

Malignant Ascites

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Ascitis is common entity in adults. Increased vascular permeability, decrease plasma oncotic pressure & lymphatic obstructions are common causes.

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Ascites occurs due to altered vascular permeability & obstructed lymphatic drainage. Malignant ascites accounts for approximately 10% of all cases of ascites [1]. About one-third of all patients with malignancies will develop ascites. Malignant ascites is a sign of peritoneal carcinomatosis. Common tumors causing carcinomatosis include secondary peritoneal surface malignancies from abdominal tumors like ovarian, colorectal, pancreatic, uterine [2]; & extra-abdominal tumors originating from lymphoma, lung, breast, prostate & digestive endocrine tumours such as carcinoid tumours [3]. In nearly 50% of cases of peritoneal carcinomatosis, ascites is the first detected sign of intra-abdominal malignancy [4].

Patients with malignant ascites usually present with anorexia, nausea, respiratory compromise, immobility, abdominal bloating, heaviness & weight gain despite muscle wasting. Diffuse edema occurs due to loss of proteins & electrolyte disorders. Accumulation of abdominal fluid facilitates secondary sepsis. Large veins on flanks & back occur due to blockage of inferior vena cava by malignancy. Lymphadenopathy (e.g. Sister Mary Joseph's nodule, Virchow's node for upper abdominal malignancies) may be noted. Radiographic signs include a ground-glass pattern with centralization of intestines & abdominal contents. Abdominal ultrasonography is a sensitive & specific method for detecting & quantifying ascites & also permits delineation of areas of loculation. Computed Tomography scan demonstrate masses, mesenteric stranding, omental studding & diffuse carcinomatosis.

Abdominal paracentesis with ascitic fluid analysis & laparoscopic tissue sampling can diagnose malignant etiology of ascites. Ascitic fluid in malignant ascites is mostly exudative (75%) & often bloody or serosanguinous with a white cell count of > 250

cells/cubic mm with LDH content > 50% of serum values. Tumor markers like CEA, CA-125 & a fetoprotein help in identifying primary tumor causing malignant ascites. However biochemical tests like fibronectin, cholesterol, lactate dehydrogenase, sialic acid, telomerase activity & proteases are not reliable in differentiating between malignant & benign ascites.

Management of malignant ascites includes medical & surgical measures. Medical therapy of malignant ascites includes paracentesis & diuretics. Paracentesis provides immediate relief in nearly 90% of patients. Sonographic guidance is required in loculated ascites. For those requiring frequent paracentesis, external drainage catheter placed through the abdominal wall is recommended. Diuretics is an effective therapy in initial phase of malignancy achieving symptomatic relief in nearly 40% of cases [5]. However its efficacy declines with tumor progression. However those with evidence of portal hypertension (SAAG > 1.1) respond better to diuretics.

Intraperitoneal chemotherapy, targeted therapy, immunotherapy & radioisotopes are promising medical options. Recently intraperitoneal catumaxomab, a trifunctional monoclonal CD3- and epithelial cell adhesion molecule (EpCAM)-specific antibody has shown promising results in malignant ascites associated with ovarian cancer. Intraperitoneal chemotherapeutic agents like bleomycin, cisplatin, 5-fluorouracil, thiotepa, mitomycin, paclitaxel, mitoxantrone, topotecan, taxanes & doxorubicin have been studied with varied benefits. Direct intraperitoneal chemotherapy rather than systemic chemotherapy is implemented as it achieves higher tissue concentrations without systemic toxicity. Sclerosing agents like bleomycin & talc have also been studied.

Bevacizumab is a recombinant humanized monoclonal antibody to VEGF composed of human IgG1 framework regions & antigen-binding complementarity-determining regions from a murine antibody that blocks binding of human VEGF to its receptors [6]. It neutralizes biologic activity of tumor-associated VEGF, thereby reduces tumour vascularization & inhibits growth of malignancy. Newer medical treatments currently under investigation include: matrix metalloproteinase inhibitors such as Batimastat & marimastat [7], immunotherapeutic agents such as interferon, tumor necrosis factor, *Corynebacterium parvum* & Streptococcal preparation OK-432, radioimmunotherapy utilizing monoclonal antibody therapy & Aflibercept (Zaltrap), a potent angiogenesis inhibitor fusion protein[8].

Surgical measures include shunts & tumor debulking. Peritoneal-venous shunts (PVS) are primarily designed to channel peritoneal fluid & proteins from ascites into the circulation via superior vena cava. Best response is noted in ascites associated with ovarian & breast cancers. It is recommended as a palliative measure as it has a high occlusion rate upto 24%. Transjugular Intrahepatic Portosystemic Shunt (TIPS) is a shunt between portal vein & hepatic vein, designed to reduce portal hypertension & improve sodium balance which is useful in those with increased portal pressures. Permanent PleurX catheter implantation is an effective procedure in those patients with terminal cancer disease [9].

Other Palliative measures include Laparoscopy & hyperthermic intraperitoneal chemotherapy (HIPEC). HIPEC is done along with tumor debulking or cytoreductive surgery (CRS). Malignancy-related ascites signifies advanced disease in most cases and is associated with dismal prognosis. Exceptions are ovarian carcinoma & lymphoma, which respond to debulking surgery & chemotherapy, respectively. Early detection & prompt management improves the outcome in malignant ascites.

Ray et al discussed about ascitis that most common cause of malignant ascites to be HCC. So to decrease the

burden of HCC in community setting we need to implement strategies [10].

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