



ABSTRACT SUBMISSION FORM

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Language	Abstract must be written and presented in English.
Title	Title of abstract should not exceed 30 words. Do not use abbreviations in the title.
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Abstract words	Abstract is to have 200-300 words.
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Abbreviations	Define the abbreviation in the first use.
Data units	Data units must be in the International System of Units (SI units).
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The AAMLS 2023 Scientific Committee reserves the right to select papers relevant to the session. Presenters may present papers without having their papers published in congress proceedings or MJMLS.

Important Dates

Abstract Submission Deadline	31 July 2023		
Presenter Registration Deadline	31 August 2023		
Presentation File Submission Deadline	8 September 2023		





Use the following excerpt as a format guide:

INVESTIGATION OF THE CHROMOSOME 6 INSTABILITY AND THE MAPK SIGNALING PATHWAYS IN HUMAN OSTEOSARCOMA

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Abstract

Osteosarcoma is the most common primary malignancy of bone with worldwide incidence of 2 to 3 million/year. This tumour usually develops at distal part of fast growing bone and associated with instability of chromosome 6. Despite that, the preceding molecular mechanism leading to the instability is not fully known. Following the local ethical committee review, from a total of 30 cases within 7years period (from October 2008 to October 2015). The study aimed to investigate the copy number of gene encoding vascular endothelial growth factor (VEGF) located at chromosome 6p21 using Fluorescence In Situ Hybridization (FISH) and the general chromosomal instability as indicated by micronuclei formation. In addition, VEGF, Hypoxia inducible factor (HIF) and p53 diagnostic immunohistochemistry (IHC) staining were performed as to investigate relationship of hypoxic stress and tumour growth suppress. Furthermore, the mitogen activated protein kinase (MAPK) cellular proliferation pathway molecules; ERK, p38 and JNK IHC were also examined. Our findings showed VEGF expression at both protein and gene level (6p21) indicating likelihood of neovascularization. On top of that, expressed ERK, p38 but not JNK; and expressed HIF & p53 in those cases suggest tumour cell survival by evading the natural cell death program, apoptosis. In conclusion, this study presented a possible molecular mechanism underlying chromosomal instability in human osteosarcoma.

Keywords: Osteosarcoma; mitogen activated protein kinase; chromosomal instability; 6p21