Dental management of children undergoing liver transplantation

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Abstract

The number of pediatric liver transplantations undertaken in the US has increased dramatically in recent years. As the survival of liver recipients continues to improve, the dentist will need to be familiar with the management of these patients. This article describes the indications for pediatric liver transplantation, the types of liver transplants, the clinical features of liver disease, and the medical and dental management of children before and after liver transplantation.(Pediatr Dent 21:273-281, 1999)

Pediatric liver transplantation has become an accepted treatment for end-stage liver diseases, metabolic liver disorders, selected cases of acute liver failure and, less frequently, hepatic tumors.[1-3] Patient survival approaches 80%–90% at one year and 64%–78% at five years.[2-8] Increasing survival has resulted from improvements in immunosuppression, intensive care, management of complications, and surgical techniques.[8] The number of liver transplants undertaken in the US was 2690 in 1990, and 3912 in 1995.[9] As the life expectancy of liver recipients continues to rise with improving survival rates, the dentist will need to be familiar with the management of these patients. This article reviews the indications for pediatric liver transplantation, the types of liver transplants, the clinical features of liver disease, and the medical and dental management of children before and after liver transplantation.

Indications for pediatric liver transplantation (Table 1)

1. Biliary atresia accounts for 40%–70% of pediatric liver transplants in most centers. It is a condition of unknown etiology where there is progressive destruction and obliteration of the extrahepatic bile ducts leading to cholestasis, fibrosis, and cirrhosis.
2. Metabolic disorders associated with cirrhosis such as α-1 antitrypsin deficiency, cystic fibrosis, and Wilson's disease, and non-cirrhotic inborn errors in metabolism, based in the liver with life-threatening extrahepatic complications, such as Crigler-Najjar syndrome, Type I.
3. Fulminant hepatic failure due to, for example, viral hepatitis or toxic overdose of paracetamol.
4. Cholestatic disorders which may not be immediately life-threatening but where the quality of life is impaired by persistent jaundice, pruritus, fatigue, growth retardation, and osteopenia.
5. Malignant tumors of the liver without extrahepatic metastases.
6. Chronic liver disease leading to decompensated cirrhosis, for example, autoimmune hepatitis.

Types of liver transplants

The donor organ is matched to the recipient's ABO blood group. The types of liver transplants include whole graft, reduced-size graft, split-liver graft, living donor graft, and auxiliary transplantation (Table 2). There has always been a scarcity of pediatric donor grafts, especially for very young children. The situation has been alleviated by the use of reduced-size grafts, split-liver grafts, and living donor grafts, which has increased the donor pool and reduced the mortality on waiting lists. Auxiliary transplantation may be indicated in metabolic disorders, such as Crigler-Najjar syndrome, Type I, where the recipient's residual liver may have sufficient function to maintain life if the graft fails. Auxiliary transplantation may also be used in cases with acute hepatic failure where there is a chance that the patient's own liver will regenerate. The dental management of recipients is the same regardless of the type of liver transplant received.

Clinical features of liver disease

Medical aspects

Clinical features of liver disease vary with the underlying condition (Table 1). Jaundice, anemia, failure to thrive, pruritus, hepatosplenomegaly, ascites, encephalopathy, portal hypertension and esophageal varices, coagulopathy, altered drug metabolism, and propensity to infections may be present. Vitamin D malabsorption, rickets, and osteopenia are important

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Table 1. Pediatric Liver Disorders[8]

Liver Disorder	Definition	Etiology	Clinical Features	Medical & Surgical Management
Biliary atresia	Progressive disorder characterized by absence, destruction, or obliteration of the extrahepatic bile ducts	Unknown ? viral/teratogenic	Failure to thrive Hepatomegaly Jaundice Cirrhosis Portal hypertension 25% have other anomalies (i.e. atrio-septal defect, ventriculo-septal defect)	High calorie protein feed Fat soluble vitamin supplements: (A, D, E & K) Kasai procedure before 3 months Liver transplantation
Biliary hypoplasia Alagille syndrome	Absence or reduction in the number of intralobular bile ducts within portal tracts within the liver substance	Autosomal dominant	Persistent cholestasis Dysmorphic facies Cardiovascular anomaly – i.e. pulmonary stenosis Failure to thrive Vertebral and digital defects Mild mental retardation 30% +/- Development of hepatocellular carcinoma Pruritus	Nutritional support Liver transplantation if there is progression to cirrhosis and portal hypertension Fat soluble vitamin supplements: (A, D, E & K) Ursodeoxycholic acid –retards liver disease Cholestyramine, phenobarbital, & rifampicin for pruritus (itching)
Metabolic Disorders α-1 Antitrypsin deficiency	Deficiency of α-1 antitrypsin	Autosomal recessive	Cholestasis Ascites Failure to thrive Jaundice +/- diminishing with time Hepatosplenomegaly	40% develop chronic liver failure and require transplantation
Cystic fibrosis	Generalized disorder of exocrine glands characterized by abnormal transport of ions across epithelial surfaces	Autosomal recessive	Respiratory symptoms Failure to thrive Liver problems – 20% of adolescents Jaundice Portal hypertension	Bronchodilators & antibiotics for respiratory problems Nutritional support Vitamin A & E supplements Liver transplantation for end-stage liver disease Often bilirubin is normal but liver failure is more subtle and there is marked portal hypertension
Wilson's disease	Low serum copper; copper accumulates in the brain, liver, kidneys, and corneas	Autosomal recessive	Chronic hepatitis Cirrhosis Hemolytic anemia Neurological symptoms in the second decade i.e. mood and behavioral changes and extrapyramidal signs	Penicillamine and/ zinc, to chelate copper Vitamin B6 supplements Liver transplantation if liver failure
Acute liver failure	Massive necrosis of the liver cells or any other impairment of hepatic function	Paracetamol overdose Viral hepatitis: Non-A, non-B, Hepatitis A, Hepatitis B, Hepatitis C Halothane toxicity	Encephalopathy Coagulopathy Liver failure Renal failure	Intensive care management +/- Emergency liver transplantation

Table 2. Types of Liver Transplants

Type of liver transplant	Description
Whole graft	The diseased liver is replaced by a donor liver of the same size
Reduced size graft	The diseased liver of the recipient is replaced by the right or left lobe or the left lateral segment of the donor liver
Split-liver graft	The donor liver is split and the right lobe is used for one recipient and the left lateral lobe for another recipient
Living related graft	A segment from the liver of a living relative is used to replace the diseased liver of the recipient
Auxiliary transplantation	Part of the native liver is replaced by an appropriate part of the donor organ. The residual liver of the recipient is left *in situ*

complications of chronic cholestatis in children.[8] Bacterial peritonitis and recurrent cholangitis may further complicate the underlying liver disease.

Dental aspects

Intrinsic green discoloration of the teeth due to elevated serum levels of conjugated bilirubin in liver disease is well documented.[10-13] Green discoloration may also affect intra oral soft tissues including the gingivae, the tongue, the floor of the mouth, and the buccal mucosae. Other findings include enamel hypoplasia due to metabolic disturbances and delayed eruption of the teeth.[11-14] Enlarged pulp chambers and root canals secondary to vitamin D deficiency have also been recorded.[11,12,14] The prevalence of dental caries in children with end-stage liver disease would seem to be no greater than in the normal population. However, rampant caries has been reported in children with biliary atresia and was probably due to frequent and prolonged bottle feeding.[10-12,14,15] Use of numerous sugar containing oral medications is probably also a contributing factor.

Medical management

Pre-transplantation

Patients with obstructive jaundice are given oral supplements of fat soluble vitamins A, D, E, and K. The majority of children undergoing liver transplantation are malnourished, more prone to infection, and have a lower survival. A high calorie protein feed should be given to treat malnutrition and overcome fat malabsorption. Nasogastric enteral feeding may be necessary to ensure adequate nutrition. Children may be given low dose antibiotics to prevent cholangitis and spontaneous bacterial peritonitis. Pruritus is managed by cholestyramine, rifampicin, ursodeoxycholic acid, or phenobarbital, and ascites by fluid restriction and diuretic therapy. Bleeding from gastric erosions or portal hypertensive gastropathy is reduced by the administration of ranitidine and if severe, propranolol. Children with cystic fibrosis are given bronchodilators for the treatment of respiratory disease. Psychological and emotional preparation of the child and family prior to liver transplantation is undertaken.

Post-transplantation

Patients begin immunosuppressive therapy following liver transplantation to prevent acute rejection. Regimens vary from center to center but usually consist of cyclosporine (CSA; Neoral; Novartis®), corticosteroids, and azathioprine. An alternative to CSA is tacrolimus (Prograf; Fujisawa®), which has a similar mode of action. Antilymphocytic globulins, including monoclonal (i.e. OKT3), and polyclonal anti-T cell antibodies are used in some centers for induction of immunosuppression or in the treatment of severe or intractable rejection.

Acute rejection occurs usually within the first month after liver transplantation in 30%–70% of patients and is treated with high dose steroids.[16] Failure to respond to steroids is managed by conversion to tacrolimus or with anti-lymphocyte globulins. Graft loss due to acute rejection is relatively rare. Chronic rejection is usually apparent within 12 months of transplantation and is still a significant cause of graft loss in children (10%). Some patients respond to tacrolimus but regrafting may have to be considered.[17] The majority of pediatric liver recipients remain indefinitely on CSA or tacrolimus.

The most significant long-term complications of CSA and tacrolimus are nephrotoxicity, hypertension, infection (particularly viral), malignancy, including lymphomas and squamous cell carcinoma, convulsions, tremor, parasthesia, and hemolysis.[18] Other side effects of CSA include arthritis, obesity, hirsutism, thrombocytopenia, and gingival enlargement. Corticosteroids, such as prednisolone, have side effects including diabetes, osteoporosis, muscle wasting, suppression of growth in children, adrenal gland atrophy, Cushing's syndrome with moon face, striae and acne, and modification in tissue reactions which may result in spread of infection. These side effects are relatively uncommon in liver recipients as steroids are either withdrawn or maintained at low doses. Azathioprine (Imuran; Wellcome®) may cause myelosuppression, nodular regenerative hyperplasia, and increased risk of infection and malignancy.[19]

The majority of children will have at least one episode of infection during the recovery phase. Infection is the most frequent complication following liver transplantation, particularly in the first few months due to the higher levels of immunosuppression and although the levels are reduced over time, there is always a degree of increased risk. Infection may be bacterial, viral, fungal, or protozoal. Bacterial infections tend to occur within the first two weeks, particularly associated with chest and line sepsis.[20-22] All patients receive bacterial and antifungal prophylaxis in the immediate post-transplant period to prevent infection. The majority of fungal infections are caused by *Candida* species. Viral infections tend to occur after the second week. Cyclomegalovirus (CMV) is the most frequent cause of

significant viral infection in liver recipients and is treated with ganciclovir. Other viral infections include herpes simplex and zoster viruses, adenovirus, and Epstein-Barr virus (EBV). Infection with EBV may lead to infectious mononucleosis, or more importantly, a progressive lymphoproliferative disorder (LPD). Recent reports would suggest that EBV-related LPD is a particular problem in pediatric liver recipients and is associated with excessive immunosuppression.[23-25]

Vascular complications post-transplantation include hepatic artery thrombosis which usually requires re-transplantation, portal vein thrombosis and stenosis, and venous outflow obstruction from the graft. Biliary complications are the most common, occurring in 10%–15% of patients and include leaks (early) and strictures (late) which may require re-operation, but seldom lead to graft loss.

Dental Management

Pre-Transplantation

A team approach is required for the dental management of children prior to liver transplantation. The pediatric dentist should form part of this team, which should include the hepatologist, transplant surgeon, general medical practitioner, nursing staff, including clinical nurse specialists, dietitian, general dentist, dental hygienist, psychologist, social worker, and hematologist. Any potential problems should be discussed with the hepatologist before undertaking dental care in these children. The primary objective must be the elimination of dental disease prior to liver transplantation in order to reduce the risk of systemic infection arising from the oral cavity. Clinical and radiographic examinations should ideally be carried out at the first appointment. If possible, posterior bitewing radiographs should be taken in patients where there are approximal contacts, and periapical radiographs of teeth where apical pathology is suspected. Modified lateral oblique views should be obtained where bitewing radiographs are not possible. In the mixed dentition, a panoramic radiograph should be taken. In the very fearful or pre-cooperative child, dental and radiographic examinations and treatment will need to be carried out under general anesthetic at a specialist center.

Emphasis must be on the prevention of dental disease. All infants with significant liver disease should be evaluated between 6-12 months of age and advice on oral hygiene, fluorides, and appropriateness of feeding practices must be given to the parents or caregivers. The importance of effective oral hygiene, performed twice daily with a fluoride toothpaste with supervision by parents or caregivers in children up to seven years, should be stressed. Frequent scaling and polishing is necessary in children who are prescribed nasogastric enteral feeding as they are predisposed to increased calculus deposits on the teeth. Dietary advice should be given in consultation with the dietitian, but is often difficult as high calorie diets are recommended in children where there is failure to thrive. The frequent eating and drinking of sugary foods in between meals should be discouraged. Fluoride supplements,[26] mouthwash or gel, fissure sealants, chlorhexidine mouthwash or gel, and sugar-free oral medications are recommended. Professionally-applied fluoride and chlorhexidine varnishes are useful, especially in the very young child.

The main problems in dental management of children with end-stage liver disease or acute liver failure are similar to those in pediatric cancer patients receiving chemotherapy and perspective bone marrow recipients.[27] These include susceptibility to infection and bleeding tendency. In addition, children with end-stage liver disease have altered drug metabolism and the possibility of significant dysfunction of other organs. Psychological and social problems of these patients are also discussed below.

Susceptibility to infection

Children with severe chronic liver disease may be prone to systemic bacterial infection or bronchopneumonia. The dental surgeon should remove any potential source of infection in the mouth that may lead to systemic infection. A radical approach to treatment is necessary. Pulp therapy, including pulpotomies and pulpectomies, is contraindicated in primary teeth. Stainless steel crowns are usually recommended for the restoration of primary teeth with extensive dental caries. However, CSA and nifedipine induced gingival enlargement may be exacerbated by stainless steel crowns which have inadequate marginal adaptation or in patients with poor oral hygiene.

Children must have prophylactic antibiotic coverage prior to invasive dental procedures if they have chronic liver failure as they are prone to spontaneous bacterial peritonitis which carries a mortality rate ranging from 37%–77%.[28] Streptococci, which are normal commensals in the oral cavity, have been isolated from ascitic fluid of patients with spontaneous bacterial peritonitis, but the source of these bacteria was not stated.[28]

Bleeding tendency

Bleeding tendency in patients with liver disease is due to impaired synthesis of vitamin K; reduced or defective synthesis of clotting factors, in particular, factors II, VII, IX and X; increased consumption of clotting factors; increased fibrinolysis; and thrombocytopenia secondary to portal hypertension and hypersplenism. Portal hypertension can result in esophageal varices, which may rupture and hemorrhage giving rise to anemia. If surgery is required, liaise with the hematologist and hepatologist. Full blood count, platelet count, and bleeding time should be tested. Clotting time should include international normalized ratio (INR), prothrombin time (PT), thrombin time (TT), and activated partial thromboplastin time (APTT). Blood grouping and cross matching are also required.

The PT or INR may be greater than one and a half times the normal in patients with severe parenchymal liver disease. If surgery is undertaken in these patients, fresh frozen plasma or cryoprecipitate may be needed to replace coagulation factors. Platelet replacement should be considered prior to surgical procedures in patients with counts of less than 40,000/mm^3. If a transfusion is required, all procedures which are likely to cause hemorrhage should be completed during one visit and should be as atraumatic as possible. The insertion of resorbable sutures and hemostatic agents, such as microfibrillar collagen or topically sprayed thrombin, may help to prevent post-extraction hemorrhage. High vacuum suctioning should be used to prevent ingestion of blood which may contribute to hepatic encephalopathy in the susceptible patient.[13,29-31]

Analgesics, such as aspirin and non-steroidal anti-inflammatory drugs, should be avoided to reduce the risk of hemorrhage. Block anesthesia is contraindicated in children with clotting defects as intramuscular injection may result in hematoma formation. Intraligamental injection results in a sig-

Table 3. Drugs to Be Avoided or Used with Caution in Liver Disease[33]

Drug	Comment
Antibiotics	
Clindamycin	Reduce dose
Erythromycin estolate	Avoid-may cause idiosyncratic hepatotoxicity
Tetracyclines	Avoid
Flucloxacillin	Avoid-may cause cholestatic jaundice and hepatitis
Metronidazole	Reduce dose in severe liver disease
Ketoconazole	Avoid-hepatitis-like reaction; accumulation in severe liver disease
Analgesics	
Aspirin	Avoid-increased risk of gastro-intestinal bleeding
NSAIDs	Avoid-increased risk of gastro-intestinal bleeding & fluid retention
Opioid analgesics	Avoid-may precipitate coma
Paracetamol	Avoid large doses-dose related toxicity
Lcoal anesthetics	
Lignocaine	Reduce dose in severe liver disease
General anesthetics	
Methohexitione sodium	Avoid or reduce dose
Thiopentone sodium	Avoid or reduce dose
Halothane	Avoid
Muscle relaxants	
Suxamethonium	Avoid in severe liver disease
Antihistamines	Avoid-may precipitate coma
Sedatives & Hypnotics	Avoid-all can precipitate hepatic encephalopathy or coma
Carbenoxolone sodium	Avoid-sodium and water retention and hypokalaemia
Steroids	Avoid prednisone; use prednisolone

nificant bacteremia and should not be used in patients who are susceptible to infection.[32]

Altered drug metabolism

Some drugs which are used in dentistry should be avoided or used with caution in patients with liver disease (Table 3).[33] Unlike renal disease, there are no specific guidelines for dosage modifications in liver disease.[34] If dental treatment is carried out under local anesthetic, excessive amounts of lignocaine must be avoided as it is hepatotoxic. A prolonged half-life of lignocaine has been reported in adults with chronic liver failure who were prescribed the drug in tablet form.[35,36] General anesthesia should only be undertaken at a specialist center where support from all team members is available. Many of these children require general anesthesia for medical procedures, at which time dental treatment may also be undertaken, with good planning and a team approach. Nitrous oxide with pethidine or phenoperidine are the drugs of choice and isoflurane is preferable to halothane.[37]

Congenital heart disease

It is necessary to exclude the presence of congenital heart disease. Twenty to twenty-five percent of children with biliary atresia have significant dysfunction of other organs, including cardiovascular defects such as atrio-septal defect or ventriculoseptal defect.[38] Pulmonary stenosis may be present in children with Alagille syndrome. The cardiologist should be consulted and prophylactic antibiotics should be given for dental procedures that may cause bacteremia according to the recommendations of the American Heart Association.[39]

Psychological and social problems

Children with end-stage liver disease requiring liver transplantation may have enormous psychological problems and their parents may be, understandably, overprotective. The children are often dependent, demanding, and have difficulty coping with even minor procedures. The psychologist may help to reduce the patients' fears by providing support and counseling before or during the dental visit. Patients may still be resistant to even the most skillful behavior management and will subsequently need dental treatment under general anesthetic prior to transplantation.

Post-transplantation

The immediate post-transplantation period

Patients recover in the intensive care unit following liver transplantation. Some of the patients will be on parenteral nutrition for up to four or five days post-transplantation as they are unable to eat. Patients are particularly prone to bacterial and fungal infections in the immediate post-transplant period, which result in considerable morbidity and mortality. Furthermore, potentially harmful changes in the oral streptococcal flora, with increases in the proportions of *Streptococcus oralis* and *Streptococcus mitis*, have been reported in children who are receiving parenteral nutrition following liver transplantation[40] and bone marrow transplantation.[41] These species of oral streptococci are the most frequently isolated bacteria from infections in immunocompromised and neutropenic cancer patients.[42-44] The pediatric dentist, with the help of trained nursing staff and the dental hygienist, must ensure that an intensive mouthcare

Table 4. Drug Interactions With CSA[19,33]

Drug	Effect
NSAIDs	Increased risk of nephrotoxicity
Antibacterials	
Gentamycin	Increased risk of nephrotoxicity
Erythromycin	Increases plasma concentration of CSA
Doxycycline	Increases plasma concentration of CSA
Anesthetic	
Lignocaine	Increases plasma concentration of CSA
Antifungals	
Amphotericin B	Increased risk of nephrotoxicity
Griseofulvin	Possibly reduced plasma concentration of CSA
Fluconazole	Increases plasma concentration of CSA
Barbiturates	Reduced plasma concentration of CSA

policy is in place at this time. The topical application of chlorhexidine may reduce the plaque load, thereby reducing gingivitis and, possibly, the risk of bacteremia. Invasive dental treatment must not be undertaken in the immediate post-transplantation period.

Intermediate post-transplant period (Two weeks to six months post-transplantation)

Invasive dental procedures should be avoided in the intermediate post-transplant period, preferably by comprehensive dental care before the transplant. If dental treatment is necessary, in order, for example, to relieve pain, it should be undertaken in a specialist center. The problems in the dental management of children following liver transplantation, are similar to those in the pre-transplant period. Additional considerations include drug interactions with immunosuppressive therapy, the side effects of these drugs, poor wound healing, and the risk of adrenocorticosteroid suppression.

Infection is still a major problem in patients during the first few months after liver transplantation. Particular attention should be paid to oral opportunistic infections, such as candidiasis and herpetic lesions, which must be treated vigorously in order to eliminate the risk of life-threatening septicemia.[45]

Data on CSA +/- nifedipine induced gingival enlargement is limited but a prevalence rate ranging from 62%–100% has been reported in pediatric liver recipients.[12,13,46,47] Gingival changes occur most rapidly during the first two to six months after administration of CSA, reaching a plateau at about 12 months.[47] Parents and children should be informed of the possible development of drug-induced gingival enlargement and meticulous oral hygiene should be established prior to liver transplantation in order to prevent its onset. Gingival surgery will be necessary in severe cases, but recurrence is likely if the oral hygiene is inadequate. A switch to tacrolimus could also be considered.

The pediatric dentist should be aware of drugs which may interact with CSA (Table 4).[19,33] Tacrolimus is extensively metabolized by the liver and may be affected by a number of drugs (Table 5).[48] The blood level of tacrolimus is increased by erythromycin,[49-51] clarithromycin,[52] and fluconazole,[53,54] and the renal toxicity of tacrolimus is potentiated by ibuprofen.[55]

Coagulation may be defective if the liver is not functioning properly. Platelet counts may be reduced for many months in patients where hypersplenism persists. Many of these patients are on daily aspirin to prevent thrombosis. If invasive dental procedures cannot be avoided, full blood count, platelet count, clotting, and bleeding times will be required.

There is no consensus on the need for prophylactic antibiotics in liver recipients prior to invasive dental procedures. Some authors recommend the use of prophylactic antibiotic cover for all patients,[30,56-58] or for patients needing increased dose of immunosuppressive drugs or those with active dental infection,[29] while others believe that it is unnecessary as there is little evidence of systemic infection in liver recipients following invasive dental procedures.[13,59] It is the authors' opinion that antibiotics according to the recommendations of the American Heart Association[39] should be mandatory for all liver recipients prior to dental procedures that may cause bacteremia as sudden (late) death from overwhelming sepsis is now being reported.[60]

Dental treatment should be carried out under local anesthesia whenever possible. Excessive amounts of lignocaine should be avoided in cases where the liver is not functioning properly.

Patients who are taking prolonged treatments of corticosteroids, or those who have been taking corticosteroids during the previous 12 months (more than 7.5 mg per day of pred-

Table 5. Potential Interactions of Drugs Used in Dentistry With Tacrolimus (Prograf)[48]

Drug	Effect
Substances inhibiting the cytochrome P450 3A (i.e. lidocaine, josamycin, erythromycin, ciprofloxacin, miconazole, itraconazole, ketoconazole, midazolam)	May increase plasma concentration of tacrolimus
Substances inducing the cytochrome P450 3A (i.e. barbiturates, carbamazepine)	May decrease plasma concentration of tacrolimus
Tacrolimus inhibition of the cytochrome P450 3A system-mediated metabolism of other drugs	Inhibition of cortisone
NSAIDs	May reduce plasma protein binding of tacrolimus
Acyclovir, ganciclovir	Potential synergistic neurotoxicity with tacrolimus
Gentamycin and other aminoglycosides; amphotericin B	Potential synergistic nephrotoxicity with tacrolimus

nisolone) are at risk from adrenal gland insufficiency in times of stress.[61] If a stressful situation is anticipated during the dental visit, supplementary corticosteroids are prescribed beforehand in order to help the patient cope.

Long-term management post-transplantation

The pediatric dentist must continue to liaise with the patients' hepatologist and transplant surgeon prior to dental treatment. Chronic rejection and poor graft function would predispose the patient to all the problems associated with chronic liver disease in the pre-transplant period, especially an increased susceptibility to infection. Failure to comply with immunosuppressive therapy, resulting in graft rejection and death, has been reported in teenagers.[60]

The patient should be reviewed routinely every four to six months. Prevention should be reinforced and routine bitewing radiographs taken. Careful examination of the head and neck region, particularly for lymphadenopathy, and intra-oral soft tissues is essential as organ transplant patients have an increased incidence of oral malignancy, such as lymphomas, squamous cell carcinoma, and Kaposi's sarcoma.[62] Though rare, squamous cell carcinoma[63] and Kaposi's sarcoma[64] may present as gingival enlargement. Questionable lesions should be biopsied. Other intra-oral findings include candidiasis, mucosal ulceration, herpetic lesions, esophagitis associated with CMV, and hairy leukoplakia.[62] Prophylactic antibiotics should be mandatory for all liver recipients prior to invasive dental procedures.

Conclusions

Children who undergo liver transplantation have many problems which may present orally or may affect their dental management. Intensive dental care, especially prevention, is essential in all perspective liver recipients in order to reduce the risk of systemic infection arising from the oral cavity. A team approach is necessary as dental care is complicated by a number of factors the most important of which include increased susceptibility to infection, bleeding tendency, and altered drug metabolism. Additional problems following transplantation include drug interactions with immunosuppressive therapy, the side effects of these drugs, poor wound healing, and the risk of adrenocorticosteroid suppression. Antibiotic prophylaxis is mandatory in children with chronic liver failure and in all liver recipients prior to dental procedures that may cause bacteremia. Long-term careful follow-up and reinforcement of prevention are necessary in these children.

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ABSTRACT OF THE SCIENTIFIC LITERATURE

THE USE OF LINERS UNDER AMALGAM RESTORATIONS

Dental amalgam is the most commonly used material to restore posterior teeth in many countries due to ease of placement, low cost, strength, and longevity. However, amalgam does not adhere to tooth surfaces predisposing the restoration to marginal leakage leading to secondary caries and necessity for replacement of the restoration. The purpose of this study was to evaluate the effect of various liners on amalgam restorations.

Fifty human premolars, extracted for orthodontic, received Class II amalgam preparations and were divided into five groups. Prior to amalgam condensation, the preps were treated as follows: Group 1 received All-Bond 2 and Liner F, Group 2 was coated with topical fluoride gel for two minutes, Group 3 received Copalite, Group 4 had the dentin lined with Vitrebond, and Group 5 served as the control without a liner. Dispersalloy was manually condensed into all preparations. The teeth were coated with nail polish, except around the restoration margins, and were thermocycled (1000 cycles) in baths containing dye. The teeth were then rinsed for six hours, dried, sectioned longitudinally and examined microscopically.

At the enamel margins, 90% of the teeth lined with GIC and the controls showed no leakage. Whereas, 80% of the teeth lined with adhesive had some leakage, and 60% of the teeth with cavity varnish and 50% of the teeth with fluoride had leakage along the axial wall to the pulp. At the dentin margins, 90% of the teeth lined with GIC had no leakage, 90% of the teeth lined with adhesive and the control teeth had some leakage, and 100% of the teeth with cavity varnish and fluoride had leakage along the axial wall to the pulp.

The results suggest that the current adhesive systems are unable to stop marginal leakage. However, the adhesive-lined restorations performed better than those lined with varnish or fluoride. GIC used as a liner under amalgam restorations had the best performance on dentin and was equal to the control group on enamel. Since the margins of amalgam restorations are almost always in enamel, this study supports the idea that liners offer no advantage in reducing marginal leakage. Additionally, application of liners is more costly and more time consuming.

Comment: The authors reference a number of studies, in vivo and in vitro, which are consistent with their findings. Cavity liners for amalgam restorations do not enhance marginal integrity. It appears that clinicians need not spend the extra time, and more importantly, the additional cost, which must be passed on to the patient. CFGW

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