PALLIATIVE CARE NETWORK OF WISCONSIN

FAST FACTS AND CONCEPTS #176
EVALUATION OF MALIGNANT ASCITES

Karen LeBlanc and Robert Arnold MD

Background Malignant ascites is the accumulation of abdominal fluid due to the direct effects of cancer. This *Fast Fact* reviews the causes and diagnosis of malignant ascites. *Fast Fact* #177 will review its treatment.

Pathophysiology The pathophysiology of malignant ascites is incompletely understood. Contributing mechanisms include tumor-related obstruction of lymphatic drainage, increased vascular permeability, over-activation of the renin-angiotensin-aldosterone system, neoplastic fluid production, and production of metalloproteinases that degrade the extracellular matrix. Portal venous compression can also occur from metastatic invasion of the liver, leading to peritoneal fluid accumulation.

Natural History The most common cancers associated with ascites are adenocarcinomas of the ovary, breast, colon, stomach and pancreas. Median survival after diagnosis of malignant ascites is in the range of 1-4 months; survival is apt to be longer for ovarian and breast cancers if systemic anti-cancer treatments are available.

Presentation and Diagnostics Symptoms include abdominal distension, nausea, vomiting, early satiety, dyspnea, lower extremity edema, weight gain, and reduced mobility. Physical exam findings may include abdominal distention, bulging flanks, shifting dullness, and a fluid wave. Plain abdominal x-rays are not specific, but may show a hazy or a "ground glass" appearance. Ultrasound or CT scanning can confirm the presence of ascites and also demonstrate if the fluid is loculated in discrete areas of the peritoneal cavity.

There are many potential causes of ascites in the cancer patient: peritoneal carcinomatosis, malignant obstruction of draining lymphatics, portal vein thrombosis, elevated portal venous pressure from cirrhosis, congestive heart failure, constrictive pericarditis, nephrotic syndrome, and peritoneal infections.

Depending on the clinical presentation and expected survival, a diagnostic evaluation is usually indicated as it will impact both prognosis and treatment approach. Key tests include the serum albumin and protein level and a simultaneous diagnostic paracentesis, checking ascitic fluid white blood cell count, albumin, protein, and cytology.

Classification The old classification of exudative versus transudative ascites has been updated through the use of the serum-ascites albumin gradient (SAAG).

SAAG = (the serum albumin concentration) – (ascitic fluid albumin concentration).

A SAAG $\geq$ 1.1 g/dl indicates ascites due to, at least in part, increased portal pressures, with an accuracy of 97%. This is most commonly seen in patients with cirrhosis, hepatic congestion, CHF, or portal vein thrombosis.

A SAAG < 1.1 g/dl indicates no portal hypertension, with an accuracy of 97%; most commonly seen in peritoneal carcinomatosis, an infectious process of the peritoneum, nephrotic syndrome, or malnutrition/ hypoalbuminemia.

Cytological evaluation is approximately 97% sensitive in cases of peritoneal carcinomatosis, but is not helpful in the detection of other types of malignant ascites due to massive hepatic metastasis or malignant obstruction of lymph vessels.

References

1. Thomas J, von Gunten CF. Diagnosis and Management of Ascites. In: Berger AM, Von Roenn J, Schuster J, eds. *Principles and Practice of Palliative Care and Supportive Oncology*. 3rd edition. Philadelphia, PA: Lippincott, Williams & Wilkins; 2006.
2. Adam RA, Adam YG. Malignant ascites: past, present, and future. *J Am Coll Surg.* 2004; 198:999-1011.

3. Spratt JS, Edwards M, Kubota T, et al. Peritoneal carcinomatosis: anatomy, physiology, diagnosis, management. *Current Problems in Cancer.* 1986; 10:553-584.
4. Becker G. Galandi D. Blum HE. Malignant ascites: systematic review and guideline for treatment. *Eu J Cancer.* 2006; 42:589-97.
5. Aslam N, Marino CR. Malignant ascites: new concepts in pathophysiology, diagnosis, and management. *Arch Int Med.* 2001; 161:2733-7.

Version History: Version copy-edited in May 2009; then again July 2015.