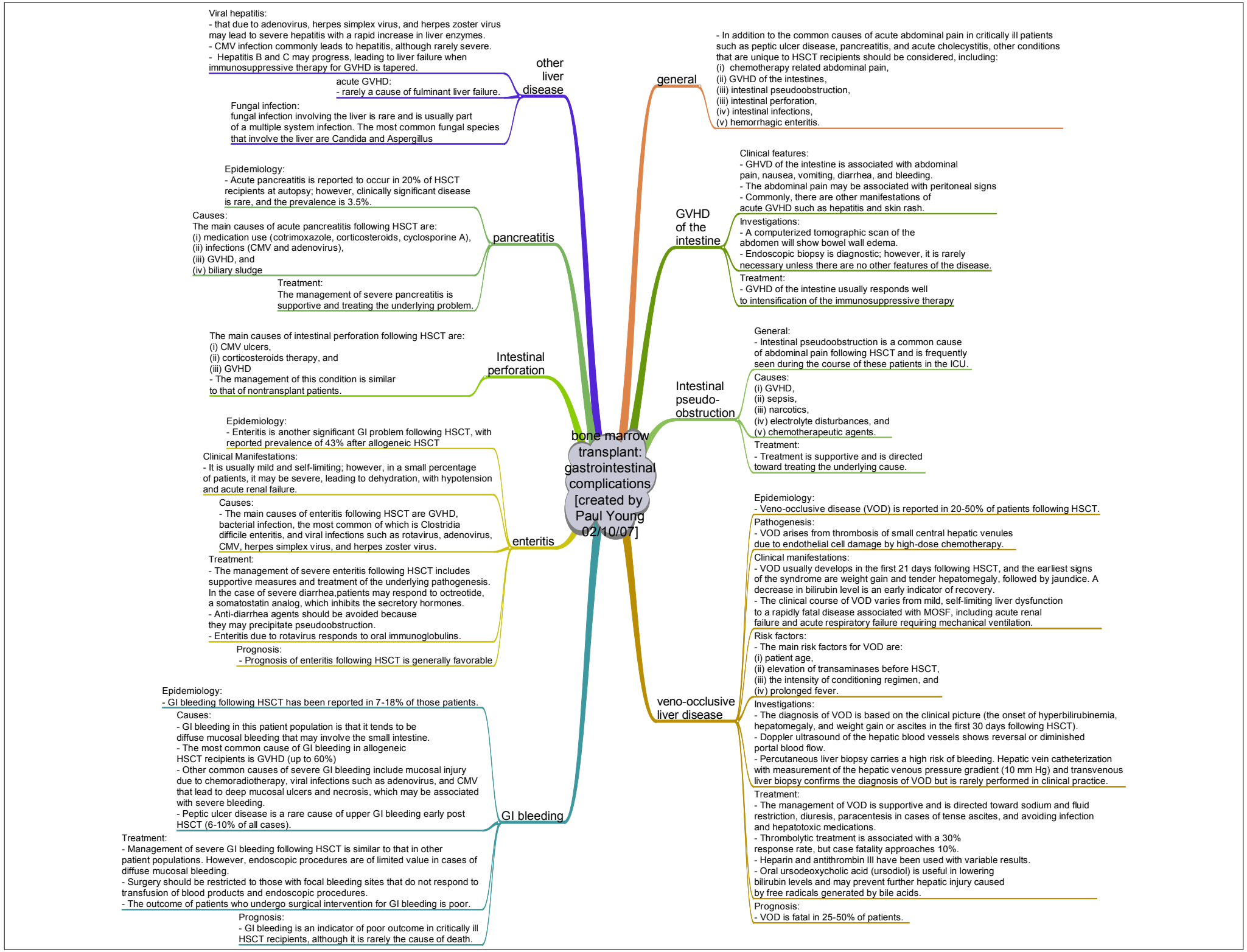
bone marrow transplant: gastrointestinal complications [created by Paul Young 02/10/07]
other liver disease
Viral hepatitis:
- that due to adenovirus, herpes simplex virus, and herpes zoster virus may lead to severe hepatitis with a rapid increase in liver enzymes.
- CMV infection commonly leads to hepatitis, although rarely severe.
- Hepatitis B and C may progress, leading to liver failure when immunosuppressive therapy for GVHD is tapered.
acute GVHD:
- rarely a cause of fulminant liver failure.
Fungal infection:
fungal infection involving the liver is rare and is usually part of a multiple system infection. The most common fungal species that involve the liver are Candida and Aspergillus
pancreatitis
Epidemiology:
- Acute pancreatitis is reported to occur in 20% of HSCT recipients at autopsy; however, clinically significant disease is rare, and the prevalence is 3.5%.
Causes:
The main causes of acute pancreatitis following HSCT are:
(i) medication use (cotrimoxazole, corticosteroids, cyclosporine A),
(ii) infections (CMV and adenovirus),
(iii) GVHD, and
(iv) biliary sludge
Treatment:
The management of severe pancreatitis is supportive and treating the underlying problem.
Intestinal perforation
The main causes of intestinal perforation following HSCT are:
(i) CMV ulcers,
(ii) corticosteroids therapy, and
(iii) GVHD
- The management of this condition is similar to that of nontransplant patients.
enteritis
Epidemiology:
- Enteritis is another significant GI problem following HSCT, with reported prevalence of 43% after allogeneic HSCT
Clinical Manifestations:
- It is usually mild and self-limiting; however, in a small percentage of patients, it may be severe, leading to dehydration, with hypotension and acute renal failure.
Causes:
- The main causes of enteritis following HSCT are GVHD, bacterial infection, the most common of which is Clostridia difficile enteritis, and viral infections such as rotavirus, adenovirus, CMV, herpes simplex virus, and herpes zoster virus.
Treatment:
- The management of severe enteritis following HSCT includes supportive measures and treatment of the underlying pathogenesis. In the case of severe diarrhea,patients may respond to octreotide, a somatostatin analog, which inhibits the secretory hormones.
- Anti-diarrhea agents should be avoided because they may precipitate pseudoobstruction.
- Enteritis due to rotavirus responds to oral immunoglobulins.
Prognosis:
- Prognosis of enteritis following HSCT is generally favorable
GI bleeding
Epidemiology:
- GI bleeding following HSCT has been reported in 7-18% of those patients.
Causes:
- GI bleeding in this patient population is that it tends to be diffuse mucosal bleeding that may involve the small intestine.
- The most common cause of GI bleeding in allogeneic HSCT recipients is GVHD (up to 60%)
- Other common causes of severe GI bleeding include mucosal injury due to chemoradiotherapy, viral infections such as adenovirus, and CMV that lead to deep mucosal ulcers and necrosis, which may be associated with severe bleeding.
- Peptic ulcer disease is a rare cause of upper GI bleeding early post HSCT (6-10% of all cases).
Treatment:
- Management of severe GI bleeding following HSCT is similar to that in other patient populations. However, endoscopic procedures are of limited value in cases of diffuse mucosal bleeding.
- Surgery should be restricted to those with focal bleeding sites that do not respond to transfusion of blood products and endoscopic procedures.
- The outcome of patients who undergo surgical intervention for GI bleeding is poor.
Prognosis:
- GI bleeding is an indicator of poor outcome in critically ill HSCT recipients, although it is rarely the cause of death.
general
- In addition to the common causes of acute abdominal pain in critically ill patients such as peptic ulcer disease, pancreatitis, and acute cholecystitis, other conditions that are unique to HSCT recipients should be considered, including:
(i) chemotherapy related abdominal pain,
(ii) GVHD of the intestines,
(iii) intestinal pseudoobstruction,
(iii) intestinal perforation,
(iv) intestinal infections,
(v) hemorrhagic enteritis.
GVHD of the intestine
Clinical features:
- GHVD of the intestine is associated with abdominal pain, nausea, vomiting, diarrhea, and bleeding.
- The abdominal pain may be associated with peritoneal signs
- Commonly, there are other manifestations of acute GVHD such as hepatitis and skin rash.
Investigations:
- A computerized tomographic scan of the abdomen will show bowel wall edema.
- Endoscopic biopsy is diagnostic; however, it is rarely necessary unless there are no other features of the disease.
Treatment:
- GVHD of the intestine usually responds well to intensification of the immunosuppressive therapy
Intestinal pseudo-obstruction
General:
- Intestinal pseudoobstruction is a common cause of abdominal pain following HSCT and is frequently seen during the course of these patients in the ICU.
Causes:
(i) GVHD,
(ii) sepsis,
(iii) narcotics,
(iv) electrolyte disturbances, and
(v) chemotherapeutic agents.
Treatment:
- Treatment is supportive and is directed toward treating the underlying cause.
veno-occlusive liver disease
Epidemiology:
- Veno-occlusive disease (VOD) is reported in 20-50% of patients following HSCT.
Pathogenesis:
- VOD arises from thrombosis of small central hepatic venules due to endothelial cell damage by high-dose chemotherapy.
Clinical manifestations:
- VOD usually develops in the first 21 days following HSCT, and the earliest signs of the syndrome are weight gain and tender hepatomegaly, followed by jaundice. A decrease in bilirubin level is an early indicator of recovery.
- The clinical course of VOD varies from mild, self-limiting liver dysfunction to a rapidly fatal disease associated with MOSF, including acute renal failure and acute respiratory failure requiring mechanical ventilation.
Risk factors:
- The main risk factors for VOD are:
(i) patient age,
(ii) elevation of transaminases before HSCT,
(iii) the intensity of conditioning regimen, and
(iv) prolonged fever.
Investigations:
- The diagnosis of VOD is based on the clinical picture (the onset of hyperbilirubinemia, hepatomegaly, and weight gain or ascites in the first 30 days following HSCT).
- Doppler ultrasound of the hepatic blood vessels shows reversal or diminished portal blood flow.
- Percutaneous liver biopsy carries a high risk of bleeding. Hepatic vein catheterization with measurement of the hepatic venous pressure gradient (10 mm Hg) and transvenous liver biopsy confirms the diagnosis of VOD but is rarely performed in clinical practice.
Treatment:
- The management of VOD is supportive and is directed toward sodium and fluid restriction, diuresis, paracentesis in cases of tense ascites, and avoiding infection and hepatotoxic medications.
- Thrombolytic treatment is associated with a 30% response rate, but case fatality approaches 10%.
- Heparin and antithrombin III have been used with variable results.
- Oral ursodeoxycholic acid (ursodiol) is useful in lowering bilirubin levels and may prevent further hepatic injury caused by free radicals generated by bile acids.
Prognosis:
- VOD is fatal in 25-50% of patients.