We report a 43-year-old woman who presented with a right frontoparietotemporal ischemic stroke. She had been diagnosed with Turner’s syndrome during childhood and had a history of chronic estrogen therapy. Cerebral angiography showed lesions characteristics of fibromuscular dysplasia involving the right internal carotid and right vertebral arteries. We are not aware of any previous reports describing an association between fibromuscular dysplasia and Turner’s syndrome. Although chronic estrogen therapy cannot be ruled out as a cause of this patient’s stroke, we suggest a possible etiologic relation between these two entities.

**Discussion**

Fibromuscular dysplasia is an unusual disease affecting predominantly women in the fifth decade of life. It most commonly affects the renal and internal carotid arteries, but many other vessels can be involved. The underlying pathogenesis is fibroblast-like transformation of smooth muscle cells, leading to the characteristic vessel wall changes.

Fibromuscular dysplasia is diagnosed angiographically, the most striking feature being the classic “string-of-beads” stenosis. In our patient we observed this pattern at the middle segment of the extracranial right internal carotid artery and at the
middle portion of the extracranial right vertebral artery.

A variety of entities (including coarctation of the aorta) have been described in association with fibromuscular dysplasia, but not, to our knowledge, with Turner's syndrome. On the other hand, Turner's syndrome, one of the most common forms of human aneuploidy (45 chromosomes, XO), has also been associated with coarctation of the aorta. This mutual association raises the question of a possible common mechanism accounting for both types of stenotic lesion.

Several hypotheses for the etiology of fibromuscular dysplasia have been proposed. The congenital hypothesis suggests that a vascular malformation explains the characteristic location of this lesion at the middle third of the carotid artery. The importance of genetic factors is also supported by the high familial incidence of fibromuscular dysplasia and the high prevalence of associated entities in individual patients. In this context, an association between fibromuscular dysplasia and Turner's syndrome, as in our patient, might be further evidence of the importance of genetic factors in the etiology of the former.
The humoral hypothesis\textsuperscript{12} is based on histopathologic studies in women taking oral contraceptives whose vessels show structural and histochemical changes in the intima and media similar to those found in fibromuscular dysplasia. Our patient was receiving chronic estrogen therapy, which might have played a role in the causation of fibromuscular dysplasia or stroke. Arterial stretching during extension and rotation of the head (the mechanical hypothesis) may also result in changes of the vessel wall compatible with fibromuscular dysplasia,\textsuperscript{9} as can a decreased blood supply to the vascular wall due to occlusion of the vasa vasorum (the ischemic hypothesis).\textsuperscript{13–15} There was no evidence of either mechanical or ischemic mechanisms in our case.

Our patient had Turner's syndrome, was taking oral contraceptives, and developed a stroke. Angiography showed changes characteristic of fibromuscular dysplasia. A relation among these four factors is difficult to establish since stroke can occur in otherwise healthy women taking oral contraceptives and in women with fibromuscular dysplasia alone. However, it is interesting to postulate a genetic predisposition leading to an association between Turner's syndrome and fibromuscular dysplasia, with hormonal factors perhaps contributing to the stroke.

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Turner's syndrome, fibromuscular dysplasia, and stroke.
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