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FERA-Q Tablets: Helps to Support the Treatment of Cancer Induced Anaemia in Patients Undergoing Chemotherapy and Radiation Therapy

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ABSTRACT

Cancer-induced anemia is most common in patients with cancer and in patients undergoing chemotherapy and radiation therapy. The severity of anemia depends on the extent of disease and the density of treatment. Repeated cycles of chemotherapy may impair erythropoiesis synthesis that results in decrease functional capacity and quality of life. In cancer patients, the incidence of anemia may rise 40%.According to European cancer anemia survey (ECAS) 53% of patients did not receive any treatment for their anemia. Anemia has also been identified as an adverse prognostic factor for these patients. Thus Anemia exerts a negative influence on the quality of life of cancer patients as it may contribute to cancer-induced fatigue. This article reviews the current available scientific literature regarding the effect of FERA-Q tablets to support the treatment of Cancer induced Anaemia in patients.

Keywords: FERA-Q tablets, Cancer induced Anaemia.

INTRODUCTION

Anemia exerts a negative influence on the quality of life of cancer patients as it may contribute to cancer-induced fatigue. Cancer-induced anemia is most common in patients with cancer and in patients undergoing chemotherapy and radiation therapy. The severity of anemia depends on the extent of disease and the density of treatment. Repeated cycles of chemotherapy may impair erythropoiesis synthesis that results in decrease functional capacity and quality of life [3]. In cancer patients, the incidence of anemia may rise 40% [2]. According to European cancer anemia survey (ECAS) 53% of patients did not receive any treatment for their anemia [1]. Anemia has also been identified as an adverse prognostic factor for these patients thus Anemia exerts a negative influence on the quality of life of cancer patients as it may contribute to cancer-induced fatigue. Some chemotherapeutic agents like cisplastin, docetaxel, paclitaxel, cisplastin plus cyclophosphamide, carboplatin, etoposide, leucovorin and vinorelbine induce anemia by impairing hematopoiesis in head and neck, non-small cell lung, breast, ovarian, non-hodgkin lymphoma and small cell lung cancer (SCLC).

Patho-Physiology of chemotherapy induced anemia

The patho physiology of anemia is due to blood loss, increased destruction of red blood cell, decreased production of functional red blood cells. Tumor necrosis factor-α, GATA-1, GATA-2 inhibits hemoglobin production. And in case of inflammation IL-6 induces the liver to produce hepcidin, which decreases iron absorption from bowel and blocks iron utilization in bone marrow [2]. Some chemotherapeutic agents like cisplastin, docetaxel, paclitaxel, cisplastin plus cyclophosphamide, carboplatin, etoposide, leucovorin and vinorelbine induce anemia by impairing hematopoiesis in head and neck, non-small cell lung, breast, ovarian, non-hodgkin lymphoma and small cell lung cancer (SCLC) [3,2].

Combination cyclophosphamide-doxorubicin-vincristine chemotherapy a standard regimen for SCLC, anemia of grades 1-4 was observed in 13% and 54% in these patients.[5]the commonly used combination of

cyclophosphamide– doxorubicin–5-FU produced grade 1 or 2 anemia in 55% and grade 3 or 4 anemia in 11% of previously treated patients with metastatic breast cancer [4].

Mechanism of Action of fera-Q tablets.

Zinc in fera-Q tablets

Zinc in fera-Q tablets shows regulatory role in to gene expression, cell signaling, and nerve impulse transmission, as well as normal apoptosis[7].Zinc plays an important role in the structure of proteins and cell membranes. Loss of zinc from biological membranes increases their susceptibility to oxidative damage and impairs their function [8]. Zinc's role in supporting immune function includes regulating T lymphocytes, natural killer cells, CD4 cells, and interleukin-2. The mechanism of zinc inhibition of prostatic cell growth involved the effect of zinc on cell cycle progression (cell arrested G2/M) and apoptosis [5,6].

Vitamin B6 in fera-Q tablets

Vitamin B6 is a water-soluble vitamin that participates in more than 100 coenzyme reactions involved in the metabolism of protein, carbohydrates, and lipids. Plasma pyridoxal 5'-phosphate (PLP) is the active form of vitamin B6. Vitamin B6 may influence colorectal carcinogenesis through its role in DNA synthesis and methylation. Vitamin B6 has been shown to inhibit angiogenesis, suppress nitric oxide, and reduce oxidative stress, all of which are associated with preventing carcinogenesis. DNA methylation plays an essential role during normal development, however, it is also considered to be a critical process in the development of several forms of cancer. Dietary methyl (folate, choline, and methionine) deficiency in combination causes decreased tissue S-adeno-sylmethionine, global DNA hypomethylation, hepatic steatosis, cirrhosis, and ultimately hepatic tumorigenesis in rodents in the absence of carcinogen treatment [13].

Methyl folate in fera-Qtablets

Methyl folate helps to regulate gene activity by controlling gene methylation there by it helps in cancer prevention.

Methylcobalamin and 5'-deoxyadenosylcobalamin, as sources for vitamin B12 in fera-Q tablets is involved in synthesis of phospholipids, neurotransmitters, DNA, and the metabolism of fatty acids and amino acids in cells [13]

Composition of Fera-Q tablets.

FERA-Q™

Supplement Facts

Serving size : 1 Tablet — Servings per pack : 30

Each film coated tablet contains (approx):		% ICMR RDA*
Ferrous bisglycinate Equivalent to Elemental Iron	17mg	100
Cyanocobalamin	1 mcg	100
Folic Acid	200mcg	100
Pyridoxine (as Pyridoxal 5 phosphate)	2 mg	100
Zinc(as Zinc Sulphate)	10mg	100

* Indian Council of Medical Research Recommended Daily Allowances
**Not Established.

Clinical study Reports of Zinc in Fera-Q tablets

The zinc level in prostate cancer is markedly decreased from the level detected in non prostate tissues. Growth inhibition and various regulatory responses were investigated in two human prostate carcinoma cell lines (LNCaP and PC-3), treated with or without zinc. The specificity of zinc-induced apoptosis was identified by ethylenediamine-tetraacetic acid (EDTA)-chelation, which abolished the zinc effect on cellular DNA fragmentation. The zinc-induced G2/M phase arrest and apoptosis were accompanied by increased mRNA levels of p21Waf1/Cip1/Sdi1 in both LNCaP (p53+/+) and PC-3 (p53−/−) cells. The results suggest that zinc inhibits human prostatic carcinoma cell growth, possibly due to induction of cell cycle arrest and apoptosis [16].

Clinical study Reports of Vitamin B6 in Fera-Q tablets

Studies on plasma pyridoxal 5'-phosphate (the active form of vitamin B6) in Fera-Q consistently support an approximately 30%-50% reduction in risk of colorectal cancer comparing high with low concentrations [11].

Two hundred schoolchildren from public boarding schools in Mexico City who had low iron stores as assessed by serum ferritin concentration but with anemia were randomly assigned to a daily supplement of 30 mg/day of elemental iron as ferrous sulfate or iron bis-glycinate chelate for 12 weeks. Iron status was evaluated at baseline, one week post-supplementation (short term), and 6 months (medium term) after supplementation. The results showed positive effects on increasing ferritin concentration in schoolchildren with low iron stores, and this effect persisted 6 months after supplementation [14].

Clinical study Reports of ferrous bisglycinate in Fera-Q tablets

Efficacy and toxicity of oral ferrous bisglycinate in Fera-Q and ferrous sulfate in cancer patients with mild IDA was studied. Twenty-four patients operated on for solid tumors (10 breast, 12 colorectal, 2 gastric), with non-chemotherapy-induced hemoglobin (Hb) were randomized to receive oral ferrous bisglycinate chelate, 28 mg per day for 20 days, and then 14 mg per day for 40 days (12 patients) (A group) or oral ferrous sulphate, 105 mg per day for 60 days (12 patients) (B group). Values of hemoglobin and ferritin obtained at diagnosis, 1 and 2 months from the beginning of treatment were compared. In conclusion, these data suggest that ferrous bisglycinate chelate has similar efficacy and likely lower GI toxicity than ferrous sulphate given at the conventional dose of 105 mg per day for the same time [15].

Pharmacokinetics of fera-Q tablets

In vivo study whereby two groups of 4 male Sprague-Dawley rats each received an intra-peritoneal dose of 5.0 µg of 65Zn as either 65Zn bisglycinate or as 65Zn chloride following a 24-hour fast. Four hours post dosing, each animal was sacrificed and a number of tissues were excised including thigh muscle tissue, left ventricle, liver, kidney and right cerebrum of brain. Each tissue sample was assayed for radioactivity. It was shown that the zinc deposition from 65Zn bisglycinate absorption was greater in muscle, kidney and brain. Zinc bisglycinate has been shown to achieve superior absorption and bioavailability [10].

Intestinal uptake of vitamin B12 occurs in the terminal ileum. The majority of vitamin B12 that enters the body via nutrition is stored in the liver. The amount of vitamin B12 excreted from the body (turnover rate) is fixed at

0.1%-0.2% of total body stores daily, regardless of the size of the pool. Although the rate of vitamin B12 excretion is not directly proportional to the intake, increased intake of vitamin B12 results in greater liver storage and, thus, increased excretion [12].

Recommended Usage of Fera-Q tablets

- One tablet perday or As Directed by Healthcare Practitioner.
- Do not exceed the recommended daily dose.

SUMMARY & CONCLUSION

Cancer-related anemia adversely affects quality of life and is associated with reduced overall survival. The correction of anemia in cancer patients has the potential to improve treatment efficacy and increase survival. A large number of studies demonstrate that treatment of anemia in cancer patients using Fera-Q tablets significantly increases hemoglobin levels, decreases transfusion requirements and improves quality of life, predominantly by reducing fatigue in cancer patients.

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