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CROSSING CHROMOSOMES IN PLEOMORPHIC SARCOMA

A. Batistatou[1, *], *K. Charalabopoulos*[2]
[1]*Department of Pathology, University of Ioannina Medical School, Ioannina, Greece*
[2]*Department of Physiology, Clinical Unit, University of Ioannina Medical School, Ioannina, Greece*

Atypical mitoses are not unusual in neoplastic cells. They reflect dysfunction of chromatid dissociation mechanism during cell division and possibly represent specific centrosomal alterations involved in carcinogenesis [1–3]. It was shown that high grade tumors consist of aneuploid cells, which are genomically unstable and have high incidence of centrosome amplification [4]. While genomic instability certainly contributes to multistep carcinogenesis, the role of centrosome amplification (presence of more than two centrosomes during mitosis), as event contributing in malignant transformation or chromosomal instability, is not clear at this time. The causative relation between abnormal number of centrosomes, multipolar mitoses and development of cancer cell was suggested by Boveri at the beginning of 20th century. Today, there is emerging evidence that centrosome aberrations should be analyzed, based on cellular context where they occur, since they can also be detected in non-malignant cells [4, 5]. Study of molecular basis of normal centrosome duplication and dysfunction of this process with oncogenic stimuli is popular field of modern research.

Furthermore, neoplastic cell mitoses are back in the center of anti-cancer research, since new generations of drugs targeting mitosis were developed [6]. Centrosomal amplification can lead to the assembly of multipolar spindles, which create spectacular multipolar mitotic figures. Herein, we present a case of 55-year-old man, who had giant cell malignant fibrous histiocytoma (undifferentiated pleomorphic sarcoma with giant cells, according to WHO Classification), in the retroperitoneal region. During microscopic analysis abundant atypical mitoses were observed. One such mitosis is depicted in the Figure. It has close resemblance to Cross.

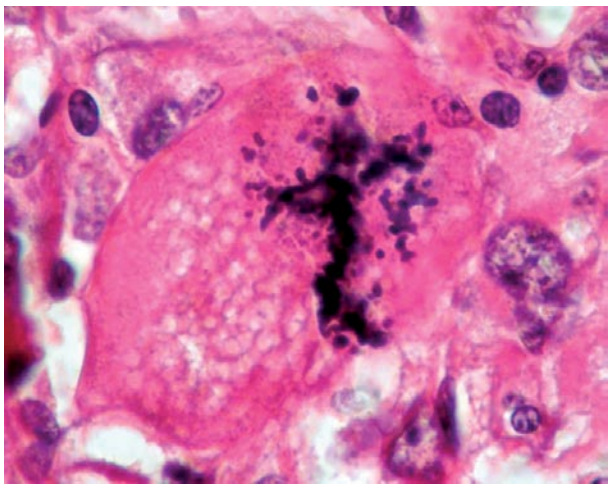

Figure. Crossing chromosomes in atypical mitosis (hematoxylin and eosin stain, X 1000)

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ПЕРЕКРЕЩИВАЮЩИЕСЯ ХРОМОСОМЫ В СЛУЧАЕ ПЛЕЙОМОРФНОЙ САРКОМЫ.

А. Батистату, К. Чаралабопулос

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*Correspondence: E-mail: abatista@cc.uoi.gr