean time of occurrence of NAS is 5 - 9 months following LT [28].

3.2. Risk Factors

1) Hepatic artery thrombosis/stenosis: hepatic artery thrombosis is a well-documented risk factor of NAS, and NAS are seen more frequently in recipient with hepatic artery stenosis [29]. Early hepatic artery thrombosis may lead to the severest NAS, because the blood supply of bile ducts is solely depended on the hepatic artery.

2) Primary sclerosing cholangitis (PSC): pre-existing PSC will raise the possibility of NAS in a retrospective study of containing 749 patients [28].

3) Prolonged ischemic time: prolonged warm ischemic time and warm ischemic time is strongly associated with the incidence of NAS. The reason why patients with prolonged ischemic time are predisposed to NAS may be that biliary endothelial and epithelial cells are vulnerable to ischemia/reperfusion injury. According to an animal study, bile duct cells are more susceptible to ischemia/reperfusion injury than hepatocytes [30].

4) Autoimmune hepatitis (AIH): a pre-transplant diagnosis of AIH in the recipients raises the possibility of NAS formation. The relative risk (RR) of AIH is 2.96 according to a retrospective study, which means recipients with AIH before LT is 2.96 times more susceptible to NAS than recipients without AIH [28].

5) Cytomegalovirus (CMV): postoperative CMV infection is identified as a risk factor for NAS [31].

3.3. Clinical Manifestations

Symptoms and signs of NAS are similar to AS, which are mainly biliary obstructive manifestations such as abdominal pain, jaundice, fever, or pruritus. Some patients are asymptomatic and are identified with elevation of liver enzymes (for example ALT, AST, alkaline phosphatase), rise of bilirubin.

3.4. Imaging Studies

1) Abdominal ultrasound (US) usually is the first imaging test for most patients suspected with biliary strictures because of its low cost, but its sensitivity is not enough high to diagnose NAS. The characteristics of NAS in US are skipped dilated peripheral ducts of intrahepatic bile duct with no visible connection to

dilated central ducts [12].

2) Computed tomography (CT) performs better than US in the sensitivity. It is repotted the sensitivity of CT is 83.3% in NAS patients [14].

3) Magnetic resonance cholangiopancreatography (MRCP) is the most accurate noninvasive imaging test for NAS. The sensitivity of MRCP for non-anastomotic strictures is 94% [32].

4) Endoscopic retrograde cholangiopancreatography (ERCP) is still regarded as gold standard for NAS, though many studies demonstrate MRCP is a promising noninvasive imaging study and may be equivalent to ERCP. In clinical practice, distal end of common bile duct or intrahepatic bile duct may be displayed unclearly in the MRCP and the diagnosis of NAS may be ambiguous. In this situation, ERCP needs to be performed to identify whether the NAS exist or not.

3.5. Diagnosis

Diagnostic method of NAS is similar to AS. Patients with biliary obstructive symptoms or signs have to finish MRCP. Besides, patients who are asymptomatic but are abnormality in liver function test or abdominal US also need to undergo MRCP. If there are multiple skipped biliary strictures in the MRCP imaging at the same time, diagnosis of NAS is usually accurate. If the MRCP still can't identify whether the strictures exist or not, ERCP will be performed to draw the last conclusion that whether there are NAS or not.

3.6. Management

3.6.1. Endoscopic Therapy

ERCP-based balloon dilation and stents placement (E-BDSP): Patients who undergo E-BDSP received a first ERCP including endoscopy and cholangiography to visualize the biliary tree, then multiple biliary stents are placed at the stricture. Compared with balloon dilatation alone, balloon dilation and stents placement are found to yield a better clinical result for NAS [33]. Therapy of NAS is generally more challenging than AS. The complete morphological resolution rate of NAS in patients who undergo E-BDSP is only 42.1% because of the refractoriness of NAS [23].

3.6.2. Percutaneous Transhepatic Cholangial Drainage (PTCD)

Though PTCD is technically easy to be performed, keeping a long-term drainage is still hard because of multiple strictures and drainage malfunctions. PTCD is generally an alternative when endoscopic therapy failed. PTCD is a reasonable choice for intrahepatic NAS, but it is only suitable to single intrahepatic NAS.

3.6.3. Surgical Treatment

1) bilioenteric reconstruction: when patients failed in the E-BDSP, surgical treatment usually the last chance to overcome NAS. Even though some patients can be suitable to the surgical approach of duct-to-duct anastomosis, the optimal

surgical regimen usually is bilioenteric anastomosis.

2) liver transplantation: the overall response rate of NAS is approximately 50% in the patients accepting treatment regimen of E-BDSP. Lots of patients with NAS still can't control the worsening of illness, although they have undergone E-BDSP or bilioenteric reconstruction. Retransplantation may be the only option for those patients with refractory NAS.

3.7. Prognosis

Approximately half of NAS patients are resistant to endoscopic therapy. NAS patients usually experience a few times endoscopic treatment, and even undergo bilioenteric reconstruction or retransplantation. The prognosis of NAS is generally disappointed. It's reported that graft survival in recipients with NAS is significantly worsen than recipietns without NAS. According to a clinical study, NAS patients of 23.5% died and of 29.4% experienced retransplantation [34].

4. Conclusion

Biliary strictures are common after LT in clinical practice especially anastomotic strictures. The pathogenesis of biliary strictures isn't explained clearly up to now. Diagnostic techniques such as ERCP and MRCP have a very high sensitivity and specificity, and therapeutic methods such as endoscopic therapy are very mature in the clinical practice. The overall resolution rate of NAS isn't satisfactory nowadays, even though we have variety of therapeutic techniques. We need to explore the pathogenesis of NAS and develop new technologies or target drugs to improve the response rate of NAS. Up to now, there is still lack of biological markers to help predict and diagnose biliary strictures. Seeking new biomarkers may also be a direction of efforts.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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