Anemia in Cancer: Some Pathophysiologica

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Key Words: Erythropoietin · TNF-alpha · Quality of life · Neoplasms

ABSTRACT

More than 30% of cancer patients experience anemia and its side effect, fatigue. Its causes can be numerous, but anemia is usually secondary to an imbalance of cytokines. Among these, tumor necrosis factor-alpha seems to be the major culprit, creating anemia by blunting the physiologic
cal effect of erythropoietin. Pharmacologically increasing the erythropoietin level corrects the anemia in about half the treated patients. Several studies have shown that quality of life is substantially improved through such therapy. The Oncologist 2003;8(suppl 1):19-21

INTRODUCTION

Anemia, commonly defined as a hemoglobin level of <12 g/dL, occurs in over 30% of cancer patients at any point in time, and its incidence increases with treatment and progressive disease [1]. This anemia can have many causes:

1. related to the patient (hemoglobinopathies, thalassemia, gastrointestinal problems, etc.);
2. related to the disease (bone marrow infiltration, bowel resection, hypersplenism, diminished nutritional state, etc.);
3. related to therapy (hypoplasia of bone marrow-bearing areas such as the pelvis secondary to radiotherapy; bone marrow and renal toxicity secondary to chemotherapy; and, occasionally, drug-induced hemolysis, etc.).

ANEMIA AND ERYTHROPOIETIN

Bone marrow stem cells are self-renewing and able to support a normal hemoglobin level over a lifetime. Red blood cells derive from committed stem cells that differentiate and multiply through the different erythroid stages. There is, as in all human cells, an inverse relationship between proliferation potential and differentiation. Both events are finely regulated by cytokines, of which erythropoietin is the most important once the erythroid pathway is entered. Hypoxia is sensed by the nephron, and the kidney responds with erythropoietin production. The erythropoietin binds to a specific receptor on the red blood cell progenitors, and its signaling induces proliferation and differentiation and has an antiapoptotic effect. Another general antiapoptotic pathway, NF-kB production—which occurs as a response to inflammatory events—has recently been linked by possible
REFERENCES


