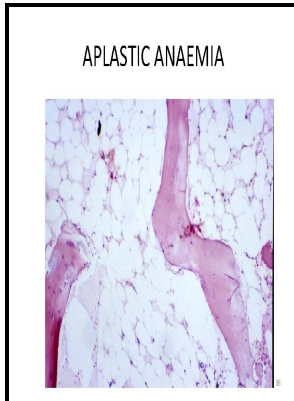


Characterisation of the haemopoietic defect in aplastic anaemia

University of Birmingham - Aplastic anaemia: Management



Description: -

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Aplastic anaemia: Management

Serial samples before and after IST were collected in 4 patients. Immunologic mechanisms in drug-induced cytopenias. Although AA MSC presented typical morphology and distinctive mesenchymal markers, stromal formation was significantly reduced; furthermore, their proliferative and clonogenic capacity was markedly decreased and ability to sustain haemopoiesis was also reduced as assessed by total cell proliferation and clonogenic ability of HSC.

THE MANAGEMENT OF APLASTIC ANAEMIA IN ADULTS, British Journal of Haematology

Regulation of macrophage and granulocyte proliferation.

Pathophysiology of aplastic anaemia

The median age of the patients was 42 years range 18-76 years.

Functional characterization of CD4+ T cells in aplastic anemia

Bone-marrow cells resistant to chloramphenicol in chloramphenicol-induced aplastic anaemia.

Aplastic anaemia

In vitro evidence of drug action in aplastic anemia.

Aplastic anaemia: Management

Nonetheless, we have shown for the first time that the reduction in Treg numbers correlates with disease severity, and the defect is most prominent

in severe and very severe AA. Proliferation and differentiation of normal granulopoietic cells in continuous bone marrow cultures. From these observations we deduce that the intrinsic defect, a premalignant haemopoietic disorder, can either be clinically quiescent by virtue of repair mechanisms, or induce auto-reactivity of the immune system against the abnormal haemopoietic tissue, drugs, chemicals and viruses acting as non-specific triggers or amplifiers.

Aplastic anaemia: Management

Pathogenesis, diagnosis, treatment and prognosis. Interestingly, percentages of the top dominant CDR3 clones revealed by high-throughput sequencing were significantly higher in AA samples than in healthy donors, regardless of the spectratyping pattern, which indicates clonally expanded Th1 cells in AA patients.

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