

Infarct associated sarcoma; a possible pathogenesis based on histological observation and clinical course in 6 cases

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Background

Bone infarct usually passes without producing symptoms. However, the infarction may become symptomatic when it becomes complicated by fracture, cystic degeneration, infection or most seriously sarcomatous transformation. Indeed, it has been proven that sarcomas develop from chronic infarct of the long bone with the incidence less than 1% of all bone sarcomas. To date less than 80 cases of infarct associated sarcoma (IAS) have been documented in the world literature. The pathogenesis of IAS is not established although the reparative tissue adjacent to an infarct has been assumed as the source of malignant transformation.

Questions/Purposes

We would like to suggest a possible histological pathogenesis of IAS based our microscopic observation and describe clinical course patients with IAS along with literature review.

Patients and Methods

Six patients with histologically proven sarcomas were collected from 2 different university hospitals between January 2003 and December 2010. Medical records, radiographic, scintigraphic images, MR images and histologic slides were reviewed retrospectively. The diagnostic criteria for sarcoma associated with bone infarct described by Desai et al. were used: (a) radiographic evidence of a single or multiple areas of bone infarct with serpentine or punctuate calcification in association with poorly-marginated osteolytic area of bone destruction; and (b) histologic demonstration of a area of infarct tissue (typified by necrotic medullary bone and fibrosis of bone marrow with dystrophic calcification) and an intimate association of fibrous tissue along the periphery of the infarct, which blended imperceptibly into the sarcoma area.

Results

The ages ranged from 43 to 63 years old. Histologic types of sarcoma were osteosarcomas, MFH, and fibrosarcoma in 2 patients, respectively. The locations were proximal tibia, distal femur and proximal femur in 2 patients each. Four patients complained pain and two patients complained growing mass. Histopathologically we observed a transitional zone (TZ) of granulation tissue with atypical cells of

varying degrees lain between infarct and sarcoma, that suggested malignant change might originate from reparative tissue around infarct area. Wide resection and reconstruction with tumor prosthesis was performed in 4 patients and amputation was done in a patient. Surgery was refused by a patient. One patient with osteosarcoma, in whom chemotherapy was not performed, died at 11 months after initial diagnosis and another patient with osteosarcoma, in whom incomplete chemotherapy was done, is alive with disease at four years after diagnosis. One patient with MFH died of sepsis during postoperative chemotherapy and another patient with MFH died of disease two years postoperatively. The patients with fibrosarcoma are doing well without evidence of disease at four years and one year after diagnosis, respectively.

Conclusions

Physicians should aware that onset of pain or growing mass over the infarct area might herald sarcoamtous transformation of the infarct. Our finding of TZ in which transition from bland interlacing spindle cell fascicles and other cells of repair tissue via cellular atypism to established sarcoma occur would sufficiently attest to the fact that sarcoma develops from the reparative tissues adjacent to old bone infarcts. Although longer follow-up of more patients is needed, survival rate of patients with IAS has seemed to be poorer than conventional sarcoma as in review of the literature.