ure; only one (10%) patient had no comorbidity. There was no history of other malignancies. Six of 10 (60%) patients had family history of different malignancies. Mean age at menarche was 13 years. Simple mastectomy was performed in one (10%) patient, modified radical mastectomy in 3 (30%) and lumpectomy with sentinel lymph node dissection in 2 (20%). Histology showed infiltrative ductal carcinoma in 6 (60%), and tubular, lobular, apocrine, mucinous, and papillary carcinomas were diagnosed in 1 case each. Lymphovascular and perineural invasion were present in 4 (40%) patients. Tumor grade was II in 4 (57%) and III in 3 (43%) of 7 patients with known tumor grade. All of the patients were estrogen (ER) and progesterone receptor (PR) positive with HER2/neu negativity. Mean tumor size was 4.6 cm. Six (60%) of the patients had T2 disease, lymph node involvement was present in 4 (57%) of the 7 patients with known status and bone metastasis was present in only one (12.5%) patient at the time of diagnosis. Adjuvant chemotherapy was not given in 7 (87.5%) patients. All patients received hormonotherapy.

In conclusion, almost all of the patients had comorbidities. All of the patients were ER (+)/PR (+), but HER2 (-). T2 tumor size prevailed, half of the patients had lymph node involvement and only one had metastasis. Breast tumors tend to have less aggressive characteristics in elderly population [2, 3]. Radiotherapy and chemotherapy were not generally used in our study.

References

1. Evron E, Goldberg H, Kuzmin A et al. Breast cancer in octogenarians. Cancer 2006; 106: 1664-1668.
2. Kimmick G, Muss HB. Breast cancer in older patients. Semin Oncol 2004; 31: 243-248.
3. Szekely B, Madaras L, Szentmartoni G et al. Comparison of breast cancer in young and old women based on clinicopathological features. Magy Onkol 2010; 54: 19-26.

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A case of familial nasopharyngeal carcinoma: Is screening necessary for siblings?

Dear Editor,

Family history is known to increase the risk of nasopharyngeal carcinoma (NPC) [1]. However, the exact underlying pathogenetic mechanisms and clinical approach for screening and early detection of individuals at risk are still not clear. Herein we present a case with two affected siblings and briefly discuss the available literature.

A 35-year-old male patient presented with a right cervical mass in November 2009. Magnetic resonance imaging (MRI) of the nasopharynx demonstrated a soft tissue mass originating from the right lateral site of the nasopharynx, leading to the nasopharyngeal cavity with left submandibular lymphadenopathy. Punch biopsy of the nasopharynx revealed non-keratinizing NPC. He had no distant metastasis and was staged as stage IIB (cT2aN1M0). The patient was then scheduled for two courses of induction chemotherapy with docetaxel (75 mg/m^2, day 1, every 3 weeks), cisplatin (100 mg/m^2, day 1, every 3 weeks) and 5-fluorouracil (750 mg/m^2, days 1-5, every 3 weeks). After induction chemotherapy, chemoradiotherapy with cisplatin (35 mg/m^2 weekly) was initiated. Repeat MRI after treatment showed complete disease regression. In October 2011 the patient came out with a right cervical mass. 18-fluorodeoxyglucose positron emission tomography-computed tomography (^{18}FDG PET-CT) showed increased uptake in the posterior wall of the nasopharynx, in 2 left superior cervical lymph nodes, and a conglomerated right posterior cervical lymph node of 19 mm in diameter. He will now receive salvage treatment for the recurrent disease.

The patient was an ex-smoker and he had smoked about 10 pack-years till he quitted in 2009. He had no habitual alcohol consumption. His past medical history was unremarkable otherwise. Family history revealed that he had had two elder brothers with the same diagnosis. One of his brothers, who had a history of 10 pack-years of smoking, was diagnosed with NPC at the age of 35. He had received induction chemotherapy and irradiation and died of progressive disease 2 years after diagnosis. The other brother was diagnosed at the age of 32 and had no smoking history. He also had received induction chemotherapy and irradiation and died of progressive disease 3 years after diagnosis.

The etiology of NPC is multifactorial. Epstein-Barr virus (EBV) infection, genetic predisposition and environmental factors such as alcohol and tobacco consumption and high intake of preserved food are suspected in etiology. EBV is thought to be the primary etiologic agent in the pathogenesis of NPC. In endemic areas of NPC EBV screening has been used for population-based screening [2]. In a recent study by Luo et al. the ratio of familial NPC was reported to be about 10% of all NPCs. In half of the cases the affected relatives were siblings [1]. Screening for early detection is also offered to first-degree relatives with NPC, since there is evidence for clustering of NPC within families [1]. EBV serology, EBV DNA and detection of BamHI-A rightward frame 1 (BARF1) mRNA, which is a viral oncogene expressed only in EBV-positive carcinomas, in nasopharyngeal brushing samples have been offered for screening [3]. Although there is no consensus on the follow-up of individuals at risk, the combinational use of EBV serology and nasopharyngoscopy might be a proper approach [4].

References

1. Luo XY, Liu WS, Chen LZ et al. Comparative study on risk factors and family history of familial and sporadic nasopharyngeal carcinoma patients. Zhonghua Yu Fang Yi Xue Za Zhi 2009; 43: 293-298.

2. Zong YS, Sham JS, Ng MH et al. Immunoglobulin A against viral capsid antigen of Epstein-Barr virus and indirect mirror examination of the nasopharynx in the detection of asymptomatic nasopharyngeal carcinoma. Cancer 1992; 69: 3-7.
3. Stevens SJ, Verkuijlen SA, Hariwiyanto B et al. Noninvasive diagnosis of nasopharyngeal carcinoma: nasopharyngeal brushings reveal high Epstein-Barr virus DNA load and carcinoma-specific viral BARF1 mRNA. Int J Cancer 2006; 119: 608-614.
4. Choi CW, Lee MC, Ng WT et al. An analysis of the efficacy of serial screening for familial nasopharyngeal carcinoma based on Markov chain models. Fam Cancer 2011; 10: 133-139.

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