Transient Compression of the Left Innominate Vein

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UNILATERAL distention of the neck veins is an uncommon but important clinical finding. In almost all instances it is indicative of disease in the superior mediastinum, particularly aneurysm of the aorta.\(^1\) Recently, however, we encountered a patient in whom overfilling of the left external jugular veins developed as a consequence of compression of the left innominate vein by an elongated but otherwise apparently normal thoracic aorta. The case is believed to be the third without aneurysm in which the exact mechanism of such compression was demonstrated angiographically and the first in which subsequent dye contrast study showed the phenomenon to be transient.

Case Report

An 83-year-old Negro was admitted to the Ben Taub General Hospital on March 16, 1963, because of progressive swelling of his feet and legs, orthopnea, increasing dyspnea on exertion, and weight gain of 20 pounds. There was no history of chest pain or myocardial infarction. He had had hypertension for 5 years and had been treated with various antihypertensive agents.

On physical examination the patient's blood pressure was 220/140 mm. Hg in both arms, and the pulse was 90 per minute and regular. The left external jugular veins were distended, but the veins on the right side of the neck appeared normal. Scattered expiratory wheezes were heard bilaterally. The apical impulse of the heart was palpable at the anterior axillary line in the sixth intercostal space. No murmurs were heard. A non-tender, smooth, hepatic edge was palpable 4 cm. below the right costal margin in the midclavicular line. Both lower extremities were moderately edematous to the level of the groin. The remainder of the physical examination was within normal limits.

The hematocrit value, total leukocyte and differential count, urinalysis, blood urea nitrogen, and fasting blood sugar were normal. Blastomycin, coccidioidin, histoplasmin, and tuberculin (first and second strength P.P.D.) skin tests were negative. Electrocardiographic findings included nonspecific S-T and T-wave changes in the left precordial leads. Venous pressure in the right and left antecubital veins and in the left femoral vein was 70, 130, and 65 mm. water, respectively. Decholin arm-to-tongue circulation time was 23 seconds on the left and 16 seconds on the right. Roentgenogram of the chest revealed generalized cardiac enlargement, minimal pulmonary vascular engorgement, widening of the superior mediastinal shadows, and a high aortic arch (fig. 1). Phlebograms made via injection into the left antecubital and external jugular veins revealed that the left innominate vein was compressed and 2.8 cm. wide (0.5 to 1.8 cm. normal width)\(^2\) (fig. 2). By contrast, the right innominate vein was shown by similar technic to be normal in configuration and only 1.4 cm. wide.

Treatment consisting of rest in bed, antihypertensive drugs, diuretics, and digitalis resulted in rapid resolution of the peripheral edema and return of the blood pressure to normal levels. Con-

Figure 1

Posteroanterior roentgenogram of chest on admission.

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Figure 2

Phlebogram showing widening and compression of left innominate vein (black arrows) by aorta (dotted arrows).

comitant with clinical improvement, dilatation of the left external jugular veins disappeared and the venous pressures and circulation time became equal and normal in both arms. Six weeks after the initial studies, roentgenogram of the chest showed the heart, lungs, and upper mediastinum to be normal in appearance. At this time the width of the left innominate vein was observed on phlebogram to have decreased to 1.9 cm. (fig. 3). Shortly, thereafter, the patient was discharged from the hospital.

Discussion

In 1934 Sabathie described prominence of the left external jugular vein as a “peripheral” sign of a diseased thoracic aorta obstructing inflow into the left innominate vein. He observed the sign about 60 times in patients in whom a high aortic arch resulted from systemic hypertension, aortitis, atherosclerosis, or aneurysm. In 1957 Bruwer et al. reported a patient with systemic hypertension in whom unilateral venous obstruction in the left arm was shown by angiocardograms, phlebograms, and subsequent thoracotomy to be caused by pressure on the left innominate vein from an elongated but otherwise normal thoracic aorta. In 1960 Smith referred to such left-sided cervical venous distention as the sign of “the kinked innominate vein.” He recalled having seen “scores” of people with this condition and recounted his experience with five patients, all of whom had systemic hypertension without aortic aneurysms. Cortes, in 1962, alluded to three patients with the sign but described only one of them in detail. The patient had systemic hypertension and distention of the left external jugular vein. Roentgenogram of the chest showed widening of the superior mediastinum. Venous angiocardogram demonstrated a tortuous aortic arch pressing the left innominate vein against the sternum.

The sign of the compressed innominate vein is brought out best by having the patient lie in the supine position with the head of the bed elevated to an angle 45° above the horizontal. The patient should face forward while his neck is inspected in good light and the cervical veins on each side are compared. Sabathie, however, recommended having the patient turn his head away from the side being examined. If the sign is not seen easily, it may be accentuated by having the patient exhale forcefully.

The pathogenesis of the sign becomes apparent from the anatomy of the superior mediastinum. The left innominate vein is

Figure 3

Phlebogram after therapy demonstrating decrease in size of left innominate vein.
bordered anteriorly by the sternum and infero-posteriorly by the arch of the aorta. Thus, if the aortic arch is displaced upward, it may press the left innominate vein against the sternum (figs. 4 and 5). Such compression is facilitated by the fact that the mediastinal veins have thin walls and are filled with blood under low pressure.

Compression of the left innominate vein by the thoracic aorta should be considered when there is overfilling of the left external jugular veins, normal filling of the right external jugular veins, elevated venous pressure and prolonged circulation time in the left but not in the right arm, and the presence on roentgenogram of the chest of an aneurysm or mere elongation of the aorta without distinct evidence of mediastinal or pulmonary disease. Even though an increase in the venous pressure in the left arm is characteristic, visible venous engorgement of the extremity need not be present. The diagnosis is confirmed by phlebography. The compression is visualized best on a roentgenogram of the chest taken in the left anterolateral projection after the administration of contrast material into the left antecubital or external jugular veins. Concomitant visualization of the aortic arch by venous angiography is ideal but unnecessary, since the aorta usually is readily visible anyway.

It should be emphasized that the combination of overfilled left external jugular veins and widening of the superior mediastinum naturally suggests a mass occluding the left innominate vein. Unless one knows that these findings may also be caused by an elongated

Figure 4

Aorta (A) pressing left innominate vein (V) against sternum.

Figure 5

Lateral view of drawing shown in figure 4. S, sternum; V, left innominate vein; A, aorta.
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thoracic aorta, extensive investigations, including thoracotomy, may be carried out unnecessarily.4

Finally, the transient nature of the left-sided cervical venous distention in the case reported here deserves comment. Presumably, when the systemic hypertension was relieved, dilatation and elongation of the aortic arch diminished, and the blood flowed unimpeded through the left innominate vein. The role played by cardiac decompensation is difficult to assess. The right innominate vein was normal angiographically before cardiac failure and hypertension were controlled, but its size was not evaluated after maximal therapeutic benefit had been achieved. It is possible, therefore, that in the present patient, the elevated central venous pressure accompanying congestive heart failure may have been a contributory factor in maintaining compression of the left innominate vein by the thoracic aorta.

Summary

A patient with left-sided cervical venous distention associated with systemic hypertension and congestive heart failure is presented. Angiographic study showed venous return from the left external jugular veins to be impeded by an elongated but otherwise normal thoracic aorta compressing the left innominate vein. Once the hypertension and cardiac failure were controlled, compression of the left innominate vein regressed and the cervical venous engorgement disappeared.

References

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