selectively procured from hematoxylin and eosin-stained slides using a 30G1/2 hypodermic needle, as described previously [2,6]. DNA extraction from the microdissected tissues was performed by a modified single-step DNA extraction method by proteinase K treatment [2,6]. Because the *AKT1* E17K mutation was detected in the exon 3, genomic DNA each from tumor cells and corresponding normal cells were amplified with one primer pair covering the exon 3. Radioisotope ([^{32}P]dCTP) was incorporated into the PCR products for detection by SSCP autoradiogram. As a positive control, we included a breast cancer in the SSCP that had been shown to harbor the *AKT1* E17K mutation [2].

On the SSCP autoradiograms, all of the PCR products from the cancers were clearly seen. However, the SSCP from them did not reveal any aberrantly migrating band compared to wild-type bands from the normal tissues, indicating there was no evidence of the *AKT1* E17K mutation in the cancers. The positive control showed aberrant bands in the SSCP. To confirm the SSCP results, we repeated the experiments twice, including tissue microdissection, PCR and SSCP to ensure the specificity of the results, and found that the data were consistent.

Many research groups have analyzed the *AKT1* E17K mutation in many types of human cancers [1–5]. However, only breast cancers have been reported to harbor the mutation at moderate frequency (8.2%, 5.8% and 4.3%) by different researchers [1–3]. By contrast, other cancers showed very low or no incidences of the mutation [2–5]. Our study presented here did not reveal the *AKT1* E17K mutation in the cancers analyzed, either. Our data indicate that the *AKT1* E17K mutation is rare in prostate adenocarcinomas, esophageal squamous cell carcinomas, laryngeal squamous cell carcinomas, urothelial carcinomas, hepatoblastomas, GIST and malignant meningiomas, and suggest that the *AKT1* E17K mutation may not contribute to AKT signaling activation in these cancers.

Acknowledgements

This study was supported by a grant from the Innovative Research Institute for Cell Therapy, Republic of Korea (A062260). The prostate cancer tissues were supplied from the Prostate Bank in Korea supported by Korea Science and Engineering Foundation.

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Isolated bone involvement of a single lumbar vertebra body as unusual presentation of relapsing Hodgkin's lymphoma

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(Received 31 March 2009; accepted 13 April 2009)

ISSN 0284-186X print/ISSN 1651-226X online
DOI: 10.1080/02841860902968060

To the Editor

A 25-year-old man was referred to our hospital with a 2-months history of progressive fatigue, fever, malaise, profuse night sweats and weight loss. The past medical history was non-contributory. Physical examination revealed axillary adenomegalies, splenomegaly and hepatomegaly. Laboratory examination revealed a hemoglobin level of 6 gr/dl and a leukocyte count of 16 500/uL with lymphocytopenia; moreover, elevated values of erythrocyte sedimentation rate (31 mm/h), C-reactive protein (25 mg/ml) and serum lactate dehydrogenase (876/UL), and a reduced serum albumin level (3.4 g/dL) were also found. Serological tests for HIV and hepatitis virus antibodies were negative. Abdominal ultrasonography showed deep adenomegalies and confirmed liver and spleen enlargements. A chest x-ray showed a large mediastinal mass. A whole body computed tomography (CT) scan confirmed these findings. A node biopsy was taken and a histological diagnosis of Hodgkin lymphoma (HL) of classical subtype was made. A massive bone marrow (BM) disease involvement was demonstrated by trephine biopsies, for which the patient was staged as IVB. Therefore, he was treated with a standard chemotherapy (CT) including doxorubicin, bleomycin, vinblastine and dacarbazine (ABVD), according to our institutional protocols and national guidelines [1]. An interim whole body positron emission tomography (PET)/CT study with 18F-fluorodeoxy-D-glucose (FDG) and a BM trephine biopsy after the second course of ABVD showed a complete remission (CR). Therefore, the patient received additional four ABVD courses and then restaged as confirmed CR, which was maintained until one year later, when a PET/CT examination revealed a FDG avid focus localized to the body of the second lumbar vertebra. Magnetic Resonance Imaging (MRI) confirmed these pathological findings, demonstrating a suspected neoplastic lesion. Clinically, the patient was completely asymptomatic. A careful restaging, including the examination of the cerebrospinal fluid, showed no other sites of disease. Therefore, in order to determine the histological features of the vertebral tumor, it was surgically removed and a HL relapse was confirmed. Therefore, the patient received three cycles of IGEV (ifosfamide, gemcitabine, vinorelbine, and prednisolone) regimen [2] as salvage treatment, achieving an adequate CD34+ cell collection after the second course. Therefore, the patient was submitted to autologous stem cell transplantation that was conditioned with BEAM (carmustine, etoposide, cytarabine and melphalan) regimen. One month after the full hematological recovery, involved field radiotherapy (RT, 36 Gy) was given as consolidation. To date, 16 months after the disease relapse, he is well and active. Our case illustrated an isolated spinal localization of an early relapsed HL in a patient with a poor profile risk at the disease onset and a negative interim FDG-PET after the second ABVD course. At the best of our knowledge, the isolated involvement of a single vertebral body by relapsing HL has not been reported so far, although this localization has been described in the context of multicentric and advanced disease [3–6]. Moreover, our experience outlined the role of FDG-PET in the early identification of HL relapse, confirming its reported utility in combination to MRI in the evaluation of spine involvement [3]. Although the limited patient's follow-up, the management adopted by us seemed an appropriate treatment approach. In conclusion, as a result of its rarity and non-specific symptomatology, the awareness of this condition is required in the evaluation and follow-up of HL patients.

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