Carotenemia in Normal-weight Bulimia: A Finding Unrelated to Other Physical Manifestations of the Syndrome

Arthur C. Jaffe, M.D.
Lauren G. McAliley, M.S.N.
Lynn Singer, Ph.D.

Carotenemia, well described in eating disorders, is considered a marker for one or another pathophysiologic process occurring during the disease. We studied 17 adolescent, normal-weight women with bulimia, 11 of whom were determined clinically to be candidates for outpatient treatment, and 6 of whom were recommended for hospitalization. The outpatient group had an elevated serum carotene level (mean = 309.0 ± 92.0 µg/dl), whereas the inpatient group had a normal level (mean = 164.8 ± 59.2 µg/dl; t = 3.92, p = .002). Our data do not support the relationship of carotenemia to malnutrition, menstrual dysfunction, or any other factor associated with the manifestations of eating disorders. We suggest that carotenemia may be an indicator of some homeostatic mechanism activated in response to the disorder.

INTRODUCTION

Carotenemia is well recognized as a curious, poorly understood laboratory finding seen with some regularity in eating disorders. It has been reported in anorexia nervosa (Pops & Schwabe, 1968; Robboy, Sato, & Schwabe, 1974) and has also been described in patients who have significant weight loss and symptoms of binge eating and purging (Curran-Celentano, Erdman, Nelson, & Gra-
ter, 1985), a disorder variously classified as bulimia (Halmi, 1983; Schlesier-Stropp, 1984), bulimia nervosa (Russell, 1979), or a bulimic subtype of anorexia nervosa (Casper, Eckert, Halmi, Goldberg, & Davis, 1980; Garfinkel, Moldofsky, & Garner, 1980). Various theories proposed to explain the carotenemia include a direct relationship with malnutrition (Frumar, Meldrum, & Judd, 1979), the eating of foods high in carotene (Robboy et al., 1974), altered metabolism of vitamin A (Robboy et al., 1974), abnormal lipid metabolism (Pops & Schwabe, 1968; Langan & Farrell, 1985), or abnormalities of thyroid function related to decreased weight or food intake (Curran-Celentano et al., 1985). There is apparently no evidence that carotenemia occurs in patients with eating disorders who are of normal weight, or that it may have any practical clinical importance.

This paper describes a group of adolescent women with bulimia, but without clinically significant weight loss or signs of malnutrition, who exhibited a mild degree of carotenemia. Furthermore, carotenemia appeared to be a marker of patients who did not have clinical indications for inpatient treatment. In contrast, those patients with normal serum carotene levels tended to be recommended for immediate hospitalization for their bulimia.

**METHODS**

During an 18-month period, 30 adolescent women were referred to the Medical-Behavioral Center at Rainbow Babies and Children's Hospital for outpatient evaluation of bingeing and purging behavior. Referrals came in a variable pattern from physicians, psychologists, or patients or their parents. All patients were seen by a pediatrician, clinical nurse specialist, dietitian, and clinical psychologist. In all cases, other organic or psychologic causes of eating disorders were ruled out. All patients were diagnosed as having bulimia based upon the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III). Determination of desirable body weight (DBW) for height was made using standard methodology. Laboratory studies were ordered in an individualized fashion. At first, serum carotene levels were not ordered routinely, but with time they became part of the standard evaluation. Thus, data on carotene levels are available on 17 patients, all of whom comprise the group discussed in this paper.

Recommendations for therapy were provided to patients at the time of outpatient evaluation, before the results of carotene assays were known. Standard practice was to recommend outpatient treatment unless the patient had serious medical complications of bulimia, was at less than 80% of DBW, had symptoms of a major depression, had previously failed a course of outpatient therapy, or was considered very likely to fail to cooperate with an ambulatory treatment regimen. Six patients were thus recommended for immediate admission: one because of premature ventricular contractions secondary to hypokalemia, one because of concern over possible suicide, two because of lack of improvement during prior outpatient therapy, and two because of clinical concern about inability to participate in an ambulatory program. It was felt that 11 patients were able to be treated out of the hospital.

Statistical evaluation of the two groups included calculation of the mean
value and standard deviation within each group for various parameters. The two groups were compared by the two-tailed Student's t-test with the Bonferroni modification for multiple comparisons; thus, each of the seven parameters was tested for criticality at a significance level of \( p < .007 \).

RESULTS

As seen in Table 1, the outpatient and inpatient groups of patients were similar in many regards. They had a mean age of 16 years at the time of their evaluation, had had the onset of the syndrome at age 14 years, and thus had had about a 2-year course of bulimia at the time of presentation. The group recommended for admission had a somewhat lower weight, which, although possibly of clinical importance, did not reach statistical significance. The mean weights in the two groups were 49.8 and 59.8 kg \( (t = 2.11, p = .05) \), and the mean percentages of DBW were 90.3 and 104.4% \( (t = 2.52, p = .02) \). These differences are small enough so that they might be expected to occur by chance in a series of multiple comparisons. Weakness in patient recall of previous weights and heights precluded accurate assessment of DBW at the onset of the disorder, or at various times during it, but there seemed to be no clear differences between the groups. Similarly, accurate determination of the amount of daily exercise proved impossible, but only two patients, both members of the outpatient group, denied engaging in a daily program.

It was also difficult to obtain a good history of the age of menarche. However, all patients but two were at the Tanner V stage of physical development. One member of the outpatient group was at stage IV, and one member of the admission group was at stage III. As shown in Table 2, both groups tended to have abnormal menstrual function at the time of evaluation. "Normal" menstruation was defined as 4-5-day periods occurring every 4 weeks. "Irregular" menstruation included abnormalities of flow or cycle length or periodicity, and "secondary amenorrhea" referred to the absence of menses for more than two consecutive cycles after the postmenarchial development of regular cycles. Although the outpatient group had four patients with normal menses while the inpatient group had none, the difference between the groups was not signifi-

<table>
<thead>
<tr>
<th>Mean Value (±SD)</th>
<th>Outpatient Group ( (n = 11) )</th>
<th>Inpatient Group ( (n = 6) )</th>
<th>( t = )</th>
<th>( p = )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at presentation (years)</td>
<td>16.3 ± 1.2</td>
<td>16.0 ± 1.4</td>
<td>.44</td>
<td>.65</td>
</tr>
<tr>
<td>Age at onset (years)</td>
<td>14.7 ± 1.4</td>
<td>14.2 ± 1.2</td>
<td>.72</td>
<td>.49</td>
</tr>
<tr>
<td>Length of disorder (years)</td>
<td>1.6 ± 1.1</td>
<td>2.1 ± 1.5</td>
<td>.66</td>
<td>.53</td>
</tr>
<tr>
<td>Weight at visit (kg)</td>
<td>59.8 ± 12.4</td>
<td>49.8 ± 7.1</td>
<td>2.11</td>
<td>.05</td>
</tr>
<tr>
<td>Percent DBW at visit</td>
<td>104.4 ± 16.9</td>
<td>90.3 ± 4.0</td>
<td>2.52</td>
<td>.02</td>
</tr>
<tr>
<td>Serum amylase (Somogyi units/dl)</td>
<td>117.6 ± 109.5</td>
<td>151.4 ± 106.9</td>
<td>.57</td>
<td>.58</td>
</tr>
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</table>
cant when subjected to a t-test approximation of the Mann-Whitney test ($t = 1.64, p > .05$) (Moses, Emerson, & Hosseini, 1984).

The occurrence of emesis was similar in the two groups. All six of the patients in the admission group admitted to vomiting 1-10 times a day. Of the 11 patients in the outpatient group, 8 also vomited at least once daily, whereas 2 reported emesis every 2-3 days, and 1 patient denied it entirely. Analysis of serum amylase values (Table 1) showed no difference between the groups that would suggest a difference in severity of emesis. Laxatives were used by 5 of the 11 outpatients and by 2 of the 6 inpatients. Use of diuretics or appetite suppressants was denied by all patients. A careful and detailed dietary history in each patient failed to reveal any unusual intake of carrots or other foods high in carotene.

Physical findings were minimal. Two patients in each group had abrasions or calluses on the dorsum of their hands, two in each group had some mild, nontender parotid swelling, and one in each group appeared to have excessive carious disease. No patient had a carotenemic appearance of her skin.

Of major interest, the two groups of patients were clearly divided by their serum carotene levels at the time of their initial outpatient evaluation. In our hospital, the normal range of carotene is 80–275 μg/dl. As seen in Table 3, the inpatient group had a mean serum carotene level of 164.8 μg/dl, whereas for the outpatient group it was 309.0 μg/dl. This difference was highly significant on analysis by Student's t-test with the Bonferroni modification for multiple comparisons ($t = 3.92, p = .002$).

Serum cholesterol levels were obtained on 4 members of the inpatient group, who had values ranging from 186 to 211 mg/dl, and on 10 outpatients, with values ranging from 169 to 203 mg/dl. There was no significant difference between the groups ($t = 0.28, p = .69$), and the values were all within the normal range for adolescents in our laboratory (120–210 mg/dl). Because of specific

Table 2. Menstrual function in two groups of patients (see text for details).

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Irregular</th>
<th>Secondary Amenorrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outpatients</td>
<td>4</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Inpatients</td>
<td>0</td>
<td>4</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3. Serum carotene levels in two groups of bulimic patients

<table>
<thead>
<tr>
<th>Serum Carotene (μg/dl)</th>
<th>Outpatient Group ($n = 11$)</th>
<th>Inpatient Group ($n = 6$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>309.0</td>
<td>164.8</td>
</tr>
<tr>
<td>SD</td>
<td>92.0</td>
<td>59.2</td>
</tr>
<tr>
<td>Range</td>
<td>183–510</td>
<td>76–243</td>
</tr>
</tbody>
</table>

Note: $t = 3.92, p = .002$. 
clinical indications, thyroid studies were obtained on two inpatients and two outpatients. All levels of T4, T3, and thyroid-stimulating hormone were within normal limits.

DISCUSSION

In previous reports on carotenemia in eating disorders, the emphasis has always been on its presumed relationship with malnutrition. Pops and Schwabe (1968) felt that the carotenemia they observed was probably associated with nutritionally related hypercholesterolemia, whereas Robboy et al. (1974) believed that it was due either to intake of foods high in carotene or, more likely, to altered metabolism of vitamin A. Parenthetically, both studies failed to separate classical, restrictive-diet anorexia nervosa from cases where bingeing and purging occurred. Curran-Centano et al. (1985) did differentiate bulimia as a specific category of eating disorder in their study of carotenemia and vitamin A status. However, all their bulimic subjects were underweight, being at a mean of 79.7% of ideal body weight, almost identical to their anorexia nervosa group. Both groups had elevation of a wide range of serum carotenoids, with a concomittant decrease of thyroid hormones T4 and T3. The authors suggested that there was a progression of metabolic alterations with the severity of the eating disorder.

The patients in this report directly contradict the relationship of carotenemia to malnutrition. A group of adolescent patients with bulimia but without low weight was shown to have a mild increase in serum carotene. The presence of normal cholesterol levels further argues against a primary role of malnutrition. Normal thyroid function in four of our patients, confirming the earlier work of Mitchell and Bantle (1983), is further evidence against a nutritional mechanism. Also, the fact that another very clinically similar group of patients in our study had normal serum carotene levels, despite being at a lower percentage of DBW, strongly suggests that the effects of mild malnutrition did not play a primary role in the hypercarotenemia we observed.

It is possible that the hypercarotenemia of eating disorders may be associated with amenorrhoea. Frumar et al. (1979) reported on hypercarotenemia in six women with hypothalamic amenorrhoea but without evidence of anorexia nervosa. Interestingly, the amenorrhoea followed stress or weight reduction in each case, and so the patients may really have had what is now recognized as bulimia (Russell, 1979; Casper et al., 1980; Garfinkel et al., 1980). In any event, there was no readily available explanation for the carotenemia. Kemmann, Pasquale, and Skaf (1983) also reported a series of amenorrheic women with carotenemia and ascribed the elevated levels to diets high in carotene or vitamin A. The bulimic patients in the present study were all interviewed by a dietitian, and such diets were not reported. Also, amenorrhoea or less severe forms of menstrual dysfunction occurred in a similar proportion both in our outpatient and inpatient groups, providing clear evidence that menstrual function did not play a primary role in the elevation of carotene in the outpatient group.

Heavy exercise is also commonly seen in patients with eating disorders and might have some association with carotenemia. Our patients all probably engaged in at least a moderate degree of exercise, and one would have expected
the inpatient group also to exhibit some elevation of carotene if exercise had a close relationship with the phenomenon. Furthermore, other workers have measured carotene levels in eumenorrheic, oligomenorrheic, and amenorrheic long-distance runners and found normal results in all three groups (Richards, Chang, Bossetti, Malarkey, & Kim, 1985).

There is no ready explanation for the very unexpected findings reported here. It is theoretically possible that carotenemia is a marker for some unknown biochemical process occurring in a specific subgroup of eating disorders. Although it is still controversial whether or not bulimia is truly distinct from anorexia nervosa, it seems likely that if a biochemical marker identified a milder population of one disorder, it would also do so in the other. If this supposition were true, it would be difficult to explain the fact that the carotenemia reported in patients with severe classical anorexia nervosa with profound weight loss is at a much higher level than in the present series. Serum carotene levels of 600–1800 µg/dl have been described in anorexia nervosa (Pops & Schwabe, 1968; Robboy et al., 1974).

Another intriguing theoretical possibility exists, although at a speculative level. Instead of being a marker for a pathophysiologic process contributing to the clinical manifestations of an eating disorder, carotenemia might in fact be associated with some sort of homeostatic mechanism activated in response to the disorder itself. The patients reported here can be considered to have a mild eating disorder with regard to weight and physical findings. Perhaps the carotenemia observed in our outpatient group is associated with some response that is also manifested by a better clinical status allowing ambulatory management. Our inpatient group may require more intensive therapy because of a failure to activate the postulated protective mechanism. The higher carotene levels observed in other patients with much more severe clinical disease may indicate more activity of this mechanism, albeit futile, in response to a higher level of activity of the underlying pathological process responsible for the eating disorder. If our preliminary findings are confirmed in a larger, prospectively designed study, and especially if serum carotene levels are shown to vary in a predictable fashion with patient status over time, it may prove useful to consider this hypothesis in greater detail.

REFERENCES


Carotenemia in Normal-weight Bulimia


