Management of Patients Who Have Undergone Hepatic Artery Chemoembolization

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Hepatic artery chemoembolization (HACE) has been a prominent ablative treatment since the late 1980s. It is used to suppress intrahepatic tumor growth in an effort to palliate symptoms and perhaps prolong survival (Stuart, 2003). HACE is indicated for malignancies, including hepatocellular carcinoma (HCC), primary carcinoid tumors, and metastatic disease of the liver. This article primarily will discuss HCC.

HCC is the fifth most common cause of all malignancies and causes about one million deaths annually (Yu & Keeffe, 2003). HCC is the third most common cause of cancer deaths in men and the seventh most common cause of cancer deaths in women (Yu & Keeffe). Surgical liver resection is the only cure for HCC; however, few patients are eligible to undergo this procedure. Hepatic artery chemoembolization (HACE) is a technique that delivers high concentrations of chemotherapeutic agents and blocks the blood supply to the liver for prolonged periods of time. HACE has demonstrated an overall increase in survival. The HACE procedure, pre- and postprocedure complications, and the care required by patients with HCC are critical for oncology nurses to understand.

Liver Anatomy and Physiology

The liver is the largest internal organ in the body, located in the upper right quadrant of the abdominal cavity. The liver’s right lobe is larger than the left, and the organ is subdivided further into eight segments that reflect its underlying vascular complexity (Devita, Hellman, & Rosenberg, 2001) (see Figure 1).

The common hepatic artery carries oxygenated blood to the liver and holds 25% of the liver’s total blood volume (Rospond & Mills, 1995). The common hepatic artery enters into the porta hepatis medially to the common bile duct, branches off of the gastroduodenal artery to become the proper hepatic artery, and bifurcates into the right and left hepatic arteries. The liver, therefore, has a dual supply of oxygenated blood from the left and right hepatic arteries (Devita et al., 2001). Hepatic tumors are supplied primarily by the hepatic artery and respond better to chemoembolization than tumors supplied by the portal vein, which carries partially oxygenated blood and holds 75% of the liver’s blood supply. The portal vein is posterior to the head and neck of the pancreas and is located near the confluence of the splenic artery, the superior and inferior mesenteric arteries, and the coronary veins. The portal vein lies posteriorly in the porta hepatis and runs from the hepatoduodenal ligament to the hilus of the liver. Here, the vein divides to form right and left branches that supply the right and left hepatic lobes. Blood and waste products leave the liver via the hepatic veins and enter the inferior vena cava.

Risk Factors and Etiology of Hepatocellular Carcinoma

HCC is a disease of multifactorial etiology. The most important predisposing factor is cirrhosis (Desjardins, 2002). Other risk factors...
for HCC in the United States are cirrhosis caused by hepatitis C, hepatitis B, alcohol, or inherited metabolic diseases such as hemochromatosis and α1-antitrypsin deficiency (Davila, Petersena, Nelson, & El-Serag, 2003; Di Bisceglie et al., 2003), although alcoholic cirrhosis appears to be less strongly associated with cancer than are hepatitis C and hepatitis B (Monto & Wright, 2001). The lifetime risk of developing HCC in hepatitis B is estimated to be 50% in males and 20% in females (Monto & Wright). Hepatitis C has a lower global prevalence than hepatitis B, but hepatitis C causes the most cases of HCC in Europe, North America, and other economically developed regions (Monto & Wright). However, a significant number of cases in the United States lack an identifiable risk factor. The incidence of HCC is approximately three times greater among men than women (Davila et al.), among Asians than African Americans, and among African Americans than Caucasians (El-Serag & Mason, 1999).

**Clinical Presentation**

HCC is insidious in onset, and patients typically are asymptomatic during the early stages of the disease (Groen, 1999), but the majority are symptomatic at the time of diagnosis. Patients complain of vague symptoms, including fatigue, weight loss, a feeling of fullness, anorexia, right upper quadrant discomfort, or a dull ache in the epigastric area (Gogel et al., 2000; Groen; Lynes, 1993; Rilling & Drooz, 2002). The most common finding on physical examination is an enlarged, irregular, and nodular liver (Cahill & Braccia, 2004). Hepatic bruits, ascites, splenomegaly, jaundice, muscle wasting, and fever develop in advanced stages of HCC (Di Bisceglie, 2002). Anorexia and weight loss also are indicators of advanced disease (Van Cleave, Devine, & Odom-Ball, 1999).

**Diagnosis**

Serum alpha fetoprotein (AFP) and imaging studies are used to diagnose HCC (Cahill & Braccia, 2004). AFP values of more than 400 ng/ml or a progressive increase in AFP levels over time are highly suggestive of HCC (Yu & Keeffe, 2003). Imaging studies are essential in evaluating the location of the tumor and the extent of the disease (Braccia & Heffernan, 2003). Ultrasound, computed tomography, magnetic resonance imaging, and angiography are commonly used imaging techniques. Although the imaging modalities are similar, direct comparison is limited because the sensitivity of detection depends on the equipment, operator skill, and technique (Szklaruk, Silverman, & Charnsangavej, 2003). Imaging preference often is based on institutional recommendations (Cahill & Braccia).

**Hepatic Artery Chemoembolization Procedure**

Although chemotherapy generally is not effective in the treatment of HCC, HACE is an important part of treatment for this disease. Worldwide, HACE is the most common therapy used for unresectable liver tumors (Nakakura & Choti, 2000). Embolization may be done with or without chemotherapy. The addition of chemotherapy is called chemoembolization and combines intra-arterial delivery of chemotherapy with embolization of the hepatic artery. This technique delivers high-dose chemotherapeutic agents (often doxorubicin hydrochloride, mitomycin, and Platinol-AQ [Bristol-Myers Squibb Company, Princeton, NJ]) to the tumor, cutting off the blood flow with an emulsion of Lipiodol® (Guerbet Group, Cedex, France) and an embolizing agent such as gelatin sponge particles (Nakakura & Choti). Lipiodol consists of oil droplets containing the chemotherapy agent and carries the drug close to the tumor, exposing the tumor cells to high doses of the agent for prolonged periods of time (Groen, 1999). Doses are based on hepatic function, not body surface area, resulting in higher drug concentrations within the tumor tissue and prolonged exposure to chemotherapy. For instance, hepatic drug exposure has been estimated to be double for doxorubicin, sevenfold for cisplatin, and eight times greater for mitomycin when delivered intrahepatically rather than via IV (Ensinger & Gyves, 1983). This increased exposure enhances tumor necrosis (Rospond & Mills, 1995). The duration of HACE’s effectiveness varies because of the rapid development within the liver of collateral vessels that feed the tumor.

The actual procedure requires placement of a catheter in the femoral artery that is threaded into the hepatic artery. An angiogram is performed to verify the patency of the portal vein system before occluding the hepatic artery. An experienced angiography team must perform the procedure because of the difficulty and hazards associated with the technique (Stuart, 2003). Because of recent technologic advances, superselective HACE may be performed by threading the catheter into the smaller branches of the hepatic artery, ensuring the patency of the main hepatic artery. Once the catheter is in place, the interventional radiologist injects the chemotherapy combination. The chemotherapy and embolic agents are injected until blood flow to the artery supplying the tumor ceases. The use of embolization material minimizes the collateralization of blood flow by creating a distal vascular blockade, which prevents blood flow to the liver and permits subsequent chemoembolization treatment (Rospond & Mills, 1995). The chemotherapy agent undergoes degradation and resorption into the hepatic circulation, allowing vessels to reopen within 48–72 hours (Rospond & Mills).

HACE is indicated for use in patients with unresectable HCC, without extrahepatic disease, or for pain control and a bridge for liver transplantation. Contraindications to HACE include advanced liver disease, active gastrointestinal bleeding, encephalopathy, cirrhosis, and tumors invading the portal vein (Llovet et al., 2002). Advanced liver disease is measured by the Child-Pugh classification system, which determines the degree of hepatic dysfunction (Angermayr et al., 2003; Jievatlas, Stoskuviene, Petrenkiene, Barauskas, & Lundzius, 2004) (see Table 1). Child-Pugh classification class C is a contraindication to HACE. Factors associated with extended survival of 8–10 months include homogenous uptake of the chemotherapeutic agent in the tumor, hypervascularity of the lesions, an Eastern Cooperative Oncology Group performance status of 0 or 1, and serum alkaline phosphate and lactate dehydrogenase levels less than three times normal (Sullivan, 2002).
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The effect of HACE on survival has been difficult to measure because of the challenges in randomizing patients without reasonable alternatives. Clinical investigations in HCC have been impaired by limited sample size, uncertainties regarding optimal delivery of treatment, and the heterogeneity of patients (Reidy & Schwartz, 2004). Despite initial reports suggesting limited benefits of HACE, more recently published, well-conducted studies demonstrate a survival benefit. According to these studies, HACE likely confers a benefit greater than that associated with embolization without chemical agents (Reidy & Schwartz). However, at least with HCC, studies have shown a prolonging of life in several selected populations when compared with supportive care (Stuart, 2003).

Studies have shown tumor response rates of 50%–80% with HACE compared to a low (10%–20%) overall response rate with systemic chemotherapy (Groen, 1999). Llovet et al. (2002) demonstrated an improvement in overall survival with HACE when the researchers assessed the survival benefits of regularly repeated arterial embolization (gelatin sponge without chemotherapy) or chemoembolization (gelatin sponge plus doxorubicin) compared with conservative treatment. The rates of survival at one, two, and three years were 75%, 50%, and 29% for the embolization group; 82%, 63%, and 29% for the chemoembolization group; and 63%, 27%, and 17% for the control group. Mean survival was 25.3 months for the embolization group, 28.7 months for the chemoembolization group, and 17.9 months for the control group (p = 0.009) (Llovet et al.). Zhang et al. (2000) found survival rates at five years were 56.8% with HACE or embolization. The median survival length and rates at five years were 3.1 years and 25% in the embolization group compared with 2.5 years and 18% in the HACE group (Ikeda et al.).

In the author’s experience, HACE allows for palliation of symptoms, a reduction in pain, and conversion of unresectable tumors into resectable ones. The procedure has been shown to reduce systemic toxicity and increase local effects of chemotherapy, thereby improving therapeutic results. However, a perceived benefit for survival has not been substantiated in randomized trials, presumably because HACE’s anticancer effect is offset by its adverse effects on liver function (Qian, Feng, & Vogl, 2003). In addition, HACE may be needed every six weeks, depending on the recanalization of the hepatic artery. Patients who undergo three or more HACE procedures may receive the most clinical benefit (Tellez et al., 1998) (see Figure 2).

Complications

Adverse effects of HACE can range from nausea to cerebral hemorrhage. Severe complications occur in less than 1% of patients but include intraperitoneal hemorrhage, tumor rupture and hemorrhage, liver failure, cerebral hemorrhage, liver abscess, cholecystitis, and tumor lysis syndrome (Rospond & Mills, 1995). Patients also may develop edema, ascites, encephalopathy, renal dysfunction, infection, and anorexia. The most common adverse effects are nausea, vomiting, fever, and increased liver function tests (LFTs); increased LFTs indicate lysis of hepatocytes and the release of intracellular contents, signifying hepatic cellular death (Lynes, 1993).

LFTs measure levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (formerly known as serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase), enzymes found in liver cells that are sensitive indicators for hepatocellular injury (Desai

**Clinical Study Results**

**Complications**
& Isa-Pratt, 2002; Limdi & Hyde, 2003). These LFTs monitor different functions of the liver, such as the excretion of anions (bilirubin), the maintenance of hepatocellular integrity (transaminases), the production and transportation of bile (bilirubin and alkaline phosphatase), and the synthesis of protein (albumin) (Limdi & Hyde). Many factors, including gender, exercise, race, and body mass index, may influence these enzymes. The degree of elevation can provide clues to the etiology of the liver disease. In most types of liver disease, ALT exceeds AST except for patients with alcoholic liver disease (Desai & Isa-Pratt).

Other tests to be monitored include albumin. Hypoalbuminemia is common in patients with liver disease. However, albumin levels are not sensitive or specific to liver disease but are dependant on a number of other factors, such as nutritional status, presence of catabolism, hormonal factors, and urinary and gastrointestinal losses (Limdi & Hyde, 2003). Bilirubin levels and ammonia also should be monitored closely. The liver detoxifies ammonia by converting it into urea. Although the role of ammonia in the development of encephalopathy is unclear, the accumulation of unmetabolized ammonia from poor liver function and portosystemic shunting is important in the pathogenesis of hepatic encephalopathy (Riordan & Williams, 1997). Ammonia levels are used to diagnose hepatic encephalopathy (Desai & Isa-Pratt, 2002); however, normal ammonia levels do not exclude patients from having hepatic encephalopathy. A clinical diagnosis should be based on the presence of neurologic signs and symptoms.

**Nursing Implications**

Patients who are hospitalized for HACE may have many questions, concerns, and fears. As educators, nurses provide information about the HACE procedure, side effects, medications, and tumor response (see HACE Patient Information Sheet). As advocates, oncology nurses supply infor-
mation regarding end-of-life issues, social support systems, and pain and symptom management. Patients and families need the holistic and compassionate care that oncology nurses can give.

**Preprocedure Nursing Care**

During the preprocedure period, oncology nurses must provide support, reassurance, education, and protection from infection while monitoring the patient’s physiologic and psychological status. A thorough patient assessment is important in planning procedural and follow-up care. Risk factors that should be noted in the patient’s history include bleeding problems, infection, circulatory problems, tumor burden, and pain. The physical examination should include evaluation for underlying liver dysfunction such as jaundice, ascites, peripheral edema, spider nevi, palmar erythema, and weight loss. Assessment of the patient’s pedal pulses prior to the procedure is essential because HACE is performed via the femoral artery. The abdomen should be examined to determine hepatic size, presence of masses, tenderness,

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**Hepatic Artery Chemoembolization Patient Information Sheet**

**What is hepatic artery chemoembolization?** Hepatic artery chemoembolization is a treatment to help control the cancer in your liver. This technique delivers three chemotherapy agents directly into the blood vessels that feed the tumor in the liver.

**How does hepatic artery chemoembolization work?** The chemotherapy kills tumor cells, while sponge particles block the artery and decrease blood flow and oxygen to the tumor, causing the tumor to shrink.

**What are the side effects of this treatment?** Many side effects are possible. Most people experience some pain, fatigue, fever, and loss of appetite. Patients generally are hospitalized for two to three days, but you can feel tired and have other side effects for as long as four weeks after the procedure.

Side effects from chemotherapy may include nausea, hair loss, anemia, diarrhea, a decrease in white blood cells (which are responsible for fighting infection), and a decrease in platelet count (platelets are responsible for clotting). Cutting off the blood supply to the tumor may cause abdominal pain, fever, bloating, fluid accumulation in the abdomen, pneumonia, damage to the gallbladder and kidneys, and worsened liver function.

**What are the risks associated with this procedure?** The overall risk of serious complications is related to the patient’s overall well-being and the underlying health condition of the liver. People with severe liver diseases such as cirrhosis, jaundice, rapidly growing tumor, and blockage of the portal vein are at higher risk for complications.

**What should you do before the procedure?** Do not eat or drink anything after midnight, review all medications with your healthcare provider and take as instructed the morning of the procedure, and do not take aspirin for at least one week prior to the procedure.

**What should you expect while you are in the hospital?** You will arrive at the hospital early in the morning. Once you are admitted, you will have an IV line placed in your arm, allowing fluids to drip into your body. Adequate hydration is needed to protect your kidney function. The fluid will consist of antibiotics and medication needed prior to your procedure. You will receive medication to prevent nausea and pain. You will be transported to the interventional radiology department for the procedure. Under light sedation, using x-ray guidance, a small catheter will be inserted into the skin in the groin. The radiologist will thread the catheter up into the artery that feeds the tumor. Once the catheter is in place, the radiologist will inject a combination of chemotherapy drugs and particles as small as grains of sand that help block the blood flow to the tumor. Once the procedure is completed, you will be sent back to your room, where you will lie flat for six hours.

**What can you expect following hepatic artery embolization?** Patients most often experience fatigue for about three to four weeks after the procedure. Your appetite may be poor, and you may lose a significant amount of weight. You may have a fever for one to two weeks; however, if it increases to more than 100.5°F, contact your healthcare provider. The pain associated with this procedure improves during the first week but may require narcotics for longer periods. You also may experience slight hair loss. In general, these symptoms are normal throughout recuperation. During the first month following the procedure, be sure to follow up with your physician.

**When should you call your physician?** If you develop severe pain or a fever greater than 100.5°F, call your physician right away.

**Will this procedure cure me?** This procedure is not a cure but a treatment that may improve your symptoms and overall survival.

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### TABLE 2. NURSING CARE FOR PATIENTS WHO HAVE UNDERGONE HEPATIC ARTERY CHEMOEMBOLIZATION

<table>
<thead>
<tr>
<th>NURSING ACTIONS</th>
<th>NONPHARMACOLOGIC MANAGEMENT</th>
<th>PHARMACOLOGIC MANAGEMENT</th>
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<tbody>
<tr>
<td>Monitor vital signs and puncture site.</td>
<td>Monitor every 15 minutes for one to two hours, every 30 minutes for two hours, every hour for four hours, and every four hours for 24 hours. Call physician if temperature is greater than 100.5°F, pulse is less than 50 or greater than 120 beats per minute, or blood pressure is less than 80/50 or greater than 140/90. Assess site for bleeding or hematoma with vital signs as above. Apply pressure with a sandbag for six hours after sheath removal.</td>
<td>For bradycardia, give IV bolus of normal saline and atropine 0.5–1.0 mg orally every four hours as needed, or 0.5–2.0 mg intravenously every two hours as needed. For fever, obtain blood cultures, give IV fluids, and administer antipyretics and IV antibiotics.</td>
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<tr>
<td>Monitor pedal pulses.</td>
<td>Assess with vital signs as above. Pulses are documented as follows: 4+ (bounding), 3+ (normal), 2+ (weak), 1+ (barely palpable), 0 (absent), or D (audible by Doppler). Obtain baseline prior to procedure. Assess for numbness, pallor, or pain. Notify physician of any change.</td>
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<tr>
<td>Monitor laboratory values.</td>
<td>Notify physician if hemoglobin is less than 8.0 g/dl, platelet count is less than 50 k/uL, or potassium is less than 3.4 or greater than 5.0 mEq/dL.</td>
<td>Patient may need a blood or platelet transfusion. For low potassium, give potassium chloride 20–100 mEq orally in divided doses or by IV 10 mEq per hour. For elevated potassium, give sodium polystyrene 15 g orally one to four times per day.</td>
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<tr>
<td>Monitor mental status.</td>
<td>Assess level of consciousness and mental status with vital signs. For acute encephalopathy, decrease stimuli and obtain glucose, renal, and liver functions and blood gases. Monitor ammonia level if change in mental status is noted. Clinical diagnosis of encephalopathy cannot be made on ammonia levels alone.</td>
<td>Give lactulose 15–30 ml orally each day for elevated ammonia levels. Lactulose is used in hepatic encephalopathy to lower serum ammonia levels. The liver is the only organ that detoxifies ammonia by converting it into urea.</td>
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<tr>
<td>Monitor fluid status.</td>
<td>Assess vital signs as above and intake and output every four hours. Notify physician for urine output less than 30 cc per hour. Assess for nausea and vomiting. If present, use ginger, instruct patients to eat food at room temperature, and teach relaxation techniques.</td>
<td>Give IV fluids and antiemetics. For acute nausea, administer dexamethasone 8 mg orally twice a day for three days or 8 mg orally two times a day for two days and then 4 mg orally twice a day for two days, prochlorperazine IV 10–25 mg every three to four hours or 5–10 mg every four to six hours orally or 25 mg per rectum, or lorazepam IV 1–2 mg/m² not exceeding 3 mg or orally 0.5–2.0 mg. For latent nausea, give ondansetron hydrochloride 12–24 mg orally each day for two to three days, granisetron hydrochloride 2 mg orally each day for two to three days, or dolasetron mesylate 100 mg orally each day for two to three days.</td>
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<tr>
<td>Monitor elimination pattern.</td>
<td>For constipation, increase dietary fiber and maintain adequate fluid intake. For diarrhea, suggest a low-residue, bland diet consisting of bananas to replace nutrients; rice, which is easily digested; applesauce, which has sugars for energy; and toast, which is tolerated easily. Patients should eat small, frequent meals at room temperature, increase food intake, increase oral fluids (six to eight glasses per day), and avoid caffeine, alcohol, and milk, as well as greasy, fatty, and fried foods.</td>
<td>For constipation, consider that bulk laxatives increase the size and weight of stool (e.g., psyllium), saline laxatives (e.g., magnesium citrate) draw water into the gut and are intended for acute use, osmotic laxatives (e.g., lactulose, sorbitol) increase osmotic pressure, detergent laxatives (e.g., docosate) act directly on the colon to reduce surface tension, and stimulant laxatives (e.g., bisacodyl, senna) induce motility. When all else fails, perform an enema. For diarrhea, administer loperamide hydrochloride 4 mg orally, initially, followed by 2 mg after each stool (as many as eight pills per day), or atropine sulfate or diphenoxylate hydrochloride 2–4 mg orally three to four times per day up to eight pills per day.</td>
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<tr>
<td>Monitor discomfort level.</td>
<td>Evaluate vital signs to assess pain intensity, and assess regularly with a pain scale. Techniques such as relaxation exercises, heat or cold packs, or distraction may be helpful. Evaluate response to pain management.</td>
<td>Give opioid analgesics: morphine sulfate (10–30 mg orally every four hours as needed, 5–20 mg or 70 kg subcutaneously every four hours as needed, or 2.5–15 mg or 70 kg in 4–5 ml of water via IV over five minutes as needed) or hydromorphone (1–2 mg by IV or subcutaneously every four to six hours as needed or 2–4 mg orally every four to six hours as needed). Give nonopioid analgesics: hydrocodone bitartrate or acetaminophen (dose varies according to product and strength), ibuprofen (400 mg orally every four to six hours as needed), or naproxen (500 mg orally initially and then 250 mg orally every six to eight hours, not exceeding 1,250 mg per day). Consult the pain service.</td>
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**Note:** Based on information from Lynch, 2002; Spratto & Woods, 2003; Yasko, 2001.
and splenomegaly (Van Cleave et al., 1999). The patient’s baseline weight should be obtained prior to the procedure. Hydration and IV antibiotics administered prior to the procedure have been shown to decrease the risk of potential toxicities. Broad-spectrum antibiotics are given to prevent infections in the bowel and biliary system. Laboratory tests must include a complete blood count, LFTs, renal function tests, prothrombin time, and international normalized ratio. Patients should have a platelet count greater than 100,000 K/dl, bilirubin less than 2.0 mg/dl, and an international normalized ratio of less than 1.5 prior to the chemoembolization procedure (Tellez et al., 1998).

Postprocedure Nursing Care

Postprocedure complications are divided into acute and delayed side effects. Patients often are hospitalized for one to three days following the procedure so that acute side effects can be controlled. These side effects include pain, nausea, vomiting, and bleeding from the puncture site. Delayed side effects include pain, nausea, fatigue, and diarrhea and may occur after the patient has been discharged from the hospital. Postembolization syndrome (delayed side effects) can last for weeks.

Acute side effects: Nursing care immediately postprocedure includes careful monitoring of vital signs and the puncture site, pain management, hydration, and administering antiemetics and IV antibiotics (see Table 2). Patients will need to lay flat with a sandbag over the puncture site for at least six hours after the sheath has been removed. Percutaneous access of the femoral artery, sheath removal, or manual pressure can stimulate the vagus nerve via the parasympathetic nervous system and may result in bradycardia. The majority of patients can ambulate six hours after the procedure. Hypotension and tachycardia may indicate signs of bleeding. The patient’s pulse must be documented; however, a change in quality is more important than the pulse rate. The patient’s mental status also should be monitored.

Postprocedure pain may be severe. The pain associated with HACE results from irritation of the liver capsule and necrosis of the tumor (Cook, 1995). Chemoembolization results in the development of right upper quadrant pain in nearly all patients (Tellez et al., 1998). Pain should be managed with oral and parenteral opioid analgesics, and a continuous opioid infusion may be indicated for severe pain. The pain service may need to be consulted for additional assistance in managing discomfort. Patients should receive IV hydration to promote adequate fluid balance and renal function and flush out chemotherapy agents. This is particularly important if Platinol-AQ is used.

Chemoembolization is moderately to highly emetogenic; therefore, acute nausea and vomiting are treated with antiemetics such as prochlorperazine (10–25 mg orally or by IV every three to four hours as needed), lorazepam (0.5–2.0 mg orally or 1–2 mg by IV not exceeding 3 mg), or 5-HT3 antagonists (Lynch, 2002). Patients’ laboratory values, including complete blood counts, electrolytes, and LFTs, are checked to monitor for the development of adverse effects such as liver failure, thrombocytopenia, and hypokalemia. LFT levels increase postprocedure but should return to baseline within 3–10 days.

Delayed side effects: Delayed side effects include nausea, infection, diarrhea, and pain. When discharged, patients should be given pain medication, 5-HT3 antagonist antiemetics, antibiotics, and antidiarrheal medications. Some common 5-HT3 antagonists are ondansetron, hydrochlorofine, granisetron, hydrochlorofine, and dolasetron mesylate. Patients may receive antibiotics such as metronidazole (500 mg by mouth twice daily for five days) and ciprofloxacin (750 mg by mouth three times per day for five days) to prevent bacterial infections in the bowel and biliary system. Patients should be instructed to monitor their temperature at home and take antidiarrheal medication at the first sign of diarrhea (loperamide hydrochloride 4 mg orally, initially, followed by 2 mg after each loose stool for a maximum of eight tablets per day). Patients should call an oncologist or oncology nurse if these symptoms are not relieved by these medications. Fatigue is common and often a dose-limiting toxicity from HACE.

Because very little chemotherapy is absorbed systemically, delayed nausea, vomiting, alopecia, and neutropenia rarely occur (Cook, 1995). Two potentially severe side effects are related to direct liver inflammation and include pain as well as rapid fluid shifts resulting in hypotension. Hypotension may occur because of fluid leaking from the blood vessels in the peritoneal cavity, also known as third spacing. If not diagnosed quickly and treated properly, patients may go into shock and develop acute renal failure.

Psychosocial Support

The diagnosis of cancer is a life-altering experience for patients. This battle is not just physical but emotional. Once diagnosed with cancer, patients may have memories of others with cancer, conjuring images of pain and death. Unlike patients diagnosed with other types of cancers, patients with HCC may experience feelings of guilt and self-blame. They may feel that their lifestyle choices caused a disease that has no cure. In these instances, nurses should consider referring patients to a social worker or psychologist.

The potential curative treatment for HCC is surgery by resection or liver transplant. Healthcare providers need to stress that ablative treatments are for palliation and are used to alleviate symptoms of the disease rather than provide a cure (Lynes, 1993). Patients and families may have difficulty accepting the absence of a cure. Oncology nurses must be proactive and discuss the plan of care with patients and their families who need reassurance and support.

Conclusion

HCC is very difficult to treat and even more difficult to cure. As the incidence of HCC continues to increase in the United States, oncology nurses must be aware of HACE. Patients with HCC who undergo HACE require skilled oncology nursing care that addresses their specific physical and psychosocial concerns. Nurses must be knowledgeable about HACE and its adverse effects, as well as the care of these patients. Research and evaluation of new treatment modalities are needed to determine effects on patient outcomes and quality of life. Oncology nurses are uniquely able to contribute to these studies.

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References


Rapid Recap

Management of Patients Who Have Undergone Hepatic Artery Chemoembolization

- As the prevalence of hepatocellular carcinoma continues to rise in the United States, oncology nurses will be faced with increased exposure to liver-directed treatments such as hepatic artery chemoembolization (HACE) and will play an important role in the care of patients who have undergone this procedure.
- HACE delivers high-dose chemotherapy directly into the liver.
- HACE allows for palliation of symptoms, reduced pain, and conversion of an unresectable tumor into a resectable one.
- During the preprocedure period, oncology nurses provide support, reassurance, education, and protection from infection while monitoring the patients’ physiologic and psychological status.
- Postprocedure complications, either acute or delayed, include pain, nausea, vomiting, bleeding, fatigue, and diarrhea.