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CASE REPORT

Spontaneously Curing Anaplastic Carcinoma in the Lymph Node

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A well documented case of a spontaneously curing anaplastic carcinoma in lymph node is presented with a 16 year follow up. Reevaluation and detailed immunohistochemical examination confirmed the original diagnosis of anaplastic carcinoma. This is the first report of a spontaneously curing anaplastic carcinoma which raises the following questions: Was the tumor in the axillary lymph node a metastasis or a primary tumor? Does the anaplastic carcinoma demonstrate the same spontaneous regression characteristics as for example the neuroblastoma? (Pathology Oncology Research Vol 3, No 2, 139–141, 1997)

Key words: anaplastic carcinoma; spontaneous regression

Introduction

While it is well known that some highly malignant tumors may occasionally mature spontaneously like neuroblastoma,[3,4] spontaneous curing of anaplastic carcinoma, – to our best knowledge – has not been reported yet.

Spontaneous regression of the primary tumor having extended metastases is also a known phenomena, for example in cases of malignant melanomas[11] and seminomas.[5] We found this case interesting due to the very unusual self healing process of this highly malignant tumor.

Case Report

A 68 year old woman (born in 1913) was admitted to the surgery in December, 1981, for a tumor removal in the left axillary region. The tumor was noticed first by the patient five months earlier and until the removal enlarged to walnut size. It was not fixed to the surrounding tissues. The tumor was removed and routinely processed for histopathological examination. The diagnosis was *carcinoma metastaticum lymphoglandularum axillarium.*

The patient's past medical history included only hysterectomy for fibromyoma in 1958. In 1981 after the diagnosis of metastatic tumor she underwent a detailed medical checkup. The careful checkup included mammography, chest x-ray, stomach x-ray with barium enhancement, irrigoscopy, urography, and gynecological examination, all with negative results, except a small diverticulum of eosophagus and diverticulosis of the sigma. Abdominal ultrasound examination raised the suspicion of multiple liver metastasis. The laboratory values were in normal range. In spite of the diagnostic efforts, the primary tumor was not found. She had no subjective complaints. White hospitalized she had a superficial thrombophlebitis of cruris.

Other diseases in the later history: included hypertension, cholelithiasis, osteoporosis with scoliosis, mild arteriosclerosis. In 1993 a transitory ischemic attack happened with transient diplopy. In January, 1996 she had acute bronchitis after this a complete checkup followed, and the abdominal ultrasound examination failed to find the previously mentioned multiple liver metastasis. All other examinations were also negative.

The patient, now 83 years old, has no complaints or any sign of malignant disease.

During the time between 1981 and today she was followed by her general practitioner and was never hospitalized. Now she has the following medication: Brinaldix (clopamid) 5 mg every second day, Digoxin 0.125 mg daily, Corinfar (nifedipine) 10 mg/day, Nootropil 1200 (piracetam) 2 tablets/day and Lactulose, Fe, multivitamins and calcium for the geriatric problems.

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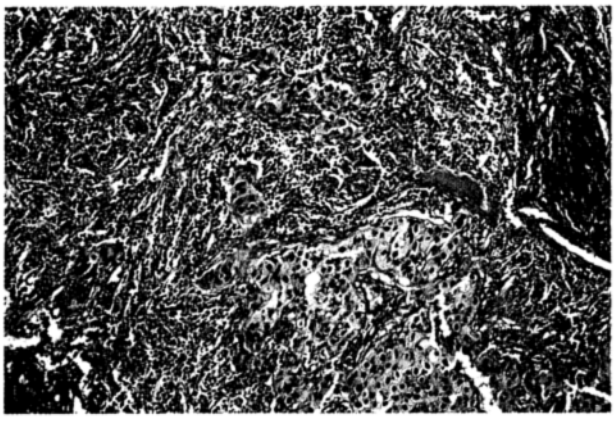

Figure 1. Lymph node, almost replaced by tumor cells forming nests, small groups and cords

Figure 2. Intravascular spread of the tumor in the capsular area

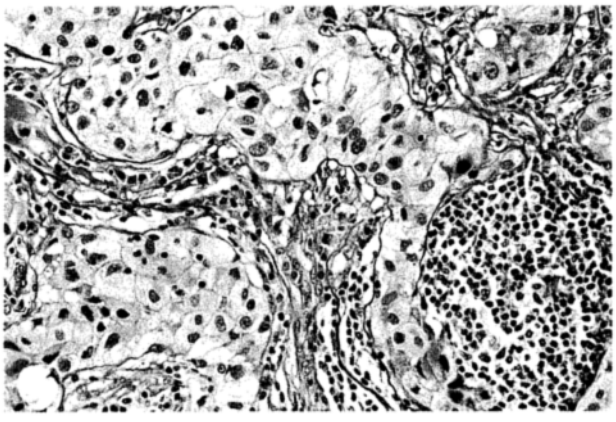

Figure 3. Tumor cells with abundant pale cytoplasm. Note abortive gland formation. Alcian-blue-PAS

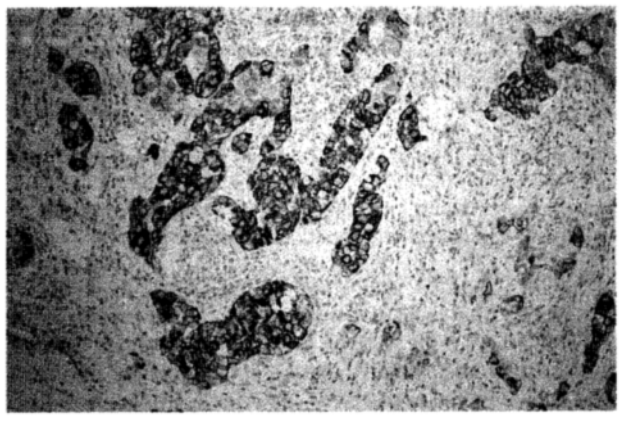

Figure 4. Intensive cytokeratin (AE-1) positivity of the tumor cells

Pathological Examination

The surgical specimens consisted of a 2 cm diameter and two 1 cm diameter greyish-white firm lymphoglands in adipose tissue. Routinely processed 5 μm thick sections were stained with haematoxylin and eosin and alcian blue PAS. For immunohistochemical examination a panel of monoclonal antibodies was used (avidin-biotin-peroxidase method): keratin low mol weight (AE-1), EMA (epithelial membrane antigen), S100 (monoclonal), HMB-45, oestrogen receptor (BioGenex); LCA, CD-30 (Ki-1) (Dako).

The lymphoglands are replaced by tumor tissue but small preserved lymphoid architecture is obvious. The malignant cells are arranged in nests, small groups and cords *(Fig.1)*. In a few places abortive gland formation can be observed. The lymph nodes have a thick capsule. In the small lymphatic vessels of the outer part of the capsule there are groups of anaplastic tumor cells (*Fig.2*). The tumor cells have abundant cytoplasm, which is pale-eosinophil, sometimes granulated and vacuolated. The nuclei show polymorphism and there is moderate mitotic activity (*Fig.3*). The tumor has mild desmoplasia. There is no suggestive feature for primary origin. In 1996 we took the original tissue blocks (15 years after of the original discovery of the tumor) and reexamined the tissue with up to date methods. This was necessary because Ki-1 large cell lymphoma which can mimic anaplastic carcinoma. The immunhistochemical study showed strong immunoreactivity of the tumor cells for low molecular weight cytokeratin and EMA (*Fig.4*). There was no immunoreactivity for LCA, S100, HMB-45, CD-30 (Ki-1) or oestrogen receptor. According to these tests there is no doubt about the identity of the tumor, which is an anaplastic carcinoma and where possibility of large cell lymphoma or melanoma malignum is excluded.

Discussion

The presence of epithelial cells in lymph node is usually the result of metastasis from a regional carcinoma. Their detection has great clinical importance. Far less often

epithelial inclusions may be present in lymph nodes.[1,2,12] Their origin is not well understood, though several theories have been proposed, including developmental heterotopia or metaplasia of local multipotent cells. These can cause differential diagnostic problems.

Small nodules of mammary ducts, cysts and myoepithelial cells have been observed in axillary nodes. They are usually located in the subcapsular area. Small groups of naevus cells can be present in the capsule and hilus of axillary nodes.[7,9]

The epithelial cells do not show cytological features of malignancy such as hyperchromatism, nuclear polymorphism and mitosis. There is no inflammatory or desmoplastic reaction.

Occasionally even malignant tumors enter a stage of dormancy, and in rare, but well-documented instances they may regress spontaneously.[3,4,5,6,8,10,11] Spontaneous regression is defined as complete disappearance of cancer that cannot be attributed to treatment. The explanation for the phenomenon is inconclusive.

This case is interesting because of the very unusual self healing process of a histologically "highly malignant" tumor. The tumor in the lymph node was either a metastasis or primary (originated from epithelial inclusion). In any case the lesion was cured probably by "nature" (spontaneously), since only a paramedical drug was given for a short period.

The other important question, is the frequency of this spontaneous healing process in the case of anaplastic carcinomas, on similar malignancies e.g. neuroblastoma, remains to be answered.

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