Gastrointestinal Disorders in The Dialysis Population

Chagriya Kitiyakara, M.B. B.S., MRCP (UK)
Narin Achalanan, M.D.*

EXTRACT

Gastrointestinal symptoms are among the commonest complaints in the dialysis population(1). The causes and treatment of many gastrointestinal problems are similar in patients receiving the renal replacement therapy as in the general population. This review focuses on gastrointestinal disorders with special relevance to patients with end-stage renal disease (ESRD) receiving dialysis with some mention of patients, who have had kidney transplantation.

Key words: Gastrointestinal, dialysis, renal replacement

INTRODUCTION

Gastrointestinal symptoms are among the commonest complaints in the dialysis population(1). The causes and treatment of many gastrointestinal problems are similar in patients receiving the renal replacement therapy as in the general population. This review focuses on gastrointestinal disorders with special relevance to patients with end-stage renal disease (ESRD) receiving dialysis with some mention of patients, who have had kidney transplantation.

Anorexia

In the dialysis population, anorexia is common and is associated with significant morbidity(2). Inadequate dialysis, post-hemodialysis fatigue, anemia and various comorbid, psychosocial and socioeconomic factors contribute to anorexia. Abdominal discomfort, the absorption of glucose and peritonitis also reduce appetite in the continuous peritoneal dialysis(CAPD) population.

Nausea and Vomiting

Nausea and vomiting occurs in up to two-thirds of patients on dialysis(3). In new hemodialysis (HD) patients, nausea is a feature of the disequilibrium syndrome(4) due to rapid changes in BUN and subsequent transcellular water flux. Uremic toxins cause nausea and inadequate dialysis needs to be excluded. In addition, nausea can occur at the end of HD sessions in association with hypotension(5). Excessive fluid removal and rapid changes in plasma osmolality contribute to the development of these symptoms as well. Less commonly, nausea is part of a reaction against

*Division of Nephrology, *Division of Gastroenterology and Tropical Medicine, Ramathibodi Hospital, Bangkok 10400, Thailand
cellulosic dialyzer membranes\(^6\). Changing to synthetic biocompatible membranes may be helpful. Intercurrent illness, medications (eg oral iron, digoxin toxicity) may be the cause in some cases. Gastrointestinal diseases including diabetic gastroparesis, constipation and gastritis need to be excluded and treated as appropriate.

**Upper Gastrointestinal Disease**

**Oropharynx**

Metallic taste, stomatitis, and less commonly, parotitis may occur in patients with chronic renal failure. Oral ulcers may be due to infections, connective tissue diseases or drugs. Herpes simplex and candidiasis may occur in the immunocompromized renal transplant recipients\(^7\). Antiviral or antifungal should be used as appropriate.

**Esophagus**

CAPD patients have increased gastroesophageal reflux symptoms\(^10\). In one study, symptomatic patients had higher reflux score based on 24 hour pH monitoring and decreased supine lower esophageal sphincter tone upon dialysate infusion compared to asymptomatic controls\(^9\). By contrast, other investigators found no differences in lower esophageal sphincters tone between symptomatic and asymptomatic patients\(^10\). In general, the diagnosis and treatment for gastroesophageal reflux in ESRD is similar to the general population\(^11\). Minimizing CAPD exchange volumes especially at night may be considered in some patients provided that clearance is not compromised.

Erosive esophagitis was found to be more prevalent in dialysis patients than the general population by some investigators\(^12\) but not by others\(^3\). Patients with renal transplants are at increased risk from opportunistic infections e.g candida, CMV and herpes simplex\(^7\). In these cases, esophageal symptoms may be accompanied by odynophagia. Such patients should have the diagnosis confirmed by endoscopy and treated appropriately.

**Gastritis and Duodenitis**

Most endoscopic studies found a high prevalence of gastritis and duodenitis in the dialysis population\(^3,13-18\). Dyspeptic symptoms correlate poorly with the presence of pathology\(^3\). In one series of mostly asymptomatic HD subjects, macroscopic gastritis or duodenitis were present in 36% of patients and 24% had erosions\(^18\). In another study, the prevalence of histologic gastritis was 89% compared to 21 % in controls\(^15\). The relative prevalence of chronic superficial gastritis and atrophic gastritis appear to vary between studies\(^3,19,20\).

Several factors likely contribute to the increases in gastritis in dialysis patients. Uremia per se may contribute. In undialyzed patients who died of uremia, hemorrhagic, ulcerative and pseudomembranous changes were found in the mucosal membranes at all levels of the digestive tract including the stomach\(^21\). In another autopsy study in dialyzed patients, frequent mucosal changes were seen, but these severe inflammatory features were absent\(^22\).

*Helicobacter pylori* is a major cause of gastritis in the general population\(^23\). The increase in the availability of urea, a substrate for *H. pylori* metabolism might be expected to increase the prevalence of *H. pylori* infection in the dialysis population. However, the rate of seroprevalence of *H. pylori* in the HD\(^24-28\) and transplant recipients\(^25,29\), CAPD\(^29\) population is 30-70%, which is comparable to the general population. Furthermore, there does not appear to be an increase in pathogenic strains of *H. pylori* (i.e.producing vacuolating cytotoxins or CagA-positive\(^30\)) in the dialysis population\(^31\).

Studies on the role of acid secretion in uremic gastritis appear to be contradictory probably reflecting inadequate study size and the failure to distinguish dialysis subjects and predialysis subjects. Early studies found increased basal\(^32\) and pentagastrin stimulated\(^33,34\) gastric acid production. Gastrin levels are elevated in patients with renal failure\(^17,35-41\), even up to levels comparable to those with gastrinomas\(^35,36\). In part, this increase may be due to the decreased renal clearance of gastrin\(^42\), but enhanced gastrin synthesis may contribute\(^43\). The elevated gastrin levels have been proposed as an important stimulus for acid secretion in renal failure. However, other investigators did not find increases in pentagastrin response in dialysis patients\(^36,44\). Wide ranges for acid secretion have been found. A proportion of patients had hypochlorhydria whereas another proportion had hyperchlorhydria\(^39\). Rather than being the cause of gastric hypersecretion, the elevated gastrin levels may be the consequence of a lack of negative feedback response to the undersecretion of acid, because of atrophic gastritis \(^40,41\).
Several studies found an inverse correlation between gastrin levels and acid secretion in the dialysis population\(^{18,41}\). *H. pylori* infection may also account for hypergastrinemia in some patients since eradication of *H. pylori* is associated with the reduction in serum gastrin and reductions in gastric juice ammonia and pH levels\(^{44}\).

Biliary reflux can worsen gastroduodenitis. Since gastrin decreases pyloric sphincter tone, high gastrin levels may lead to biliary reflux, thus worsen mucosal injury\(^{45}\). In the duodenum, impaired bicarbonate secretion\(^{46-47}\) and increased pepsinogen release\(^{19,46,48}\) may also contribute to mucosal inflammation.

In patients with gastritis, non-steroidal anti-inflammatory agents (NSAIDs) should be discontinued. Acid suppression with H2 receptor blockers or proton pump inhibitors should be initiated. Antacids containing magnesium or aluminium should be avoided\(^{49}\). Sucralfate is also associated with aluminum toxicity\(^{50,51}\).

**Peptic Ulcers**

Between different studies, there is a large variation in the frequency of peptic ulcers in patients with chronic renal failure compared to non-uremic subjects. Early reports suggest an increase in incidence of peptic ulcer in the dialysis population\(^{37,52}\). However, more recent and larger studies found no increase in either gastric or duodenal ulcers\(^{13,18,53}\). These differences might be attributed to the differences in diagnostic method (radiology vs endoscopy), sample size and criteria for selection of patient and control groups as well as the use of antibiotics and H2 or proton pump inhibitors. Thus in a study 114 of unselected, asymptomatic and symptomatic dialysis patients, only 2% of patients were found to have endoscopic evidence of peptic ulcers, which was similar to the prevalence in the general population\(^{18}\).

Similar to the general population, *H. pylori* should be identified and treated. The diagnosis of *H. pylori* is established by endoscopy by one of three methods: biopsy urease test, histology or less commonly, bacterial culture. However, ultrarapid urease test may be less sensitive and specific for *H. pylori* infection in dialysis subjects with elevated plasma urea concentrations\(^{54}\). Noninvasive strategies for diagnosis of *H. pylori* infection are also available \(^{55}\). These tests include stool antigen testing, isotope-urea breath test or serology. using ELISA technology for IgG or IgA.

The 13C urea breath test relies on bacterial hydrolysis of isotope-labeled carbon dioxide after administration of isotope-labeled urea. Even when this test is performed after dialysis, it still has decreased specificity for *H. pylori* in uremic subjects\(^{56}\). In one study of ESRD patients using endoscopic biopsy specimen as the gold standard, stool antigen testing was over 97% sensitive and specific for the presence of *H. pylori* whereas Ig G screening was only 87% sensitive and 80% specific\(^{57}\). Moreover, stool antigen testing was 100% sensitive and 97.5% specific for detecting failure of *H. pylori* eradication whereas serology was only 22% specific.

As in the general population, eradication of *H. pylori* and cessation of NSAIDs and smoking and the acid suppression is the mainstay of treatment. Treatment of *H. pylori* infection is similar to the general population\(^{23}\) except regimens containing bismuth may best be avoided to accumulation in ESRD\(^{58}\). These protocols appear to be effective in CAPD\(^{59}\) and hemodialysis patients\(^{60}\) with cure rates similar to non-uremic subjects.

Renal transplant recipients, however, have significantly increased risk of peptic ulcers with higher rates of complications\(^{14,61}\). Low dose aspirin, high dose steroid in treatment of acute rejection appear to be important risk factors, whereas the presence of *H. pylori* appears to be less important \(^{62}\). Opportunistic infections such as CMV also need to be considered as a cause of gastroduodenal ulceration in this population\(^7\). Pretransplant evaluation and perioperative H2 antagonists or proton pump inhibitors should be used to prevent post transplant gastroduodenal ulceration\(^{63}\).

**Gastric Emptying**

Undialyzed, uremic patients frequently have delayed gastric emptying\(^{64}\). However, gastric emptying in both symptomatic or asymptomatic non-diabetic HD patients was found to be impaired by some investigators\(^{65}\) but not by others\(^{66,67}\). Gastric motility is regulated by gastric myoelectrical activity. Increased abnormal myoelectrical activities in both fasting and fed state have been documented in renal failure\(^{68,69}\). HD decreased the normal gastric frequency in some studies\(^{70}\), but not in others\(^{69}\). Instillation of peritoneal fluid may induce gastric dysrhythmia CAPD patients\(^{69}\). Abnormal gastric rhythm in CAPD subjects was associated with early satiety. Indeed, slower gas-
tric emptying has been documented in patients studied at the time of peritoneal fluid dwell period\(^{71,72}\).

Gastric motility disorders however, are common in dialysis patients with diabetes mellitus\(^{73}\). Patients with gastroparesis had lower muscle mass and had 3 times as many days in hospital and one third median survival compared to diabetics on dialysis without gastroparesis. The importance of gastric motility dysfunction in dialysis subjects is underscored by the findings that, in patients with subclinical gastric emptying defects, treatment with prokinetic therapy increased the serum albumin\(^{74,75}\).

Uremic toxins and electrolyte abnormalities may contribute to the development of gastric dysmotility in uremia. Some non-diabetic patients with renal failure have a degree of autonomic neuropathy\(^{76}\). In addition, the plasma concentrations of gastrointestinal hormones / peptides are elevated as a consequence of decreased renal and extra-renal clearance\(^{37,77-81}\). The fasting levels of gastrin, cholecystokinin, gastric inhibitory polypeptides, glucagon, vasoactive intestinal peptides, motilin are elevated but the appropriate post-prandial increase is maintained. The serum levels revert back to normal after successful transplantation but are relatively unaffected by HD and CAPD\(^{77-82}\). These hormones at concentrations found in renal failure could affect GI motility (gastrin, motilin, cholecystokinin) or could regulate appetite and satiety (glucagon, cholecystokinin). However, the overall effects of the accumulation of gastrointestinal peptides on GI motility are unclear, since these hormones have complex synergistic and antagonistic interactions.

Prokinetic agents may be considered in the treatment of impaired gastric emptying with appropriate dose adjustment. In the non-uremic population, metoclopramide appeared to be as effective as domperidone, but was associated with higher extrapyramidal side effects\(^{83}\). Intravenous erythromycin has been used to kick start the stomach in severe gastric stasis\(^{84}\) and intraperitoneal erythromycin (100mg/2L bag) has been used successfully in CAPD patients with severe uncontrolled vomiting\(^{85}\). Since potential complications include pseudomembranous colitis and GI toxicity, chronic use of erythromycin (oral) should be limited to those who do not respond to traditional therapies. Cisapride is a highly effective prokinetic agent. Unfortunately, the drug has been withdrawn from the US market because of several incidences of torsade de pointes and QT interval prolongation especially in patients with renal failure\(^{86}\). However, in a small retrospective analysis, Hentges et al was unable to demonstrate a significant effect on the QT interval after the administration of cisapride in stable HD patients\(^{87}\). The authors concluded that in ESRD patients with severe symptomatic gastroparesis unresponsive to other therapies, cisapride might be considered, provided that there is no underlying cardiac disease or concomitant use of drugs that inhibits the cytochrome P450 3A4 (eg erythromycin). Clearly these patients will require regular electrocardiogram monitoring for prolongation of the QT interval and other arrhythmias if cisapride is used.

**LOWER GASTROINTESTINAL DISEASE**

**Small Bowel Function**

In undialyzed severely uremic subjects, the small bowel may show mucosal edema, hemorrhage and even severe necrotizing ulcers\(^{21}\). In the dialyzed population, minor morphologic changes consisting of modest reduction of villus height, increased crypt depth and infiltration of lamina propria with inflammatory cells associated with normal d-xylose absorption and normal dipeptidases and disaccharidases activity are found\(^{88}\). If malabsorption is suspected, d-xylose absorption and d-xylose breath test appears to be an effective absorption test in this population\(^{89}\). In the transplant population, there is an increase in the incidence of small bowel lymphoma and carcinoma, however, the incidence of these rare malignancies is still low\(^{90}\).

**Ischemia**

Patients with ESRD have accelerated atherosclerosis and a high prevalence of cardiomyopathy\(^{91}\) that can result in increased risk of mesenteric ischemia and infarction. During HD, splanchnic blood flow is decreased even when blood pressure remains within the normal range\(^{92}\). This effect is usually transient but likely is more severe and more persistent if there is hypotension. Several studies cite hypotension occurring during HD as a precipitating event to ischemic bowel infarction\(^{93-96}\). Twenty-nine of 1,370 long term HD patients developed non-occlusive mesenteric infarction over a five year period\(^{96}\). Fifty-five percent had ischemia of the small bowel and the rest had ischemia of the colon. Overall mortality rate was 45%.
Indeed, mesenteric ischemia is one of the commonest causes for emergency bowel surgery in the dialysis population and is associated with high morbidity and mortality (97-99).

**Constipation and Bowel Obstruction**

Constipation is a common symptom in dialysis patients. Metabolic abnormalities associated with uremia and medications including aluminum, calcium carbonate and oral iron, constitute major risks to the development of constipation (100). In CAPD patients, constipation can impair peritoneal fluid drainage. In treating constipation, predisposing medications should be stopped. Intravenous iron could be used instead of the oral form since they cause constipation three times less often (101). Osmotic laxatives e.g. lactulose, stool softeners e.g. docusate sodium, and stimulants e.g. bisacodyl can be safely recommended. Soap suds, mineral oil or bisacodyl suppositories can be used. GoLytely can be used for bowel preparations since the water and osmolyte content are not absorbed. Psyllium (Metamucil) fiber should be avoided because of the presence potassium and high fluid intake requirement. Magnesium containing laxatives should be avoided because of the danger of hypermagnesemia. Phosphate (Fleet) enema should be avoided because of excessive phosphate absorption (102).

Obstruction and pseudoobstruction occur more commonly in dialysis patients (103,104). CAPD patients are at increased risk of developing hernias (105,106). Occasionally, peritoneal dialysis catheter-related complications may present with bowel obstruction (107). Sclerosing encapsulating peritonitis is a relatively uncommon cause of intestinal obstruction in CAPD patients (108-110). Patients with this complication may present decreased peritoneal fluid clearance and weight loss in association with repeated peritonitis. Early recognition and conversion to HD is essential. Surgical procedures may be necessary but have high complication rates. Immunosuppressive agents have been used in a small group of subjects with some beneficial effects (111).

**Diverticular Disease**

The prevalence of diverticulosis and diverticulitis is increased in patients with renal failure due to adult polycystic kidney disease but not due to other causes (112,113). In this population, the diagnosis of diverticulitis can be confused with bleeding or infection of a cyst. The presence of diverticular disease may special impact on patients on renal replacement therapy. Active diverticulitis is a contraindication to CAPD. Although diverticular disease is not a contraindication to CAPD, the presence of multiple or large diverticula may increase the risk of developing peritonitis by enteric organisms (114). Colonic complications after renal transplantation are uncommon, but have a high morbidity and mortality rate (115,116). Perforation from diverticulosis may be increased in patients on high dose steroids, in whom symptoms and signs may be masked initially (117). Thus dialysis patients with active diverticulitis may require surgical treatment prior to transplantation. Some have recommended colonic screening in patients over 50 years of age prior to transplantation to lessen the impact of colonic diverticular disease. However, in the absence of diverticulitis, it is not clear if this practice has clear impact on outcome (118) even in patients with adult polycystic kidney disease (119).

**Clostridium Difficile Infection**

Frequent antibiotic use, impaired immune function and decreased gastrointestinal motility predisposes dialysis patients to C difficile infection (120). Hospitalized HD patients may have increased risk of Clostridium difficile infection (121). Large outbreaks have been reported in dialysis centers (133). Although often dialysis patients have severe symptoms, some can be asymptomatic. Because of the risk of spread in HD units, there should be a low threshold in testing for C difficile toxin in patients with diarrhea.

**Ulceration and Perforation**

Undialyzed, severely uremic subjects may have gross mucosal changes affecting the colon (21). However, routine endoscopic studies of the colon in asymptomatic patients on maintenance dialysis showed few abnormalities (116). In addition to the usual causes of intestinal perforation, including ischemia, obstruction and diverticular disease, less common causes of perforation have been reported in the dialysis population.

Bowel perforation has also been reported as a complication of indwelling peritoneal catheters in CAPD (123). The use of sodium polystyrene (Kayexelate) in the treatment of hyperkalemia may result in bowel necrosis (124,125). The risk appears to be higher in the perioperative period and during concurrent the administration of hypertonic sorbitol enema (126). Thus
sorbitol enema should not be used and sodium polystyrene should be avoided in patients with bowel ileus.

Idiopathic colonic ulcers have been reported in dialysis patients\(^{127,128}\) and may resent with bleeding, or peritonitis, and show a high mortality rate. Spontaneous idiopathic perforation\(^{129-131}\) and segmental necrosis\(^{132}\) of the ascending colon has also been described. The causes of these diseases are unknown, but may relate to the use of aluminum-based antacids, ischemia and impaired intestinal motility.

In CAPD patients, bowel perforation is suggested by the findings of unusual enteric\(^{133}\) or mixed organisms\(^{134}\). Fecal material in the dialysate and diarrhea containing dialysate are uncommon. However, mixed organisms can be found even in absence of perforation or diverticulitis\(^{135}\). The diagnosis of bowel perforation in patients on CAPD can be delayed because of mild symptoms, and the absence of classical abdominal guarding and rigidity and confusion with the more typical staphylococcal peritonitis\(^{136}\). In addition, the presence of free air by plain x-ray occurs in up to 30% of patients on CAPD, irrespective of whether there is a perforated viscus\(^{137}\). The lack of free air on CT scan may have a greater negative predictive value than X-ray, but cannot rule out bowel rupture with absolute certainty\(^{138}\).

**Diseases Affecting Both Upper and Lower Tracts**

**Angiodysplasia**

Angiodysplasia are composed of ectatic, dilated, thin walled vessels, that are lined by endothelium alone or by only small amounts of smooth muscle\(^{139}\). Angiodysplasia is a more frequent cause of gastrointestinal bleeding and recurrent bleeding in patients with chronic renal failure than in those with normal renal function\(^{139-146}\). They are often multiple and can be found throughout the entire GI tract, from the stomach to the large bowel. Their prevalence increases with the duration and severity of renal failure\(^{146}\). Renal failure is also a risk factor for the development of the watermelon stomach, another type of vascular malformation in the antral region of the stomach\(^{147}\). The causes for the increased prevalence of vascular malformations have been attributed to vascular calcification, constipation and chronic venous congestion from volume overload\(^{139}\).

Angiodysplasia are usually diagnosed by endoscopy or angiography. \(^{99m}\)Tc-labelled red cell scintigraphy can also be used to localize lesions\(^{148}\). Endoscopic therapy could be applied to bleeding lesions. Local instillation with vasopressin during angiography or embolization may be considered in patients with severe bleeding and surgical resection considered in patients with uncontrolled, life-threatening bleed. The management of angiodysplasia, which is not actively bleeding is less clear. In patients with angiodysplasia with previous hemorrhage or with otherwise unexplained occult blood loss, therapeutic ablation could be considered. The inaccessibility of some lesions, the multiplicity of angiodysplasia and potential complications associated with treatment could limit the success of local therapy.

Hormonal therapy with estrogen has been used to treat patients with angiodysplasia. The mechanism of action is unclear but treatment has been associated with improved bleeding times\(^{149}\). At present, randomized controlled trials are lacking in the dialysis population. In small uncontrolled studies, the combination of synthetic estrogen-progesterone\(^{150}\) or estrogen\(^{151}\), have been shown to be beneficial in chronic renal failure patients with angiodysplasia\(^{162}\). In a prospective study, combined estrogen and progesterone or estrogen alone was given to non-uremic patients with recurrent GI bleed with angiodysplasia or an unknown bleeding source\(^{163}\). None of the 38 patients who were treated with combination hormonal therapy rebled as long as they continued their prescribed dosage. The role of progesterone is not clear but all five of the patients treated with estrogen alone had reblooding episodes, but had no further bleeding after switching to combined therapy. Side effects occurred in 11 patients. However, not all studies have shown benefit for hormonal therapy\(^{153}\). Thus the potential benefits of hormonal therapy need to be balanced against their possible side-effects. More recently, octreotide has been used in a small number of patients with angiodysplasia\(^{154}\). The antifibrinolytic agent, tranexemic acid also been used during acute bleeding, and chronically to prevent rebloody\(^{155}\). The use of these agents need to be defined in the context a clinical trial

**Amyloidosis**

Although beta 2-microglobulin amyloidosis typically affects the joints and bones, systemic deposition
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can also occur in the gastrointestinal tract\(^{(156)}\). The presence of visceral beta 2-amyloid deposit is associated with longer duration on dialysis and should be considered in patients who have been on dialysis for over 10 years\(^{(157)}\). Amyloidosis can cause bleeding or massive diarrhea\(^{(158,159)}\), intestinal infarcts\(^{(160)}\), perforation\(^{(161)}\) and pseudo-obstruction\(^{(162,163)}\). GI complaints have been reported even in the absence of overt joint complaints\(^{(163)}\). Definitive diagnosis is made by biopsy and staining with congo red and immunostaining for beta 2-microglobulin\(^{(157)}\). Vascular histopathology range from mild focal thickening of vessel walls to massive vascular beta 2-microglobulin amyloid deposition with thrombosis and marked expansion of the submucosa. Currently, no specific treatment for visceral beta-2 amyloidosis can be recommended.

**Gastrointestinal Bleeding**

Dialysis patients have increased risk of gastrointestinal bleeding. Even in patients without overt symptoms, there appears to be 7-folds increase in GI blood loss\(^{(164)}\). This is due to the combination of the increased prevalence of angiodysplasia and gastritis and due to the coagulopathy associated with uremia and systemic heparinization\(^{(165)}\). Like the general population, peptic ulcers are among the most common causes of acute GI bleeding\(^{(12,146)}\). Recurrent bleeding is also more likely. Upper gastrointestinal hemorrhage in dialysis subjects is associated with the use of ulcerogenic drugs such as steroids, iron and NSAIDs\(^{(166,167)}\). GI hemorrhage occurred in 13% of patients with acute renal failure despite the routine use of prophylactic H2 antagonists\(^{(168)}\). Upper GI disease with gastroduodenal erosions and ulcers account for over 50% of all acute bleeding episodes. The severity of the underlying illness and liver disease are major risk factors.

Because of the higher prevalence of angiodysplasia in patients with chronic renal failure, both upper and lower GI endoscopy is more appropriate than barium studies in evaluating GI bleeding. In the general population, intravenous omeprazole decreases rebleeding from active gastroduodenal ulcers\(^{(169)}\) and may decrease this risk in patients with renal failure. During acute bleeding episodes, platelet defects should be corrected by maintaining adequate dialysis, administration of desmopressin (DDAVP 0.3 ug/kg iv), or cryoprecipitate\(^{(165)}\). Maintenance of adequate level of hematocrit and red cell mass with transfusion and erythropoietin may also be beneficial. HD should be performed with no heparin and blood products may need to be administered during dialysis to avoid volume overload or electrolyte imbalance.

**Colorectal Cancer Screening**

The premise behind colorectal cancer screening is that malignancy develops from benign colonic polyps to cancer over several years\(^{(170)}\). The 2 principal screening tests, recommended in patients with average risks over the age of 50 are fecal occult blood testing (annually) and flexible sigmoidoscopy (every 5 years). For colorectal screening, the important studies showing improved survival with fecal occult blood\(^{(171)}\) and flexible sigmoidoscopy\(^{(172)}\) show a lag time of 5 to 7 years before the survival effect is shown. Dialysis patients have a shorter life-expectancy than do patients of the same age without renal failure with the overall 7 year survival of only 30% for non-diabetic and 10% for diabetic patients\(^{(173)}\). Thus routine colorectal screening should be reserved for those with good prognosis.

The incidence of GI malignancy does not appear to be consistently increased in dialysis patients\(^{(174)}\). The more frequent blood loss in patients with renal failure increases the prevalence of positive heme occult blood. In one small study, the rate of heme occult positive blood was 15% in dialysis population compared to 5% of controls\(^{(175)}\). Thus the incidence of colon cancer/polyp in heme occult positive blood was lower than the general population but some occult malignancies were identified\(^{(175,176)}\). Stool blood testing may still have a role in identifying potentially treatable, non-malignant GI lesions responsible for poor erythropoietin response\(^{(177)}\). In the transplant population, however, there is a three folds risk of colonic carcinoma\(^{(91)}\). The possible role for colonoscopy screening has been discussed for the general population at moderate risks\(^{(178)}\), but the benefits of screening remains to be evaluated in the transplant population.

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