Cancer Genomics: Chapter 18. Genomics in Multiple Myeloma: From Conventional Cytogenetics to Novel Whole-Genome Sequencing Approaches

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Cancer Genomics: Chapter 18. Genomics in Multiple Myeloma: From Conventional Cytogenetics to Novel Whole-Genome Sequencing Approaches Francesca Cottini, Kenneth C. Anderson, Giovanni Tonon Multiple myeloma (MM) is a clonal proliferation of abnormal plasma cells in the bone marrow (BM), associated with a monoclonal protein and end-organ damage. MM originates from a pre-malignant condition, called monoclonal gammopathy of undetermined significance (MGUS) and can progress to an extramedullary disease, termed plasma cell leukemia (PCL), which invades the bloodstream. MM cells manifest a wide spectrum of genomic abnormalities, creating a strong intertumoral heterogeneity. Historically, MM patients have been divided into two subgroups: hyperdiploid cases (with >46 chromosomes) and non-hyperdiploid cases. However, the introduction of novel technologies such as fluorescence in situ hybridization (FISH), array comparative genomic hybridization (aCGH) and sequencing techniques is helping to unveil the complexity of MM genomes. In particular, MM cells present: recurrent translocations which deregulate known oncogenes, such as CCND1, FGFR3-MMSET, c-MAF and MYC, numerous copy number variations (CNVs) including deletion of chromosome 13, deletion of chromosome 17p13, and amplification of chromosome 1q21; and various somatic mutations in genes involved in cancer proliferation (RAS, BRAF, FGFR3), protein homeostasis and RNA processing (FAM46C, DIS3, XBP1 and LRRK2); NF-κB signaling; histone methylation; and tumor suppression (TP53). This chapter will summarize our current knowledge of the MM genomic field, focusing on the different types of abnormalities and their relationship with the phases of disease.

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