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Review article

The role of magnetic resonance imaging in the evaluation of transfusional iron overload in myelodysplastic syndromes

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ABSTRACT

Myelodysplastic syndromes represent a group of heterogeneous hematopoietic neoplasms derived from an abnormal multipotent progenitor cell, characterized by a hyperproliferative bone marrow, dysplasia of the cellular hemopoietic elements and ineffective erythropoiesis. Anemia is a common finding in myelodysplastic syndrome patients, and blood transfusions are the only therapeutic option in approximately 40% of cases. The most serious side effect of regular blood transfusion is iron overload.

Currently, cardiovascular magnetic resonance using T2 is routinely used to identify patients with myocardial iron overload and to guide chelation therapy, tailored to prevent iron toxicity in the heart. This is a major validated non-invasive measure of myocardial iron overloading and is superior to surrogates such as serum ferritin, liver iron, ventricular ejection fraction and tissue Doppler parameters.

The indication for iron chelation therapy in myelodysplastic syndrome patients is currently controversial. However, cardiovascular magnetic resonance may offer an excellent non-invasive, diagnostic tool for iron overload assessment in myelodysplastic syndromes. Further studies are needed to establish the precise indications of chelation therapy and the clinical implications of this treatment on survival in myelodysplastic syndromes.

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Introduction

Myelodysplastic syndrome (MDS) comprises an acquired primitive stem cell disorder resulting in ineffective hematopoiesis manifested by variable degrees and numbers of cytopenias, as well as an increased risk of transformation to acute leukemia. MDS is relatively common with a reported incidence of 3.5–4.9 per 100,000 people.[1] The incidence increases to 28–36 per 100,000 in over 80-year-old individuals, making it as common as myeloma in this age group.[2] Red blood cell (RBC) transfusions comprise the most effective

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treatment of anemia in MDS patients, in the expense of organ damaging iron overload.

Magnetic resonance imaging (MRI) has been successfully used for the evaluation of myocardial and liver iron overload. MRI is the only technique able to provide non-invasive information about iron overload, as well as microcirculation defects and the detection of myocardial scars.

Myelodysplastic syndromes

MDS represent a group of heterogeneous hematopoietic disorders derived from an abnormal multipotent progenitor cell, characterized by a hyperproliferative bone marrow, dysplasia of the cellular hemopoietic elements, and ineffective hematopoiesis (Figure 1). Peripheral blood cytopenias and marked morphologic dysplasias are prominent and ineffective erythropoiesis results in symptomatic anemia.[3] Cellular dysfunction results in an increased risk of infection, bleeding tendency due to thrombocytopenia, and need for transfusions in most MDS patients.[4] MDS can be classified as primary (idiopathic) or secondary (therapy-related), the latter being associated with prior radiotherapy, chemotherapeutic agents, and immunosuppression therapy.[5] Other risk factors for MDS development include benzene exposure, occupational chemicals, tobacco exposure, excessive alcohol, viral infections, and autoimmune disorders, as well as chronic inflammation.[5] A useful classification of MDS according to their pathogenesis, cytological features and specific karyotypes, was proposed initially by the French-American-British (FAB) Cooperative Study Group.[6] More recently, the World Health Organization (WHO) worked out an updated classification that represents an extension of the FAB proposal, with several modifications.[7] Alterations in many individual biological pathways have been implicated in MDS pathophysiology. However, the primary hypothesis involves an initial deleterious genetic event within a hematopoietic stem cell, subsequent development of excessive cytokines/inflammatory response leading to a proapoptotic/proliferative state, resulting in peripheral cytopenias despite a hypercellular bone marrow. Furthermore, the presence of detectable cytogenetic abnormalities in approximately 40–70% of patients with primary MDS and over 80% with secondary MDS, as well as the validated prognostic value of specific cytogenetic aberrations in MDS, supports the theory of an incidental genetic event.[8]

Anemia, transfusion and iron overload in myelodysplastic syndrome patients

A limited number of effective treatment options are available to treat anemia and thus help to prevent iron overload and other transfusion-related side effects in MDS patients. A direct approach is to correct anemia by administering hematopoietic growth factors, i.e. erythropoietin with or without granulocyte-colony stimulating factor (G-CSF).[9] Other drugs, such as lenalidomide, cyclosporine-A and antithymocyte globulin act in certain subgroups of MDS patients and may improve or correct anemia.[10–12] Allogeneic stem cell transplantation is the only curative approach.[13]

RBC transfusions are considered in MDS patients when hemoglobin (Hb) <8 g/dL, and may provide temporary relief from the symptoms of anemia, but they also add extra iron to the body.[14] And while there are therapies, as mentioned above, that can restore the production of RBC so that patients can become transfusion independent, they are not effective in all MDS patients. In fact, for approximately 40% of MDS patients, transfusions are the only option to treat the symptoms of anemia.[4]

Supportive therapy with regular RBC transfusions can lead to elevated levels of iron in the blood and other tissues. The actual prevalence of iron overload in transfused MDS patients has not been systematically documented.[15] Each unit of packed RBC contains about 250 mg of iron. As a general rule, iron overload occurs after the transfusion of 20 units of RBC.[15] Thus, MDS patients who receive transfusions for their anemia are at risk for iron overload. In addition to iron overload as a result of multiple transfusions, MDS patients with sideroblastic anemia may develop iron overload subsequent to excessive absorption of iron from food.[16]

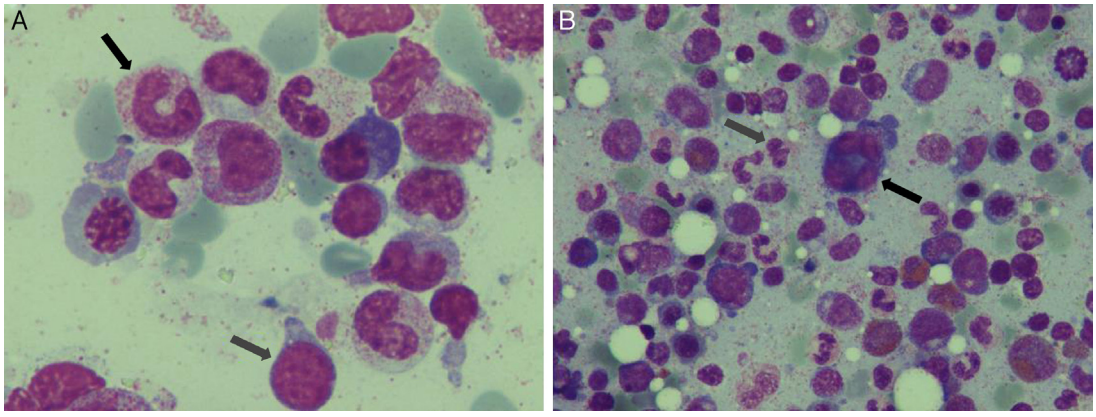

Figure 1 – Characteristic bone marrow films in myelodysplastic syndrome. (A) Giant granulocyte (black arrow) and blast cell (gray arrow). (B) Dysplastic megakaryocyte with multiple separated nuclei (black arrow) and pseudo-Pelger cells (gray arrow). Images courtesy of Dr V. Karali (First Department of Propaedeutic and Internal Medicine, Athens University Medical School, Athens, Greece).

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