

TREATMENT OF ARTERIOSCLEROSIS AND VAGUE ABDOMINAL DISTRESS WITH NIACINAMIDE HYDROIODIDE (WITHOUT SIDE-EFFECTS)

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THE CLASSIC clinical syndrome of "arteriosclerosis" has not altered since Virchow described his "endarteritis deformans" (1). But many more individuals now suffer from the disease, because of greater longevity and an aging population, which has been brought about by increasing public health measures and advances in specific medical treatment (2).

Controversy over the various causes and mechanisms has placed emphasis first on one etiological factor then on another. Virchow (1) felt that it was an inflammatory process. Marchand considered it an "essentially degenerative and hyperplastic" change in the arteries. MacCallum seems to be the first to mention the fat accumulation (3) which theory has given rise to low-fat, low-cholesterol treatments.

Whatever theory holds it is well known that with advancing age the arteries lose elasticity. Associated with this change the muscularis tends to atrophy and undergo fibrous tissue replacement. These alterations generally begin in the thirtieth or fortieth year of life and become well marked ten or twenty years later.

Modern writers (4) regard "arteriosclerosis" as atheromatosis—the process in which arteries become less elastic, characterized by formation of soft atheromatous plaques containing cholesterol, which may later become infiltrated with calcium salts. This vascular change causes the clinical symptoms usually associated with "arteriosclerosis." Diminution of blood supply to particular vital organs, such as brain, heart, kidneys, or gastrointestinal tract (13) may cause manifestations of mental aberration, coronary disease, or vague abdominal distress. The age of appearance of such local disorders depends on genetic factors. These changes are characterized by a vast complex of clinical symptoms. The syndromal picture is often complicated by the simultaneous occurrence of hypertension.

Vague abdominal distress is one of the most difficult of the clinical syndromes associated with the arteriosclerotic process to treat. This functional gastrointestinal disorder is revealed by changes in motility, secretion, and the inability to properly absorb foods to maintain normal nutrition (5).

It has been popular to regard the syndrome of vague abdominal distress rather lightly as being of psychic origin. While psychic changes may occur simultaneously, vague abdominal distress may be caused by changes in the vegetative nervous system and by uncoordinated and abnormal functions of the gastrointestinal tract (6, 7). These changes become more

severe with increasing severity of the arteriosclerotic process. No particular gastrointestinal symptom should be emphasized as indicative of the syndrome. Because of the vague nature of the syndrome various series tend to emphasize different individual symptoms (8). Among the symptoms particularly noted are, increased motility, nausea, flatulence, diarrhea, belching, constipation, epigastric pain, and spasticity.

MEDICATION USED

The medication used was Niacinamide Hydroiodide in combination with iodides. This has been found most conveniently given in the form of tablets* supplying 50 mg. of Niacinamide Hydroiodide and 270 mg. of Sodium Iodide per day (2 tablets). In some cases this dose has been given in multiples of two or three times without untoward effect. In many cases it has been desirable to begin medication with large intravenous doses of Niacinamide Hydroiodide 100 mg. and Sodium Iodide 1 gram** twice weekly. This has been continued for one or two months after which tablets were given to maintain medication.

CASE SUMMARY

The average patient in the group of fifty-nine cases (from which essential hypertension was eliminated) had a blood pressure of 149/87. This was unaffected by the medication.

While there were a few overweight individuals in the group the average patient was within normal limits. The average weight was 149 pounds.

Twenty-six cases out of fifty-nine, showed the classic sign of arcus senilis. There were thirty-six cases of the fifty-nine, which demonstrated various degrees of enlargement, sclerosis, and calcification of the aortic arch. Rubra was present in 18 cases. There was no change after treatment.

Forty-five of the fifty-nine cases showed varying degrees of vague abdominal distress. This was completely relieved in thirty cases, partially relieved in nine cases, and unchanged in six cases.

Fifty-five of the fifty-nine cases had varying degrees of dizziness. After medication dizziness persisted in only sixteen cases—clearing in thirty-nine cases.

Thirty-three of the group exhibited chronic headache. After medication headache persisted in only thirteen cases.

*The combination was supplied as Iodo-Niacin in tablet and ampule form by The Cole Chemical Company of St. Louis, Mo. The tablet formula was Sodium Iodide 135 mg. and Niacinamide Hydroiodide 25 mg.

**The ampule formula was: Each 5 cc. contains—Niacinamide Hydroiodide 100 mg., Sodium Iodide 1 Gram.

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Disturbed orientation was exhibited by twenty-four cases. Twelve of these were relieved by medication.

Excessive fatigue was experienced by **fifty-one** of the fifty-nine cases. After treatment **thirty cases** still complained of excessive fatigue.

Before retest, each patient had been on medication for three months. Improvement persisted for over one year during medication in all cases.

DISCUSSION

Improvement in the clinical syndrome of "arteriosclerosis" has thus been maintained in a clinical series. Medication with Niacinamide Hydroiodide was sustained, but there were no signs of iodism or other untoward effects.

Because of the close chemical relationship of iodine and bromine (9) the same mechanism is postulated for iodism as for bromism. Iodism and bromism (10) are caused by poisoning of co-enzyme I and II (11). These co-enzymes function in the promotion of cellular oxidation. This poisoning is prevented by Niacinamide Hydrobromide and, in the present medication, by Niacinamide Hydroiodide, which supplies the integral part of the molecule of di-and-tri-phosphopyridine (12) (co-enzymes I and II).

Iodism, bromism, and pellagra are caused by poisoning or deficiency of the di-and-tri-phosphopyridine nucleotides. Iodism, bromism, and pellagra have characteristic signs and symptoms referable to the skin. The iodides particularly, however, cause catarrh of the respiratory passages. Iodism may arise from comparatively small quantities of iodide and is most commonly seen when administered repeatedly (13). The exposed areas are most affected. Acneform lesions, erythematous patches, or papular eruptions together with **gastric distress** are most common. Iodism is often so severe that it precludes the use of the ordinary iodine salts (13). However, no such disagreeable consequences have been experienced with the use of Niacinamide Hydroiodide in combination with iodides.

Recent research has explained how iodism, bromism, and pellagra are related to porphyrinurea (14, 15, 16, 17) by impairment of the co-enzyme mechanisms.

This report shows the clinical counterpart of the above noted chemical and laboratory relationships. Clinical findings demonstrate that Niacinamide Hydroiodide when used alone or in combination with metallic iodides has not caused iodism or untoward symptoms in any of this series of more than fifty cases.

Thirty-three females and 36 males, ranging in age from forty-three to eighty-four, with an average of sixty-one years were treated and observed for more than a year. No case developed respiratory catarrh, skin eruptions, or any other untoward reaction to the Niacinamide Hydroiodide whether used alone or in combination with sodium iodide.

CONCLUSIONS

1) The signs and symptoms of "arteriosclerosis" as a clinical entity are reviewed.

2) The beneficial effects of iodide treatment combined with Niacinamide Hydroiodide on the signs and symptoms of arteriosclerosis including vague abdominal distress have been shown.

3) The rationale of the use of an iodide containing the integral portion of the molecule of the co-enzymes to prevent iodism in the form of Niacinamide Hydroiodide is explained.

4) The mechanism of iodism is discussed and correlated with bromism and pellagra as a dysfunction of the co-enzyme oxidation mechanism.

5) Absence of iodism, or any untoward effects from Niacinamide Hydroiodide is shown in a large clinical series treated for over a year.

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