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efficiency of iron removal from the lumen. Moreover, the application of this technique to the examination of the removal of therapeutic doses of iron also would be interesting. The arrival of free iron in the lower reaches of the intestinal tract may have implications for the pattern of colonization of colonic flora.

One additional variable which is inherent to the control of iron absorption but ignored in the Matseshe study is the status of iron nutriture of the subjects themselves. This always has been a confounding variable in the conventional studies employing radioisotopes of iron.[6] The four subjects had serum ferritin concentrations of 274, 148, 42 and 25 ng per milliliter. The subjects with low ferritin had a hemoglobin concentration of 12.6 g per deciliter and a hematocrit of 37 percent. Although the number of subjects is small, it would have been interesting to know if the efficiency of iron removal or its location correlated with the iron nutriture of the subjects.

The application of this ingenious technology of the Mayo Clinic investigators[4] to an examination of the absorptive physiology of iron in human subjects complements the studies conducted with radioisotopes, and reinforces the contemporary view that the critical factors in the regulation of iron entry into the body are intramucosal events, not removal of the mineral from the intestinal lumen. □

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DEPRESSION OF SERUM LEVELS OF RETINOL AND RETINOL BINDING PROTEIN DURING INFECTION

L.A. Mejía INCAP

The serum levels of retinol and retinol binding protein are depressed during infection and are restored after recovery from the infectious episode.

Key Words: infection, vitamin A levels, retinol binding protein levels, fever

The interaction between hypovitaminosis A and infection is well recognized. Vitamin A deficiency diminishes the resistance to infection and infection also can impair vitamin A nutriture.[1]

In relation to the latter phenomenon, Shank et al.[2] reported in 1944 that in patients with rheumatic fever there is a decrease in vitamin A concentration with the development of the disease. This observation was confirmed by Jacobs et al.[3] in patients with rheumatic fever and other infectious diseases.

Recently, Arroyave and Calcano showed that during infection the levels of both retinol and retinol binding protein (RBP) are lower than the levels observed after recovery.[4] In this investigation, two groups of Guatemalan subjects were studied. The first group included 24 urban and rural children between two and seven years of age. They

belonged to the low socioeconomic stratum and at the time of the study were residents of a children's convalescence home in Guatemala City. At the beginning of the study, seven children had chicken-pox, seven had bronchitis, nine had upper respiratory infection and one had febrile stomatitis. The other group of subjects consisted of an urban group of 30 ambulatory adult patients, whose ages ranged from 18 to 52 years. They belonged to the medium or high socioeconomic stratum and were residents of Guatemala City. At the beginning of the study, 21 of them had upper respiratory infection, two had bronchitis, two had tonsillitis, two had diarrhea and there were three single cases respectively of a non-specific gastrointestinal alteration, urinary infection and shigellosis. The dietary intake remained constant in all subjects throughout the study and no additional vitamin A was given in any form. Three blood samples were obtained from the subjects at the time of the disease (S_0), 15 days after recuperation (S_1) and at eight days or more after the second sample S_1 (S_2). In these samples, serum levels of retinol, RBP, carotenes, total proteins, albumin and globulin were determined. The most clear and marked effect of infection was on serum retinol. On the average, both groups had significantly ($p<0.01$) lower levels of vitamin A during the infectious episode (S_0) than the levels attained after recovery (S_1 and S_2). In children, these levels changed from 24.8 ± 2.8 μg per deciliter in S_0 to 32.9 ± 1.7 μg per deciliter in S_1 and to 36.3 ± 1.5 μg per deciliter in S_2. In adults the levels changed from 50.2 ± 2.8 μg per deciliter in S_0 to 59.5 ± 2.9 μg per deciliter in S_1 and to 58.6 ± 2.1 μg per deciliter in S_2. The change in individual values ($S_2 - S_0$) ranged from 0.6 to 33.1 μg per deciliter in children and from 0.01 to 42.7 μg per deciliter in the adults. Five children and seven adults showed a greater than 20 μg per deciliter change in this variable.

The serum levels of RBP were also lower during infection than the levels observed after recovery. In children, there was an insignificant change from 2.9 ± 0.2 mg per deciliter in S_0 to 3.2 ± 0.2 mg per deciliter in S_1 and to 3.2 ± 0.1 mg per deciliter in S_2. In adults, the levels changed significantly ($p<0.01$) from 4.0 ± 0.2 mg per deciliter in S_0 to 5.0 ± 0.2 mg per deciliter in S_1 and to 5.4 ± 0.2 mg per deciliter in S_2. Furthermore, there were significant correlations between serum levels of retinol and RBP in the subjects with and without infection suggesting that the change observed in retinol was accompanied by a parallel change in RBP. The serum levels of carotenes were significantly lower during infection only in the group of children ($p<0.05$). Albumin levels were reduced significantly in all infected subjects ($p<0.01$) and only the infected children had an increase in serum globulin ($p<0.01$). There was no change in total proteins and none of the biochemical parameters studied differed significantly between S_1 and S_2. Based on the data obtained from the group of children, the authors inferred that there was a more marked effect of infection on the changes observed when this was accompanied by fever. There were only four cases without fever, however, and in the adult group, regardless of fever, the results were similar. Therefore, no strong conclusion can be made in this regard. Although there was no clear effect of the specific type of infection on the results obtained, a greater reduction in serum retinol and RBP was observed in children suffering from upper respiratory infection.

In general, the results confirm the view that infection lowers the serum levels of retinol and that serum RBP levels drift in the same direction. In relation to the mechanism by which serum retinol is reduced, it was suggested to be related to an impaired release of retinol from the liver or to an increased turnover of RBP. The period of study was relatively short during which there was no change in vitamin A intake. The true nature of this interesting phenomenon awaits further study. □

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