Case Report

Aortic Pain Associated with Deteriorated Aortic Elastic Properties: An Early Manifestation of Aortopathy

CHARALAMBOS VLACHOPOULOS, NIKOLAOS ALEXOPOULOS, ELEFTHERIOS TSIAMIS, CHRISTODOULOS STEFANADIS

Peripheral Vessels Unit, 1st Cardiology Department, Athens Medical School, Hippokration Hospital, Athens, Greece

With the exception of acute aortic dissection, the aorta is not widely considered as a symptom-producing organ. However, chest pain may be of aortic origin in the absence of dissection; deteriorated aortic elastic properties may facilitate the occurrence of symptoms. We describe chest pain of aortic origin in a 60-year-old patient with a stiff, dilated but non-dissected aorta. Attention to aortic elastic properties may help exploration of the etiologic and mechanistic environment in which aortic pain occurs, thus identifying a special subgroup of patients who require special monitoring and therapeutic approaches.

Address:

Charalambos
Vlachopoulos

*17 Kerassoundos St.,
115 28 Athens, Greece*
e-mail:
cvlachop@otenet.gr

With the exception of acute aortic dissection, the aorta is not widely considered as a symptom-producing organ. However, chest pain may be of aortic origin, especially in the presence of aortopathies. Functional abnormalities of the vessel manifested as deteriorated aortic elastic properties precede overt aortopathy and may facilitate the occurrence of symptoms.

Case description

A 60-year-old patient with a history of hypertension and no other risk factors for coronary artery disease suffered from chest discomfort on exertion or emotional distress. The pain was constricting, retrosternal, radiating at the back with variable duration of no less than 5-10 minutes. Standard clinical examination and laboratory testing was negative for cardiovascular diseases. Transthoracic and transesophageal echocardiogram showed a moderately dilated ascending aorta with no evidence of other morphological changes of the aorta. The patient was further investigated for coronary ischemia. On exercise stress testing he completed a maximal Bruce protocol with no electrocardiographic evidence of ischemia; however, the symptom was reproducible when systolic pressure exceeded 200 mmHg. Thallium-201 stress myocardial perfusion imaging was negative. Coronary angiogram showed normal coronary arteries and aortography showed a normal aortic valve and a moderately dilated ascending aorta (4.1 cm) with no morphological evidence of dissection. Subsequently, the elastic properties of the aorta were studied at baseline and during handgrip isometric exercise (for 3 minutes at 50% of the maximal voluntary contraction).

The investigation of aortic function was approved by our institution's Ethical Committee and written informed consent was obtained from the patient.

Evaluation of elastic properties

Aortic elastic properties were evaluated by calculating *aortic distensibility* and by deter-
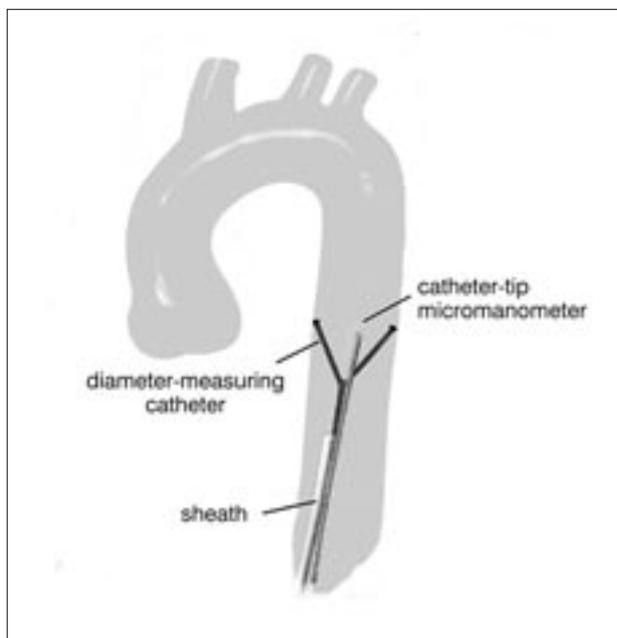
Figure 1. Schematic representation of the diameter and pressure catheter positioned at the same point of the thoracic aorta.

mining *pressure-diameter relation* using a high-fidelity technique that was developed in our laboratory and has been previously validated.[1,2] In brief, the technique is as follows: Instantaneous aortic diameters were measured in the descending thoracic aorta by a Y-shaped intravascular catheter (Cordis Europe, Cat. No. 5RER-060) that was developed in our laboratory and uses sonometry to measure diameter. Aortic pressures were obtained simultaneously at the same point of the aorta by a catheter-tip micromanometer (Model SPC-320 Millar Instruments). Both catheters are inserted into the aorta through a long introductory 8F sheath (Figure 1). A VF-1 mainframe (Crystal Biotech) was fitted with appropriate modules for measuring aortic diameter and pressure and acquiring an electrocardiogram. The digitized data were stored and processed using commercially available software (Microsoft Excel for Windows).

Aortic distensibility (= 2 × [systolic - diastolic aortic diameter] / [diastolic aortic diameter x pulse pressure]) is an accurate index of elastic properties. Large distensibility values represent improved aortic elastic properties, and small values represent deteriorated properties. The *aortic pressure-diameter loop* is derived by plotting digitized data for pressure (ordinate) and the simultaneously acquired diameter (abscissa) during a cardiac cycle. Diameter lags behind pressure as a re-sult of the viscoelastic nature of the aortic wall. Thus, this plot assumes the shape of a clockwise hysteretic loop, the ascending portion of which corresponds to systole and the descending portion to diastole. A loop with a steeper slope denotes reduced elastic properties, since for a given change in pressure the vessel responds with a relatively small change in diameter. Moreover, pressure-diameter relation provides valuable insights regarding the mechanisms involved in the changes in aortic function. The pressure-diameter loop operates along a hypothetical line of elasticity. Changes of blood pressure without any changes in the intrinsic properties of the vessel cause sliding of the loop along the same hypothetical line of elasticity (passive changes), whereas changes involving the intrinsic elastic properties of the vessel (active changes) are characterized by shifting of the loop to another hypothetical line of elasticity.[1,2] Our method provides a reliable and accurate determination of pressure-diameter relation in conscious humans, overcoming several methodological limitations of current techniques.

Results

Although the patient remained asymptomatic during diagnostic cardiac catheterization, he developed chest pain (like that previously experienced) during handgrip exercise when his systolic blood pressure reached a level above 200 mmHg. No electrocardiographic evidence of ischemia was observed during the manifestation of symptoms. Nor was there any metabolic evidence of ischemia, since the myocardial lactate extraction ratio (100 × [arterial lactate - coronary sinus lactate] / [arterial lactate]) remained practically unchanged (baseline: 23%; during symptoms: 22%). The pain resolved spontaneously when the blood pressure returned to values below the "threshold" of 200 mmHg. Aortic distensibility was 0.71×10^{-6} cm^2.dyne^{-1} at baseline and decreased by 39% on handgrip (0.43×10^{-6} cm^2.dyne^{-1}). Aortic pressure-diameter loops slid along the same line of elasticity with handgrip, acquiring a steeper slope (Figure 2).

Discussion

Aortic pain mechanisms

The aorta can be a chest pain producing organ, even in the absence of dissection, and deteriorated aortic elastic properties may facilitate the occurrence of symptoms. Apart from the specialized sensory tissue of the
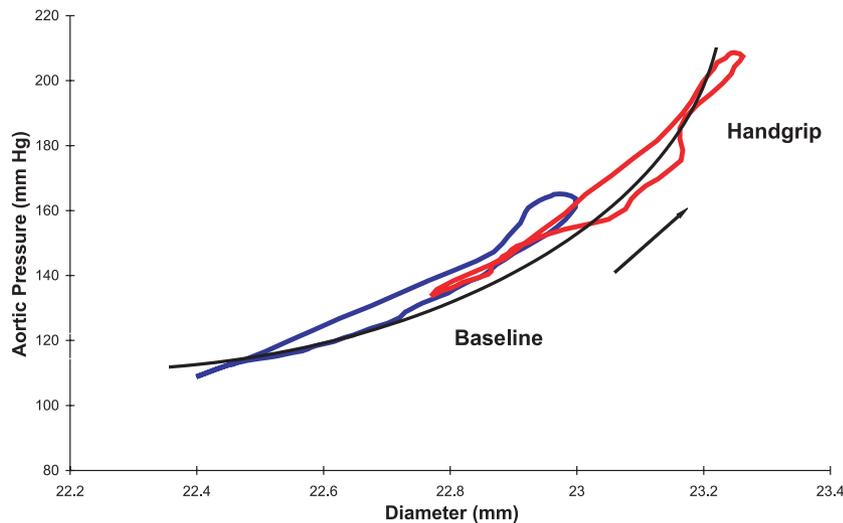
Figure 2. Pressure-diameter loops at baseline and during handgrip exercise. During handgrip, the loop has a steeper slope, indicating deteriorated elastic properties. However, during handgrip, the loop has slid along the same hypothetical line of elasticity (black line - passive change of aortic elastic properties).

aortic arch (the baroreceptors and the chemoreceptors) the aorta has a network of pain fibers. Afferent neurons from the aorta pass to the sympathetic chain ganglia from the aortic plexus, traverse the rami communicantes, and reach their cell bodies in the dorsal root ganglia. Early experimental studies showed that distension of the aorta and application of stimulating substances to the outer surface causes pain.[3] In the clinical setting, pain is observed during balloon angioplasty of aortic coarctation. Studies have proved that stretching of the coronary wall by stent expansion or balloon dilatation during angioplasty causes chest pain.[4] Patients with Marfan syndrome, with aortic root dilatation and without histopathologic evidence of aortic dissection, often complain of chest pain. Thoracic pain is also the commonest symptom encountered in patients with annuloaortic ectasia without evidence of aortic dissection. Aortic dilatation and stretching are likely mechanisms in this subset of patients. Another mechanism of aortic pain may be ischemia of the aortic wall due to the compression of the lumen of the vasa vasorum, the nutrient vessels of the aorta, by the increased wall tension.[5]

In the absence of myocardial ischemia, distension and stretching of the aorta during pressure elevation appears to be a very convincing cause of pain in the patient described in this report, who had a dilated aorta (and hence increased tension according to Laplace's law) and altered aortic elastic properties even at baseline. The predicted distensibility for a patient with his demographic characteristics, derived from a cohort of 200 patients studied in our department, was 1.32×10^{-6} $cm^2 \cdot dyne^{-1}$. Elastic properties deteriorated further with isometric exercise. Thus, stretching of a stiff aorta that cannot accommodate the increase in blood pressure might be the cause of pain.

Implications

Aortic pain may be an early manifestation in the natural history of aortopathy, denoting dysfunction of the vessel at a stage that precedes structural disorders such as aortic dissection. Aortic elastic properties are important determinants of left ventricular function, coronary blood flow and ventricular-vascular coupling.[6-9] Chronically reduced elastic properties lead to an increase in pulse pressure. This increase in pulsatile stress leads to mechanical fatigue of the aortic wall, predisposing to dilation and dissection. Moreover, a stiff aorta cannot accommodate any transient increase in stroke volume, increasing pulse pressure disproportionately and leading to further stretching of the aorta. Since aortic pain may result from ischemia of the aortic wall, it may represent an early stage of the process that leads to structural changes, including medial degeneration and necrosis.

Thus, identification of the aorta as the source of chest pain may outline a subset of patients that constitute a special nosologic entity, with its own natural history, that requires special monitoring and possibly re-orientation of therapeutic approaches in an effort to alter the disease process.

References

1. Stefanadis C, Stratos C, Vlachopoulos C, et al: Pressure-diameter relation of the human aorta. A new method of determination by the application of a special ultrasonic dimension catheter. Circulation 1995; 92: 2210-2219.

2. Stefanadis C, Vlachopoulos C, Tsiamis E, et al: Unfavorable effects of passive smoking on aortic function in men. Ann Intern Med 1998; 128: 426-434.

3. Wooley CF, Sparks EH, Boudoulas H: Aortic pain. Prog Cardiovasc Dis 1998; 40: 563-589.

4. Jeremias A, Kutscher S, Haude M, et al: Nonischemic chest pain induced by coronary interventions. Circulation 1998; 98: 2656-2658.

5. Stefanadis C, Vlachopoulos C, Karayannakos P, et al: Effect of vasa-vasorum flow on structure and function of the aorta in experimental animals. Circulation 1995; 91: 2669-2678.

6. Nichols WW, O'Rourke MF: McDonalds' Blood Flow in Arteries: Theoretical, experimental and clinical principles, 4th edition. Edward Arnold (Publishers) Ltd, London, 1998; pp 293-316 & 418-438.

7. Vlachopoulos C, O'Rourke MF: Genesis of the normal and abnormal arterial pulse. Curr Probl Cardiol 2000; 25: 297-368.

8. Vlachopoulos C, Dima I, Aznaouridis K, et al: Acute systemic inflammation increases arterial stiffness and decreases wave reflections in healthy individuals. Circulation 2005; 112: 2193-2200.

9. Vlachopoulos C, Aznaouridis K, Stefanadis C: Clinical appraisal of arterial stiffness: the Argonauts in front of the Golden Fleece. Heart 2005, Dec 9: [Epub ahead of print].