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**CASE REPORT**

**PATHOLOGY/BIOLOGY**

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# Death Related to Aortic Coarctation in a Young Female During Sexual Intercourse

**ABSTRACT:** Coarctation of the aorta (CA) is diagnosed mainly in pediatric patients, and therapy is conservative if asymptomatic, but surgical treatment is required if advanced arterial hypertension is present. Moderate to severe forms contraindicate any type of physical activity requiring cardiac effort. Here, we describe the first documented death of an apparently healthy 35-year-old woman because of cardiac tamponade by rupture of an aortic aneurysm, possibly related to congenital CA, prolonged use of oxymetazoline hydrochloride, and physical and/or emotional stress during sexual activity. Our patient was asymptomatic for classical CA symptoms. The patient's breathing difficulties likely in hindsight were due not so much to nasal congestion, but rather to an ineffective oxygenation of the blood from the abnormal heart. In an attempt to treat the "nasal disease," the patient ingested chronic and excessive doses of decongestants, aggravating her fatal disease. The danger of inhaling large doses of nasal decongestants without an appropriate medical indication is highlighted here.

**KEYWORDS:** forensic science, sudden death, aortic coarctation, sexual intercourse, nasal decongestants, hypertension

Coarctation of the aorta (CA) is a congenital constriction or narrowing of the aorta between the arterial branches of the upper and lower body; it accounts for 5–10% of all congenital heart conditions (1).

CA is of variable position, extent, and severity and may be associated with other congenital abnormalities such as ventricular septal defects or, more frequently, bicuspid aortic valve (in about 85% of cases) (1).

CA is often regarded as a simple and isolated congenital anomaly, but is more correctly interpreted as part of a diffuse arteriopathy, with a propensity for aneurysm formation and dissection remote from the coarctation site (1).

Hypertension occurs in many patients with coarctation. However, the explanation for the increase in blood pressure remains uncertain. The three theories are mechanical (obstruction increasing arterial resistance), neural (obstruction resetting carotid baroreceptors), and renal (ischemia of the kidneys) (2).

This disorder is diagnosed mainly in pediatric patients, and physical examination can provide important clues to this diagnosis. Characteristically, there may be upper limb systolic hypertension, while systolic blood pressure measured in the legs is lower, but commonly diastolic blood pressure levels are similar, and the latter have values that fall within the normal range, unless another concomitant cause of diastolic blood hypertension is present. Left ventricular heave or ejection systolic murmur may also be present (3).

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Dyspnea, fatigue, and cold feet are the most common symptoms of CA. Vascular dysfunction characterized by diminished brachial dilatation to nitroglycerin, increased pulse wave velocity, and reduced compliance have been reported in the literature, but young adults presenting with coarctation typically have no symptoms.

Mild CA allows patients to engage in physical effort involving moderate heart commitment. However, greater degrees of CA contraindicate most daily activities to prevent cardiovascular complications. CA requires surgical treatment if at least a moderate degree of arterial hypertension is present.

The natural history of this disease is death because of such complications as heart failure, aortic aneurysm formation and rupture, ischemic heart disease, cerebrovascular accidents, and bacterial endocarditis.

## Case Report

Here, we describe the first documented case of a death of a 35-year-old woman afflicted with an undocumented CA. This woman had shown no previous signs of CA and lived a normal life. However, one day during sexual intercourse, she was in distress. An ambulance was immediately called to transport her to the hospital, but she was dead on arrival. A forensic autopsy was requested by the Prosecutor.

The past medical history of the woman was unremarkable, except for a reported difficulty in breathing that she treated using an excessive daily intake of oxymetazoline hydrochloride nasal spray (a potent alpha-1-adrenergic agonist commonly used to vasoconstrict blood vessels in the nasal mucosa).

Autopsy revealed hemopericardium because of rupture of a large aneurysmal dilatation of the ascending aorta (Fig. 1) associated with CA in the first segment of the thorax, bicuspid aortic valve (Fig. 2*A*), concentric left ventricular hypertrophy, and an increase

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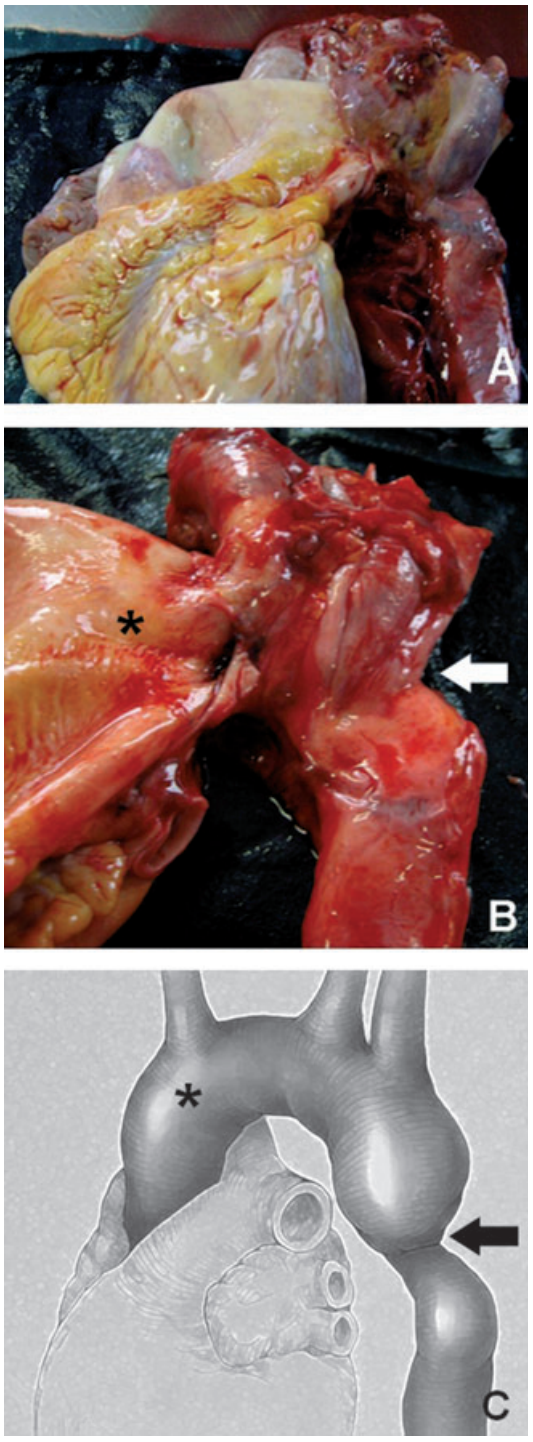


FIG. 1—*(A) Macroscopic specimen showing heart and thoracic aorta; (B) close up on aortic coarctation; and (C) diagram showing heart and thoracic aorta (*on the aortic aneurysm, arrow on the coarctation).*

in volume and size of the intercostal arteries (Fig. 2*B*). The anterior descending coronary artery and right coronary artery contained atherosclerotic plaques. Examination of the other organs was unremarkable. In the toxicological analyses performed on heart blood, urine, and bile, no common drugs of abuse were detected.

The cause of death was established as cardiac tamponade because of rupture of a large aneurysmal dilatation aorta, probably related to chronic hypertension caused by congenital CA, prolonged

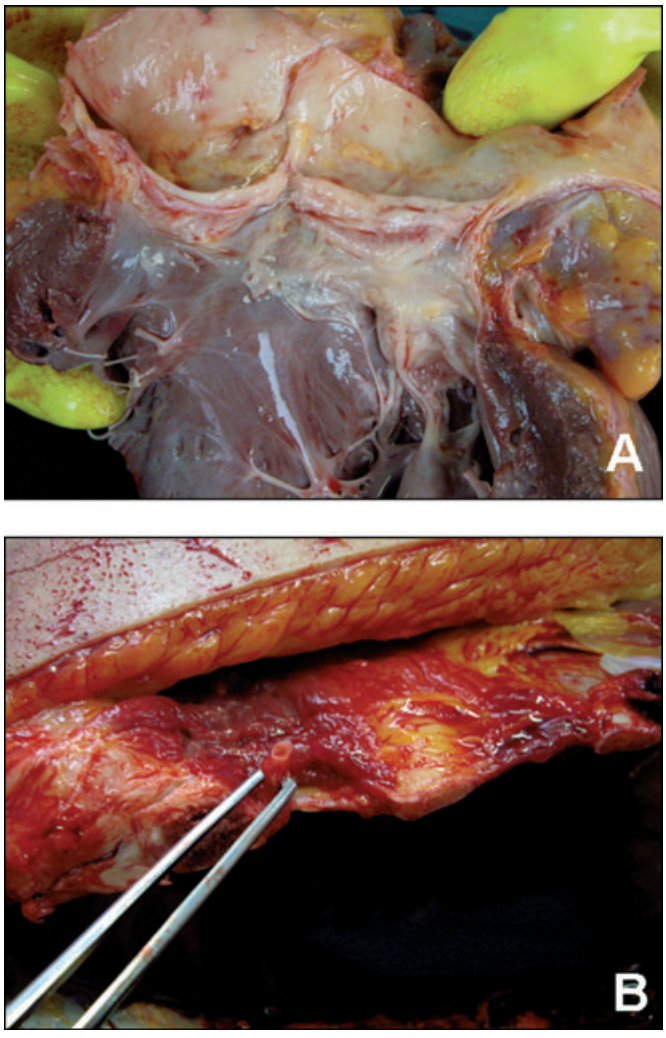


FIG. 2—*(A) Macroscopic specimen showing bicuspid aortic valve; and (B) macroscopic specimen demonstrating an increase in volume and size of the intercostal arteries.*

use of oxymetazoline hydrochloride, and physical and/or emotional stress during sexual activity.

## Discussion

The annual incidence of sudden cardiovascular death during sexual intercourse is estimated to be 1.9 per 1000 autopsies for men and 0.16 per 1000 autopsies for women. However, the cause of sudden death related to sexual activity remains obscure (4).

Lee et al. (5) reported that coronary artery disease and subarachnoid hemorrhage with ruptured berry aneurysm were important causes of sudden death related to sexual activity.

We described an autopsy case of a man dead because of rupture of a cerebral artery aneurysm with subsequent subarachnoid hemorrhage after sexual intercourse. Toxicological analysis demonstrated that he had consumed a drug (Sildenafil) prior to sexual activity (6). Like any form of physical exertion, sexual intercourse increases heart rate and blood pressure (7).

During orgasm, the heart rate can rise to 110–180 beats per minute and the respiration rate to 40 breaths per minute. Systolic blood pressure increases 20–60 mmHg before it reaches its plateau phase. Diastolic pressure is 10–20 mmHg higher. Rate-pressure product

and oxygen consumption increase only *c*. 25%, so that sexual activity is regarded as a mild- to moderate-intensity activity (7). However, this physical and emotional situation may bear certain health risks, particularly for patients with cardiac disease.

As shown in various studies, severe cardiovascular diseases do not prevent patients from engaging in sexual activity. The risk appears to be less pronounced in established marital relationships. However, sexual activity with an extramarital partner, especially in an unfamiliar setting, can result in increased emotional stress, more anxiety and sexual arousal, and increased sympathetic activation with all its cardiovascular consequences. To confirm this, Parzeller et al. (8) showed that the majority of deaths associated with cardiovascular events occurred during extramarital intercourse.

Therefore, it seems important to draw the phenomenon of coital death out of the taboo sphere and to inform patients with CA about prodromes and the risk of their condition in relation to any form of physical and/or emotional stress.

Our patient was asymptomatic for classical CA symptoms until the fatal event. The patient's breathing difficulties likely in hindsight were due not so much to nasal congestion, but rather to an ineffective oxygenation of the blood from the abnormal heart. This is supported by the autopsy showing a concentric left ventricular hypertrophy.

Oxymetazoline is a potent nonselectively alpha-adrenergic agonist commonly used to vasoconstrict blood vessels in the nasal mucosa because vascular beds widely express $\alpha 1$ receptors. Upon nasal, oxymetazoline hydrochloride decreased nasal congestion because it increases the diameter of the airway lumen and it reduces fluid exudation from postcapillary venules. Otherwise, systemic application of $\alpha 2$ agonists causes vasodilation, because of centrally mediated inhibition of sympathetic tone via presynaptic $\alpha 2$ receptors (9). The danger of inhaling large doses of nasal decongestants without an appropriate medical indication is highlighted here.

In particular, a chronic and excessive use of oxymetazoline hydrochloride through the highly vascular nasal mucosa can lead to an increase in systemic vascular resistance, and consequently, an increase in blood pressure (greater diastolic values) and reflex bradycardia secondary to stimulation of baroreceptors located in the carotid artery and aorta (10).

The predominance of the alpha-adrenergic effects and the lack of chronotropic properties make reflex slowing of the heart rate particularly evident with this drug, to the extent that oxymetazoline has been used to end attacks of paroxysmal atrial tachycardia.

Söderman et al. (11) described a variety of central nervous system and cardiovascular reactions after oxymetazoline 0.01–0.05% nose drops were administered to five patients aged 5 weeks to 5 years. Symptoms included agitation, excitement, insomnia, convulsions, and vasoconstriction.

An aneurysmal dilatation of the aorta such as that witnessed here typically requires an etiology of prolonged hypertension, affecting both systolic and diastolic blood pressure. However, in CA, the blood pressure between the upper and lower body artery branches usually differs in maximal but not minimal values, with the latter falling within the normal range.

A high blood concentration of oxymetazoline hydrochloride produces a diastolic hypertension. In our patient, this drug was an important causal factor for aneurysm because this decongestant, as shown by the studies of Westerveld et al. (12) is also able to completely inhibit the induction of inducible nitric oxide (NO) synthase. The latter is an NO-producing enzyme, and NO is an important biological modulator that plays a significant role in vasodilation and blood pressure control. Diastolic pressure is well established to be influenced mainly by arterial tone, unlike systolic pressure, which better reflects cardiac contractility.

In conclusion, the large aneurysmal dilatation of the ascending aorta of our patient, the failure of which was the cause of her death, was likely the result of a chronic state of hypertension, which in turn was caused by congenital CA and an excessive intake of oxymetazoline hydrochloride.

In this case, we consider sexual intercourse "the final straw that broke the camel's back," that is, it precipitated the fatal occurrence from a situation that was already highly unstable.

The situation was exacerbated by our patient being unaware of her dangerous cardiovascular condition, with her mild respiratory symptoms being mistakenly attributed to nasal congestion. In an attempt to treat the "nasal disease," the patient ingested chronic and excessive doses of decongestants, aggravating her fatal disease.

Greater efforts should be directed toward research of the relationship between sexual activity and cardiovascular disease to avoid a repeat of this tragic outcome. Better information about commonly prescribed drugs to treat CA is needed.

## References

1. Warnes CA. The adult with congenital heart disease: born to be bad? J Am Coll Cardiol 2005;46:1–8.
2. Prisant LM, Mawulawde K, Kapoor D, Joe C. Coarctation of the aorta: a secondary cause of hypertension. J Clin Hypertens 2004;6:347–50.
3. Lynch MJ, Woodford NW, Dodd MJ. Sudden death due to aortic rupture complicating undiagnosed coarctation of the aorta in a teenager—a case report and review of the literature. J Forensic Leg Med 2008;15:443–6.
4. Parzeller M, Bux R, Raschka C, Bratzke H. Sudden cardiovascular death associated with sexual activity. A forensic autopsy study (1972–2004). Forensic Sci Med Pathol 2006;2:109–14.
5. Lee S, Chae J, Cho Y. Causes of sudden death related to sexual activity: results of a medicolegal postmortem study from 2001 to 2005. J Korean Med Sci 2006;21:995–9.
6. De Giorgio F, Arena V, Arena E, Lodise M, Valerio L, Chiarotti M. Subarachnoid hemorrhage during sexual activity after Sildenafil intake: an accidental association? Am J Forensic Med Pathol 2011. In press.
7. Cheitlin MD. Sexual activity and cardiac risk. Am J Cardiol 2005;96: 24M–28M.
8. Parzeller M, Raschka C, Bratzke H. Sudden cardiovascular death in correlation with sexual activity—results of a medicolegal postmortem study from 1972–1998. Eur Heart J 2001;22:610–1.
9. Katzung BG, Masters SB, Trevor AJ. Basic and clinical pharmacology, 11th edn. New York, NY: The McGraw Hill Companies, Inc., 2009.
10. Thrush DN. Cardiac arrest after oxymetazoline nasal spray. J Clin Anesth 1995;7:512–4.
11. Söderman P, Sahlberg D, Wiholm BE. CNS reactions to nose drops in small children. Lancet 1984;10:573.
12. Westerveld GJ, Voss HP, van der Hee RM, de Haan-Koelewijn GJ, den Hartog GJ, Scheeren RA, et al. Inhibition of nitric oxide synthase by nasal decongestants. Eur Respir J 2000;16:437–44.

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