Fibromuscular Dysplasia, Antiovulant Drugs, and Ergot Preparations

To the Editor:

Fibromuscular dysplasia of the carotid arteries was not described by the founders of pathology or of neurology, and not recognized clinically until 1965. Personal experience with 6 female patients, as well as reports in the literature, leads me to suggest that the condition is not rare, and a recent article in STROKE reports 25 patients. The cause for increased recognition of fibromuscular dysplasia, in both carotid and renal vessels, may be the increased use of angiography; but the etiology of fibromuscular dysplasia remains unknown.

Review of 21 articles reporting cases of fibromuscular dysplasia of the carotid vessels confirms the statement by Iosue and Stanley that 80 to 85% of cases are females, and several articles since that of Hartman in 1971 have suggested that antiovulant or estrogen medications can be causally related. Three of 6 patients we have seen had received ergot preparations.

Internists and neurologists are well aware of limb ischemia and vascular occlusion induced by prolonged use of ergot preparations in susceptible individuals. The entity of ergotamine headache has also become increasingly recognized, and it is clear that migrainous agents all seem suitable approaches in the medical management of such cases.

It can thus be hypothesized that fibromuscular dysplasia is the result of inappropriate multiplication of cells in certain muscular arteries, and that such proliferation is enhanced by antiovulants. In addition to pregnancy, hormones, hypertension, stretch, and genetic predisposition, the use of ergot preparations may also enhance such proliferation in susceptible individuals. The entity of ergotamine headache has also become increasingly recognized, and it is clear that migrainous agents all seem suitable approaches in the medical management of such cases.

The vulnerability of the carotid and renal arteries to fibromuscular dysplasia has been attributed to the relative dependence of these vessels on nutrition from the vaso vasmor originating from the lumen of these muscular arteries, in contrast to other vessels which have a more stable vascular supply. Stanley et al. have further suggested that regions of particular stretch, such as the right renal or both carotid arteries, are particularly vulnerable to episodic ischemia. An additional concept possibly applicable in the explanation of fibromuscular dysplasia is the monoclonal hypothesis which suggests that the proliferating cells of an early arteriosclerotic plaque stem from one mutated cell. Such cells may be stimulated by ischemia, toxins, or stretch; and the effect of estrogen is to enhance such neoplastic proliferation of smooth muscle cells.

It might be well for us to review briefly some of the problems involved and point out to the authors and the readers of this column why we feel that Drs. Easton and Sherman have contributed something in a negative fashion. Although it would appear superficially that this is a well-prepared manuscript, it has all the appearances of a basket of fruit containing disparate members of a particular genus; i.e., apples, pears and whatnot. It is surprising to me that the editors found this article worthy of publication.

Having made some serious criticisms, it is incumbent upon us now to be constructive.

Starting at no particular place in the communication, the mere statement that there is similarity of results among 11 Board certified neurological and vascular surgeons has no bearing on the matter whatsoever. The statement that these men are Board certified does not attest in any way to their skill or knowledge of the subject; merely that they sat for their Boards and are probably competent to a certain degree in their specialty. It does not otherwise indicate their particular skills in the management of carotid artery disease.

Patient selection is fairly straightforward and has been delineated by many of the authors referred to in the References. It's important to know whether we're dealing with a patient who has progressive TIAs, stable TIAs and asymptomatic stroke. Here it would seem that the symptoms and the timing would be important factors.

Oculoplethysmographic studies will give us an idea of whether there is a stenotic component associated with the TIAs, whether 80 to 85% of cases are females, and several articles since that of Hartman in 1971 have suggested that antiovulant or estrogen medications can be causally related. Three of 6 patients we have seen had received ergot preparations.

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