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## Adults with Liver Failure in the Intensive Care Unit: A Transplant Primer for Nurses

Cheryl McGinnis  
cheryl.mcginnis@mail.waldenu.edu

Stacia Hays  
*University of Florida*

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## Adults with Liver Failure in the Intensive Care Unit: A Transplant Primer for Nurses

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## **Introduction**

Liver transplantation has become the standard of care for the treatment of a variety of liver diseases that in the past would have resulted in an early death. According to the United Network for Organ Sharing (UNOS) there are currently 14,433 candidates waiting for a liver transplant in the United States. A total of 7,841 liver transplants were performed in the United States in 2016[1]. Despite increased education regarding organ donation and advances in patient management, there continues to be a gap in the supply and demand of organs for transplant. Due to this scarce resource, pre and post- transplant patient management remains a key focus in achieving optimal outcomes. Transplant medicine is a highly complex, specialized field, requiring specialized knowledge and management of the patient in the pre-transplant phase and the early post -transplant period. Patients requiring a liver transplant often are managed in the outpatient setting but can require intensive care placement for acute liver failure or decompensated end stage liver disease (ESLD). Critical care nurses play a role in the care of these patients. The best patient outcomes occur when expertise and experience coincide[2]. The purpose of this article is to provide critical care nurses with an overview of liver disease and transplantation for adult patients with a diagnosis of liver failure.

## **(S)Overview of Liver Function**

The liver is the largest internal organ in the human body. Weighing an average of 3 pounds in the average adult, it performs more than 500 critical functions (REF). At any time, it holds one quarter of the body's blood supply, and filters 1.4 liters of blood per minute and is responsible for 25% of the cardiac output. The portal vein supplies 75% of the liver's blood flow, and the remaining 25% of blood supply to the liver comes from the hepatic artery. Substances transported to the liver may be metabolized, stored, modified, or detoxified and then released into the circulation or intestine to be excreted. The liver plays a vital role in fat metabolism, gluconeogenesis, and the metabolism of carbohydrates. It is an important metabolizer of proteins, converting amino acids from foods to produce energy, fats, or carbohydrates. The by-product of this energy conversion is ammonia, which the liver converts to urea which is released into the blood, transported to the kidneys, and finally removed from the body in urine. The liver synthesizes several clotting factors, II, VII, IX, X, as well as factor V and factor XI (**Northrup, Caldwell**). It plays a primary role in gluconeogenesis and regulates blood glucose levels and produces approximately one liter of bile per day. Bile is vital for the absorption of fat-soluble vitamins and elimination of toxic substances.

The liver is the primary site for metabolism of medications through enzymes within the Cytochrome P450 (CYP 450) pathways. Drugs that affect these pathways are referred to as either inhibitors or inducers; those that cause increased concentrations of other drugs that utilize this pathway are inhibitors and can lead to subtherapeutic drug levels. Conversely, drugs that cause decreased concentrations are inducers and can cause toxic drug levels to occur. **J Adv Pract Oncol. 2013 Jul-Aug; 4(4): 263–268.**

**Published online 2013 Jul 1. Basic Review of the Cytochrome P450 System Anne M McDonnell, Cathyen H. Dang.** This pathway can upregulate or downregulate the

**Management of renal failure in end-stage liver disease: A critical appraisal. Liver Transplantation Volume 22, Issue 12 December 2016 Pages 1710–1719**

**Xingxing S. Cheng, Jane C. Tan, W. Ray Kim** Hypotension is a key risk factor. As seen in Fig. 2, a simplified representation of the work done by Stadlbauer et al., [21] the relationship between renal blood flow and mean arterial pressure is increasingly altered as the cirrhosis stage worsens, a phenomenon known as loss of autoregulation. In a patient with advanced cirrhosis, who is frequently hypotensive at baseline, his or her kidneys “live” on the steep part of the curve, therefore any slight alteration in volume status or systemic vascular resistance with even a minor hypotensive effect can lead to a drastic reduction in renal blood flow and, subsequently, GFR.

**Liver disease, especially cirrhosis, is characterized by reduced synthesis of the procoagulant proteins II, VII, IX, X, as well as factor V and factor XI** **Coagulation in Liver Disease: A Guide for the Clinician** **Patrick G. Northup, Stephen H. Caldwell** **Clinical Gastroenterology and Hepatology Volume 11, Issue 9, September 2013, Pages 1064-1074**

<b>CATEGORY</b>	<b>FUNCTION</b>
<b>Metabolic and Nutrition</b>	<ul style="list-style-type: none"> <li>-Gluconeogenesis</li> <li>-Fat, protein, carbohydrate metabolism</li> <li>-Produces bile</li> <li>-Synthesizes albumin</li> <li>-Activation of Vitamin D</li> <li>-Converts fats to ketones</li> <li>- Stores fat soluble vitamins, B 12, trace minerals</li> <li>-CYP 450 pathways</li> </ul>
<b>Coagulation</b>	<b>Produces clotting factors II, VII, IX, X, Factor V, XI</b>
<b>Immunologic</b>	<b>Detoxifies blood</b>

	<b>Converts ammonia to urea</b>
<b>Endocrine</b>	<b>Produces angiotensinogen, secretes somatomedin</b>
	<b>Hemolysis of red blood cells</b>

### **(S)Causes of Liver Failure**

Liver failure is often categorized as acute or chronic end stage liver disease, resulting in the need for a liver transplant to prolong the patient's life. Inflammation: early stage of liver disease, shows it is fighting off infections or heal injury.

Fibrosis: inflammation untreated liver scars. Scar tissue is unable to do the work that healthy tissue can do. At this stage, the scar tissue can be repaired and liver can heal and have improved to normal function.

Cirrhosis: left untreated, significant fibrosis leads to scarring that cannot be reversed. Cirrhosis can lead to a variety of chronic liver complications, such as liver cancer.

***DIAGNOSES SEE TABLE FROM OFFICE***

### **(S)Indications for Liver Transplant**

The need for liver transplant is considered when a patient with cirrhosis has signs and symptoms of hepatic decompensation in spite of the use of maximized medical therapies (Martin et al 2014 AASLD Eval for Liver tran in adults 2013). The most common indicators for transplant are variceal bleeding from the esophagus and/or stomach, bleeding due to portal hypertension,

significant coagulopathy, hepatic encephalopathy, spontaneous bacterial peritonitis, and severe and persistent ascites. SEE TABLE 1 below from Martin, et al, AASLD Guidelines 2013. Once hepatic decompensation develops, the patient with cirrhosis can quickly decompensate since additional complications such as sepsis, hepatorenal syndrome, and hepatic carcinoma are likely to hasten mortality.

### **Acute Hepatic Failure**

In the Intensive Care Unit (ICU) patients awaiting liver transplant typically have been admitted in acute or “fulminant” hepatic failure (AHF). This is defined as “hepatic encephalopathy within 8 weeks from the appearance of clinical evidence of hepatic failure without a history of previous liver disease”. (AASLD/UNOS). The clinical presentation typically includes hepatic dysfunction, significant coagulopathy, and may include hepatic encephalopathy (HE). If left untreated HE may progress to cerebral edema, ultimately leading to multi-organ system failure and death occurring in up to half the cases (**Acute Liver Failure William Bernal, M.D., and Julia Wendon, M.B., Ch.B. N Engl J Med 2013; 369:2525-2534 December 26, 2013 DOI: 10.1056/NEJMra1208937**). Although the etiology of acute hepatic failure can be due to a variety of factors in the United States the most common cause is due to drug-induced injury. Acetaminophen and certain anticonvulsants are well-known for causing AHF, as well as undiagnosed autoimmune disease and viruses.

#### **Signs and symptoms of acute hepatic failure**

- **Encephalopathy**
- **Cerebral edema and/or associated signs of ICP**
- **Jaundice**
- **Ascites**
- **Right upper quadrant tenderness**
- **Change in liver span**
- **Hematemesis or melena**

- **Hypotension and tachycardia**

The development of cerebral edema is a major cause of morbidity and mortality in patients with acute liver failure. Studies have demonstrated that an ammonia level  $f > 100 \text{ LM}$  on admission represent an independent risk factor for the development of high-grade hepatic encephalopathy, and a level of  $> 200 \text{ LM}$  predicts ICH (AASLD Lee, Stravitz, Larson March 2012 Hepatology)

TABLE 1 ACUTE LIVER FAILURE COMPLICATIONS OF CIRRHOSIS:

- Ascites
- Chronic gastrointestinal blood loss due to portal hypertensive gastropathy
- Encephalopathy
- Liver cancer
- Refractory variceal hemorrhage
- Synthetic dysfunction

### **Liver Transplant Evaluation**

Patients with acute fulminant liver failure (AFLF) or end stage liver disease (ESLD) experience the sequelae of liver disease, often requiring admission to the intensive care unit (ICU) for medical management. Chronic and acute liver failure patients are considered candidates for liver transplantation, now a treatment modality to decrease patient death. Intensive, supportive critical care patient management and liver transplantation provides an opportunity for patient survival for the majority of patients with acute fulminant liver failure[3]. The one year survival rate for patients with a primary (first) liver transplant is 91.8%.[1]. Currently there are approximately 170 transplant centers in the United States[1]. Patients referred for transplantation will be seen and evaluated at a transplant center by the transplant team. The ICU nurse can work with the

transplant team to provide psychological support and education to the patient and family during the evaluation process.

Transplant centers provide the expertise of a multidisciplinary transplant team approach to the evaluation process. The patient undergoes both physical and psychological testing to determine readiness for transplantation and the required long term care post- transplant care. The transplant evaluation is described as a 3-fold process with goals of establishing a diagnosis of ESLD; excluding any absolute or relative contraindications to transplant; and determining the suitability and degree of patient illness to better allocate resources and optimize survival[4]. The transplant evaluation process may vary from center to center with patient specific diagnostic testing being required. Common components of the transplant work-up include blood tests, diagnostic imaging and testing, psychological evaluation and consultation with other specialist depending on the patient's condition. Blood testing includes: liver function tests, PT/INR, a comprehensive metabolic panel, CBC with differential, blood type and screen, hepatitis screening (A, B, C), virology screening for cytomegalovirus (CMV), herpes simplex virus(HSV), Epstein-Barr virus(EBV) and HIV, tumor markers, including Alpha-fetoprotein(AFP), atrial blood gases(ABGs). Patients may require toxicology testing for alcohol and drugs pending the etiology of liver disease. Imaging studies include: chest radiography, duplex ultrasonography, abdominal CT scanning, CT scanning of the chest and pelvis or magnetic resonance angiography (MRI) if indicated. Imaging studies are required to detect malignancies in the pre-transplant patient. Pre transplant cirrhotic patients with hepatocellular carcinoma will requiring serial scanning to monitor tumor size and to determine treatment modalities pre transplant and tumor listing criteria. Additional tests to exclude malignancies may include: mammography and PAP

testing for women and PSA for men. The patient undergoing transplant evaluation will also require testing to evaluate cardiac and pulmonary function. Testing may include: stress thallium scanning, coronary angiography and echocardiography, electrocardiography and pulmonary function tests. Upper and lower GI endoscopies to evaluate for esophageal or gastric varices and to exclude malignancies of the GI tract may be required. Patients may require paracentesis for treatment of ascites and to rule out spontaneous bacterial peritonitis (SBP). Consultations and medical clearances are usually obtained from the following specialty services: cardiopulmonary, psychiatry and social work. If indicated, nephrology, infectious disease and dental work-ups may be needed before the patient can be listed. Financial clearance is always obtained prior to listing a patient for transplant.

Contraindications for liver transplant have become fewer over recent years. Transplant centers may vary in defining the relative contraindications specific to certain diseases or psychosocial conditions. Many centers mandate abstinence from alcohol, drug and nicotine use for 6 months prior to transplantation with center specific protocols for screening. A positive screen can render the patient inactive on the transplant list and even lead to delisting. Absolute or relative contraindications to transplant are listed in table 1.

**TABLE 1**

**Absolute and Relative Contraindications for Liver Transplantation**

<b>Absolute Contraindications</b>	<b>Relative Contraindications</b>
Brain Death or acute liver failure with intracranial pressure greater than 50 mm Hg or cerebral perfusion pressure less than 40 mm Hg	Advanced age(center specific)
Active alcohol abuse or drug use	Severe malnutrition
Extrahepatic malignancy	Morbid obesity
Uncontrolled sepsis	HIV(center specific)

Certain medical conditions: advanced cardiac or pulmonary disease; acquired immune deficiency syndrome	Multi organ failure
Anatomical abnormality or thrombosis of the superior mesenteric vein and portal vein which would preclude transplantation	Cholangiocarcinoma(center specific)
Persistent noncompliance (center specific)	Psychosocial conditions: dx of mental illness, non-compliance, lack of psychosocial support

5, 6

### **Organ Allocation and Listing for Transplant**

The Organ Procurement and Transplant Network (OPTN) and the United Network for Organ Sharing (UNOS) are responsible for managing the United States organ allocation[7]. Medical professionals, transplant recipients and donor families provide input in the national policies governing transplant, following the goals of the OPTN to increase numbers of transplants, provide equity to transplant access, improve waitlist, living donor and transplant recipient outcomes, promote safety for living donors and transplant recipients and promote efficient management of the OPTN [1].

Patient disease and illness severity plays a key role in organ allocation and patient listing for transplant. Scoring systems are specified according to age with The Model for End-Stage Liver Disease (MELD) used for patients 12 years and older. The MELD calculator provides a scoring system to determine how urgently a patient will need a liver transplant within the next three months [1]. The MELD calculates a patient's score based on the following laboratory tests: serum creatinine (mg/dl), bilirubin(mg/dl), INR, and serum sodium (mEq/L). The serum sodium, especially a low sodium level, is variable associated with mortality, independent of the MELD score and has recently been added to the MELD calculator[8]. Kidney function is also assessed through the question: has the patient had dialysis twice within the last week, or 24 hours

of CVVHD? If yes, the creatinine value is automatically registered at 4 mg/dl. Often patients in the ICU setting are requiring dialysis or CVVHD. The MELD calculation determines organ allocation based on the patient's score and geographic location. Scores range from 6 to 40 with a higher score indicating a sicker patient. An exception to this rule are patients that receive MELD exception points based on certain diagnoses such as hepatocellular carcinoma and portopulmonary hypertension to account for a higher waitlist mortality not reflected in the MELD score [9].

In 2013 the Share 35 policy was approved by UNOS mandating regional sharing of livers throughout 11 UNOS regions for patients with a MELD of 35 or greater. The Share 15 policy provides patients with a MELD of 15 or greater offers from the local organ procurement organization (OPO) first and then regionally Critically ill patients in the ICU setting with a life expectancy of seven days or less qualify for a status IA on the UNOS list.

Status IA organ offers can be from the local, regional or national UNOS list. If the patient's condition worsens and the patient becomes temporarily too unstable to undergo a liver transplant, the patient's status is changed to a status 7. Status 7 makes the patient inactive on the transplant list until stabilization is achieved and the status is updated. Status The patient's blood type and size are also required for UNOS listing with organ acceptance dependent on donor-patient size similarity and blood type compatibility.

Critical care nurses are involved with the ICU team in providing patient stabilization and supportive care to prevent further complications until an organ becomes available. Measures to manage the acutely ill pre-transplant patient includes seizure prophylaxis and surveillance; treatment of cerebral edema and inter cranial hypertension; management of encephalopathy and

coagulopathy; mechanical ventilation; treatment of circulatory dysfunction; infection surveillance and treatment; nutritional management; and management of fluids, electrolytes and glucose [11]. When a suitable organ is found for the patient, the liver transplant is performed and the patient returns to the ICU for post-transplant management. It is important for ICU nurses to be familiar with potential complications that can occur, often requiring immediate intervention. **Early Post-Transplant Complications**

Complications occurring post liver transplant can be categorized into early and late complications. For the purpose of this article, focus will be on the early post-transplant complications. Use of extended criteria donors (ECD) can increase the risk of less than optimal patient outcomes, including early complications. Donor variables known to affect patient outcomes include increased age, long hospitalization, extended warm or cold ischemic times and donor liver steatosis, leading to reperfusion syndrome, ischemia and graft dysfunction[12, 7].

#### Primary Graft non-function(PGNF)

Complete graft failure immediately post-transplant without a definitive cause has been defined as primary non-function. This type of liver injury can result in severe liver dysfunction with elevation in liver enzymes immediately post-transplant, secondary organ damage due to the liver dysfunction, biliary strictures and even re-transplant. Patients exhibit symptoms of coma, coagulopathy, oliguria, hypoglycemia and marked elevation in liver function tests with transaminases often greater than 5000 U/L[7]. The critical care nurse must be aware of these signs and symptoms of liver dysfunction and alert the transplant team.

#### Early Acute Cellular Rejection

Early acute cellular rejection usually occurs within the first 90 days post liver transplant while chronic rejection can occur later post-transplant, and is usually a result of low

immunosuppression levels. Acute cellular rejection is usually reversible but chronic rejection can affect long term graft and patient survival. Symptoms of acute rejection can include elevated LFTs (serum aminotransferases, alkaline phosphatase, gamma-glutamyl transpeptidase (GGT), and bilirubin levels), fever, abdominal pain and malaise. The gold standard to diagnose rejection is liver biopsy, percutaneous or transjugular. Treatment for rejection includes high dose corticosteroids, and an increase in immunosuppressant dosing or adding a second agent. Patients transplanted for HCV may be treated without the addition of steroids due to the impact on HCV progression. HCV and rejection have similar histological markers, often making diagnosis difficult to differentiate. Patients transplanted with a diagnosis of autoimmune hepatitis may require a more aggressive approach to the treatment of rejection, including high dose steroids. It is important to differentiate rejection from infection in the early post liver transplant patient to ensure appropriate treatment.

## **Vascular Complications**

### **Hepatic Artery Thrombosis**

Hepatic Artery Thrombosis (HAT) has been estimated to occur in 4-12% of adult liver transplants with causes including hepatic artery narrowing, kinking or a mismatch with the donor- recipient vessels[13]. Hepatic artery thrombosis is diagnosed through Doppler ultrasonography. Re-transplantation is indicated if blood flow cannot be restored to the transplanted graft.

### **Portal Vein Thrombosis**

Portal Vein Thrombosis(PVT) occurs in approximately 1-13% of liver transplant recipients and can be the result of technical problems during the time of surgery, thrombus formation, or a

hypercoagulable state [13]. A PVT can be diagnosed through ultrasonography showing an increase in velocity within the portal vein; CT scanning; an MRI or magnetic resonance venography (MRV). Treatment may include a thrombectomy, transjugular intrahepatic portosystemic shunt (TIPS) or re-transplant.

### **Biliary Complications**

Liver transplantation requires biliary reconstruction, connecting the common hepatic duct of the donor to the native bile duct of the recipient (choledocho-choledochostomy) or connection of the donor common bile duct to a portion of the recipient's jejunum (Roux-en-Y choledochojejunostomy). The type of biliary reconstruction is determined by surgeon preference, underlying liver disease, donor and recipient bile duct size or previous surgeries [14].

Use of a temporary biliary stent for an end-to-end biliary connection is also determined by transplant surgeon preference. Depending on stent placement technique, the stent may be passed through the GI track or require removal through an endoscopic retrograde cholangiopancreatography (ERCP) around three months post-transplant. The stent is radiopaque with placement or migration can be confirmed on abdominal x-ray.

Approximately 13-19% of adults receiving a deceased donor liver transplant can have biliary complications [13]. Biliary complications include biliary strictures and bile leaks.

### **Biliary Leaks and Strictures**

The ICU nurse will be more concerned about biliary leaks in the immediate post-operative period. Early biliary leaks are usually a result of technical issues, including lack of perfusion from the hepatic artery and occur at the biliary anastomosis. The patient may present with fever, abdominal pain and elevation in liver function tests. Imaging may include an US, cholangiogram or MRCP, showing peritonitis or fluid collections. An endoscopic retrograde cholangiography

(ERC) usually is performed for patients with a choledocho-choledochostomy and requiring biliary stent placement. Repeat LFTs are usually performed 1-2 weeks post-procedure.

Depending on the type of stent (plastic or metal), the stent will be removed or exchanged within three to six months. Biliary strictures can be at the site of the anastomosis, usually occurring within the first 6- 12 months post- transplant.**Bilomas**

A biloma can occur from biliary necrosis due to hepatic artery thrombosis. The biloma can be perihepatic or within the hepatic parenchyma. Treatment may include antibiotics, percutaneous drainage or surgical repair.

### **Early Acute Cellular Rejection**

Early acute cellular rejection usually occurs within the first 90 days post liver transplant while chronic rejection can occur later post- transplant, and is usually a result of low immunosuppression levels. Acute cellular rejection is usually reversible but chronic rejection can affect long term graft and patient survival. Symptoms of acute rejection can include elevated LFTs (serum aminotransferases, alkaline phosphatase, gamma-glutamyl transpeptidase (GGT), and bilirubin levels), fever, abdominal pain and malaise. The gold standard to diagnose rejection is liver biopsy, percutaneous or transjugular. The three histological features indicating acute cellular rejection are mixed inflammatory infiltrate in the portal triad; destructive or nondestructive nonsuppurative cholangitis involving the interlobular bile duct epithelium and endotheliitis[16]. Treatment for rejection includes high dose corticosteroids, and an increase in immunosuppressant dosing or adding a second agent. Patients transplanted for HCV may be treated without the addition of steroids due to the impact on HCV progression. HCV and rejection have similar histological markers, often making diagnosis difficult to differentiate.

Patients transplanted with a diagnosis of autoimmune hepatitis may require a more aggressive approach to the treatment of rejection, including high dose steroids. It is important to differentiate rejection from infection in the early post liver transplant patient to ensure appropriate treatment.

**Infection**

Post- transplant infections can be classified into three periods: transplant to one month; the second through sixth month and greater than six months. In the early post- transplant period, patients are at greater risk of infections, especially opportunistic, due to the high levels of immunosuppression. Bacterial, nosocomial infections are prevalent in the early hospitalized period due to central vascular access sites, indwelling catheters and drainage tubes, surgical complications, wound infections, and prolonged mechanical ventilation. C. Difficile infections are common during the hospitalization due to immunosuppression and antibiotic treatment. .

While donors are screened for all types of infections prior to transplant, if unexplained symptoms of infection occur, consider the possibility of a donor infection. Infection prophylaxis is started during the hospitalization with length of treatment per transplant center protocols. Refer to Table 2.

**TABLE 2**

**Prophylaxis During the Early Post Transplant Period**

<b>Infection</b>	<b>Prophylaxis</b>
Pneumocystis jirovecii (formerly P. carinii) pneumonia (PCP)	Trimethoprim-sulfamethoxazole 6-12 months. If allergic use Pentamidine, Dapsone, Atovaquone
Cytomegalovirus (CMV)	Ganciclovir IV or Valganciclovir oral
Candida	Antifungal such as Fluconazole, Mycelex, Nystatin

## Renal Dysfunction

Hypovolemia during and early post operatively along with high levels of immunosuppressant agents with known renal toxicity can cause worsening of kidney function. It is important for the ICU nurse to monitor the patient's renal function and urine output, observing for worsening function, indicating the need for dialysis. Immunosuppression will need to be modified in the picture of worsening kidney function due to the nephrotoxic effects of the calcineurin inhibitors (Cyclosporine and Tacrolimus).

## Additional Complications

Post-operative nursing management includes patient assessment for bleeding, wound infection or dehiscence and neurological changes. Coagulopathy and encephalopathy should improve post-transplant as liver function normalizes. Careful monitoring of the patient's mental status is required with any subtle change noted to the transplant team. High immunosuppression levels or electrolyte imbalances can cause seizure activity.

## Immunosuppression

Common immunosuppressant agents are listed in Table 3. Transplant centers have specific protocols for immunosuppression dosing, monitoring and therapeutic drug levels related to post transplant time periods.

**TABLE 3**

### Immunosuppressive Agents

Drug	Class
Cyclosporine	calcineurin inhibitor(CNI)
Tacrolimus	calcineurin inhibitor (CNI)
Sirolimus or Everolimus	Inhibitor of mechanistic target of Rapamycin (MTOR)
Mycophenolate mofetil	Inhibitor of mechanistic target of Rapamycin (MTOR)

Azathioprine	Inhibitor of mechanistic target of Rapamycin (MTOR)
Prednisone	corticosteroid

**Long Term Follow Up**

Long term management of the liver transplant patient becomes a joint venture between the primary care provider in the local community and the transplant center. Co-management can ensure the best patient outcomes. Transplant centers have specific protocols for the timing of transplant labs, including immunosuppression levels, protocol liver biopsies, and specific diagnostic testing based on the patient’s diagnosis at time of transplant. Primary care providers are involved with providing preventative care and managing complications of long term immunosuppression. Transplant patients must be monitored and treated for renal insufficiency, hypertension, hyperglycemia, malignancies and disease recurrence (HCV

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