Cell adhesion and metastasis

Inflammation response induces vascular permeability by affecting endothelial cell-cell adhesions.

Vasoactive compounds secreted by tumor cells and adhered platelets induce vascular permeability.

Secondary tumors produce growth factors such as VEGF to promote the formation of new blood vessels (angiogenesis).

Rolling through weak adhesive interactions.

Arrest through strong adhesive interactions.

Arrest through neutrophil aggregation.

Oncogenic transformation:

- Proliferation
- Migration

Normal epithelial cell architecture controlled by contact-inhibition.

Destabilization of cell-cell adhesions by internalization of E-cadherin and increased expression of N-cadherin.

The rigidity of the surrounding tumor matrix influences tumor progression, affecting invasion, migration, and cell-cell interactions. EMT (Epithelial-Mesenchymal Transition) leading to cytoskeleton remodeling during EMT.

Cell polarization, migration, and invasion.

Extracellular matrix degradation.

Gelatinase enzymes such as MMP-2 and MMP-9 released by tumor cells, degrade collagen found in the basement membrane.

Intravasation

Invasion

Capture by cell adhesion

Extravasation

Capture by physical occlusion

Secondary tumor growth

Blood flow

Key:

- E-Cadherin
- N-Cadherin
- Integrin mediated cell-matrix adhesions
- Sites of degradation
- Matrix degrading proteases
- Selectins
- CD44, CEAD, PODXL
- ICAM1, VCAM1
- Platelets
- Inflammatory cells
- Neutrophils
- Growth factors (VEGF, Histamine) and reactive oxygen species

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