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ANTIOXIDANTS FOR HAEMOCHROMATOSIS

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Hemochromatosis is a potentially fatal iron storage disease with excessive iron deposits especially in the liver. The idiopathic form occurs usually in males and manifests from the age of 30 to 50. It is thought to result from a lifetime of excessive iron absorption, the cause of which is not known. Prominent long-term effects are liver cirrhosis, diabetes mellitus, cardiac failure and strong skin pigmentation.

Iron is deposited in the 3-valence form as ferritin and hemosiderin. Serum ferritin levels closely reflect tissue levels and are greatly increased. The diagnosis is confirmed by a liver biopsy. The conventional treatment consists of phlebotomy with weekly to monthly bleedings until plasma iron or ferritin levels reach the lower normal range. Chelation of iron as with desferrioxamine to increase urinary excretion is not very effective and usually confined to anaemic patients.

CASE HISTORY

A 48-year old male collapsed at work in December 1987. The main symptoms were dizziness, tachycardia, heavy sweating, difficulty breathing and extreme' weakness. Treatment in an Intensive Care Unit with numerous tests produced no definite diagnosis. Loss of weight and weakness continued after discharge. The diagnosis of hemochromatosis was established in March 1989 by liver biopsy and a serum ferritin level of 1585 ng/ml (normal for males: 30-400 ng/ml).

Phlebotomy was performed initially fortnightly and later monthly of one litre of blood. Serum ferritin levels fell initially steeply but very slowly in the last 6 months to a low of 440 ng/ml at the end of October 1990. During all this time the patient was very weak. In early December 1990 a specialist diagnosed Meniere's disease as an additional complication.

Phlebotomy was stopped and nutritional therapy started in December 1990. It consisted predominantly of a vegetarian raw food diet with supplements of B complex, sublingual vitamin B 12, Kyolic garlic, lecithin, freeze-dried acidophilus culture, zinc, copper, manganese, and high levels of antioxidant vitamins, in particular 20g of ascorbate, 1000 iu of natural vitamin E and 10,000 iu of vitamin A, all in divided doses. Ascorbic acid was not well tolerated and sodium ascorbate used instead. In order to minimise any increased iron absorption due to ascorbate this was initially taken with water or fruit well before meals.

After about 3 days on this program, on December 11 the serum ferritin level was 458 ng/ml. However, on December 28 the ferritin level was 393 ng/ml, within the normal range for the first time since diagnosis. After this the diet was relaxed to include cooked and flesh foods. In order to assess the effectiveness of a lower ascorbate level, the daily intake was reduced to 5 g and (to preclude any direct influence of high ascorbate levels on tests) no

supplementation was used in the 24 hours before the next test on January 10,1991, which showed a further drop of the ferritin level to 380 ng/ml.

With the start of the nutritional therapy the patient began to feel well for the first time in 3 years with rapidly increasing strength and disappearance of all signs of Meniere's disease. The red blood cell count which was below the normal range in December, moved into the normal range and haemoglobin, which had been near the lower limit, moved towards the middle of the normal range. Preliminary maintenance supplementation consists of about 5 g of ascorbate, 500 iu of vitamin E, 10,000 iu of vitamin A, B complex and lecithin.

DISCUSSION

Vitamin C therapy is generally regarded as potentially harmful in cases of iron overload. Ascorbic acid may increase the absorption of iron and it is perceived that toxic products may form from the combination of ascorbate with iron salts liberated from ferritin tissue stores (1). In this way, cardiac damage, often transient, has been detected in thalassemia patients taking 500 mg/day of ascorbic acid (2). Such tissue damage may result from the peroxidation of membrane lipids and, not surprising, in tissues severely deficient in antioxidants. To avoid or limit toxic effects from iron liberated by ascorbic acid it has been proposed to use vitamin C supplementation only in combination with desferrioxamine in the treatment of iron overload (2).

However, it would appear much more effective to protect cell membranes from iron-induced peroxidation damage during therapy with a high level of antioxidant supplementation. Also sufficient vitamin B6 is required to prevent iron overload.

It is well known that high concentrations of ferric ions oxidise the protective antioxidants, notably the vitamins C and E. This means that with a non-supplemented diet we can expect pronounced tissue deficiencies of these antioxidants in iron overload diseases.

It has recently been reported that vitamin A supplementation improved the iron status (serum iron, haemoglobin as well as transferrin saturation) without at the same time increasing ferritin levels, while iron supplements without additional vitamin A increased ferritin levels. Also vitamin A deficient subjects developed anaemia despite sufficient dietary iron (3,4). This may be interpreted as a normalising function of vitamin A in the transport and usage of iron, while iron alone may mainly increase iron stores in case of vitamin A deficiency.

A similar normalising role in iron metabolism may be exhibited by the other antioxidants. V ly improves the absorption of iron, it is also required to move iron in and out of ferritin tissue stores. Without adequate antioxidants, ferric iron stores may build up because iron cannot be liberated from tissue ferritin and transferred onto plasma transferrin, a step that requires a temporary reduction of ferric to ferrous iron.

However, recent studies show that excess tissue ferritin is catabolized by lysozyme activity, which is inhibited by ascorbate. The mechanism for this inhibition is not known (1). I suggest that in the case of iron overload, physiological levels of ascorbate are actually present to a high percentage in oxidised form as reversible dehydroascorbate or as irreversible oxidation products.

The observed ly ld then actually be due to oxidised ascorbate. Similarly, any liberated iron

would be in the form of ferric dehydroascorbate and other oxidised products and it is these, which cause the peroxidative membrane damage. With sufficiently high antioxidant levels, on the other hand, ascorbate may well stimulate lysozyme activity and liberated iron would be present as harmless or even beneficial ferro ascorbate.

Stevens et al (5) reported an increased risk of cancer in men with excess stored iron who also had lower total iron binding capacity and higher transferrin saturation. Generally no correlation was found between iron status and dietary iron intake. Again, I suggest that there may be an association between iron overload as well as cancer with the antioxidant status. High antioxidant levels can be expected to lower not only excess iron stores but also independently of the iron metabolism reduce the risk of cancer.

Judging by the health of the Bantu people it has been speculated that scurvy may be beneficial with iron overload (2). I suggest that on the contrary, the loss of vitamin C due to cooking in iron pots was the primary reason for their iron overload.

There are two sites or steps at which antioxidant deficiency might cause or contribute to hemochromatosis. The synthesis of haem requires the reduction of ferric to ferrous ions. While this reduction proceeds enzymatically through ferrochelatase, there may be either a deficiency of this enzyme or an impeded function if the cell is deficient in antioxidants. In addition, ferrochelatase is inhibited by lead ions. This would lead to low haemoglobin levels, possibly raised methaemoglobin and increased ferritin stores as a cellular deficiency of ferrous ions for the haem synthesis could stimulate increased absorption of iron.

The second possibility appears to be more important in hemochromatosis and could arise from a difficulty in recycling iron from the continual breakdown of haemoglobin in the spleen. A led daily in this way, but this requires a reduction-oxidation step to transfer ferritin iron in the tissue onto plasma transferrin. With antioxidant deficiency there would be only a partial recycling. M f the iron stores would build up in the liver where the decomposed haemoglobin arrives through the portal vein after its liberation from old erythrocytes in the spleen.

However, very high ferric iron stores in the liver would also make this organ more antioxidant deficient than other tissue. The highest antioxidant activity may well be in the intestinal mucosa as these have first call on the antioxidants absorbed from food. Therefore, transferrin will preferentially pick up iron from the intestinal mucosa and avoid the liver stores as too difficult to convert. Another piece of evidence for this proposed mechanism may be seen in the rapid normalisation of ferritin levels in the reported case without any abnormal loss of iron with the urine.

CONCLUSION

In summary, it is postulated that the basic biochemical defect which leads to the development of hemochromatosis is a tissue deficiency of antioxidants which inhibits the recycling of iron from old erythrocytes and this in tum induces increased intestinal absorption for the necessary synthesis of haem. The obvious advantages of antioxidant therapy as compared to the traditional management of hemochromatosis will hopefully stimulate more research in this area.

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The hemochromatosis patient mentioned in this article did not have any further hemochromatosis problems for more than 20 years, I did not maintain contact with others who seem to have been helped. According to my present understanding the number one issue is to use lots of all kinds of antioxidants (including turmeric) with as much spaced out sodium ascorbate as possible without getting bowel tolerance problems. It does not matter if taken with meals or at other times, e.g. 2 to 3 grams 5 or 6x daily.

Natural food is always good but does not make much direct difference for hemochromatosis. More important are intestinal sanitation, anti-inflammatory remedies and antimicrobial therapy as in The Ultimate Cleanse, and especially antifungal therapy as in OVERCOMING CANDIDA. The most important general r ly DMSO, see MSM and DMSO, also helpful is COPPER SALICYLATE. An additional possibility is the use of natural chelating agents. I am now a health writer and have not worked with patients for many years. Please do not try to contact me in this matter as I do not have any additional information. Also see Natural Therapy for Haemochromatosis.

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