

E6. MULTIPLE MYELOMA FROM BIOLOGY TO THERAPY.

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Multiple myeloma is a malign plasma cell dyscrasia whose origin lies in a germinal center B lymphocyte.

This germinal center cells undergo somatic hypermutation and isotypic switch.

Malignant transformation is sequential multistep process involving the progressive accumulation of several abnormalities starting with an aberrant immune answer initiated by a B cell.

Cytogenetic changes are: numeric - trisomies of impar chromosomes, or structural - translocations which are not random.

The resultant clone, malign by its biology, may rest asymptomatic (MGUS).

About 1% of those cases per year, progress to clinical certain malignancy.

The final transformation is provided by a new genetic abnormality of the malignant cell or/and changes in bone marrow microenvironment concerning immune surveillance, angiogenesis, etc.

MM is a heterogeneous disease by pathogenesis and not only. The biology of malignant process is reflected in disease aggressively, prognostic, treatment response and overall survival.

Prognostic stratification is primary based on:

- presence/absence of genetic abnormalities (t(14;16), t(14;20) or del 17p13 by FISH) or
- molecular biology (molecular expression of the genetic abnormalities).

MM high risk (with the abnormalities mentioned above) is characterized by short overall survival (2-3 years) with conventional treatment, including ASCT (autologus stem cell transplant).

In conclusion: the molecular biology of the malignant clone has a major role in choosing a personalized chemotherapy for the patient with MM.