Clinicopathological analyses of meningitis carcinomatosa of breast carcinoma: report of three cases

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Background and Purpose

Meningitis carcinomatosa is a rare, but serous complication of breast cancer and very difficult to treat. Here we report clinicopathological analyses of three patients of meningitis carcinomatosa, which we experienced in our hospital (Mito Medical Center) during 1997-2009. Pathological reviewing according to the current classification was also performed.

Patients' Report

Case 1:

A 68-year-old Japanese woman had a diagnosis of breast carcinoma of the clinical stage IIIA (cT2N2M0). She underwent right total mastectomy with axillary lymph node dissection. The pathological diagnosis was invasive ductal carcinoma, grade 3, with ER(-), PgR(-), and HER2 (-) (0). Twenty-five of 30 lymph nodes dissected contained carcinoma metastasis. She received cyclophosphamide, methotrexate and fluorouracil as a postoperative chemotherapy. Nine months after surgery, she presented with severe headaches, and a meningeal enhancement of the brain by MRI and a hydrocephalia on CT. Cerebrospinal fluid (CSF) cytology was positive for malignant cells. She died eight weeks after the onset of meningitis. Postmortem autopsy revealed infiltration of atypical, isolated carcinoma cells in the subarachnoid space of brain.

Case 2:

A 47-year-old Japanese woman noticed a palpable mass in her left axilla. On admission, a tumor, 1.5cm in the largest dimension, was noted in the lateral and superior part of the left breast. She diagnosed as breast carcinoma of the clinical stage IIIC (cT1N3M0) and received epirubicin hydrochloride and paclitaxel as preoperative chemotherapy. After the treatment, she underwent left partial mastectomy and axillary lymph node dissection. The histopathological diagnosis was status post chemotherapy of invasive carcinoma, with ER(+), PgR(-), and HER2 (-)(0). The presence of lobular neoplasia in situ suggested that the tumor was originally invasive lobular carcinoma. Four of 17 lymph node dissected contained scar tissue with small number of tumor cells. As postoperative radiation, she received total 50 gray to the primary site. Eight months after surgery, she complained severe headache, sensory disturbance on her face and leg paralysis. The CSF cytology was positive for malignant cells. She died eight weeks after the onset of meningitis.

Case 3:

A 40-year old Japanese woman underwent right total mastectomy with axillary lymph node dissection for breast carcinoma of the clinical staging as stage IIIB (cT4N2M0). The histopathological diagnosis was invasive lobular carcinoma, solid and classical type, with ER(+), PgR(+), and HER2(-)(0). Nine of 14 lymph nodes dissected contained carcinoma metastasis. She received cyclophosphamide, epirubicin hydrochloride and fluorouracil

as postoperative chemotherapy. Twenty-two months later she underwent second surgery for local recurrence. Twenty-four months after the primary surgery, she complained headache, confusion, impaired orientation, sensory disturbance on her face and leg paralysis. CSF cytology was positive for malignant cells. She had disturbance of consciousness for a few days. She received glucocorticoid administration, which markedly relieved the patient from the CNS symptoms. She remained free from CNS symptoms for three years after the onset of meningitis and died of peritonitis carcinomatosa.

Discussion

Breast carcinoma patients who develop meningitis carcinomatosa usually have fulminant courses and the prognosis is poor. The majority of cases of this complication are reported to be invasive lobular carcinoma or mixed invasive ductal and lobular carcinoma. As reported, our cases 1 and 2 showed an ominous course. However, the case 3 recovered from the CNS symptoms only after glucocorticoid therapy. This is an unexpected result, and much attention should be focused on this point. In all three cases, we found advanced axillary lymph node metastasis, and carcinoma cells were negative for E-cadherin expression. The absence or marked reduction of E-cadherin expression could be related to a reduced cell adherence, and this may facilitate dissemination into the subarachnoid space of CNS.