Acute Fatal Asphyxia Due to Aortic Aneurysm in Patient with Four Saccular Aneurysms of Thoracic Aorta: Case Report

GARFIELD S. BARNET, M.D.† and ARTHUR S. GLUSHEN, M.D.††
Aspinwall, Pennsylvania

Acute asphyxiation as the cause of death in patients with aneurysm of the thoracic aorta is rare. Ficarra,¹ in reporting a case with chronic compression of the trachea and right lung by an aneurysm of the ascending aorta, with death attributed to asphyxia resulting from chronic ischemic and anoxic anoxia, stated that “clinical and pathological literature on aneurysms fail to report any instance of asphyxia due to aortic aneurysms.” Pernet² reported an instance of mechanical asphyxia due to gigantic aneurysm. No other reference to acute asphyxiation has been encountered, although it is well known that mechanical pressure on the trachea and resultant respiratory difficulties are common in aneurysm of the thoracic aorta.

Compression of the trachea by aortic aneurysm is most apt to occur when the aneurysm is situated in the transverse portion of the arch. Lucke and Rea³ found compression of the trachea by two of 23 aneurysms at the junction of the ascending and transverse arch, and by five of 46 aneurysms of the transverse arch. Keefer and Mallory⁴ state that tracheal compression usually results from pressure by aneurysm of the transverse arch or of the innominate artery.

In cases of aneurysm of the ascending or transverse arch, Kampmeler⁵ found dyspnea to occur in 65 per cent and to be due most often to tracheal, bronchial, or pulmonary compression. Boyd⁶ found dyspnea reported as the first or chief complaint in 31 per cent of cases of aneurysm of the thoracic aorta, and that more severe and steadily increasing dyspnea resulted from pressure on the trachea or large bronchi. Several authors⁶⁻⁷ refer to a paroxysmal type of dyspnea, of unknown causation, but rarely seen in the absence of pressure on the trachea or large bronchi. Changes in posture may precipitate a feeling of suffocation.

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²†Resident, Internal Medicine. ††Chief, Cardiology Section, Veterans Administration Hospital, Aspinwall, Pennsylvania.
Kampmeier\textsuperscript{4} noted respiratory stridor in 11.7 per cent, and "choking spells" in 7.3 per cent of 205 patients with aneurysm of the transverse arch; such findings were much less frequent in cases with aneurysm of the ascending or descending arch. Of 247 patients with thoracic aortic aneurysm who died, he found 46 (18 per cent) died of respiratory obstruction. Keefer and Mallory\textsuperscript{4} mention that gradual suffocation may result from retention of tracheal and bronchial secretions. None of these writers makes mention of acute asphyxiation, as was dramatically observed in the case to be described.

The occurrence of multiple saccular aneurysms of the thoracic aorta is not infrequent, but the precise incidence cannot be determined from the data in the literature. Paullin and Minnich\textsuperscript{5} state that multiple aneurysms are fairly frequent, there being one fairly large saccular dilatation accompanied by many smaller ones. Wilburne and Taylor\textsuperscript{6} reported two patients who had three and one patient who had four aneurysms of the thoracic aorta. Lucke and Rea\textsuperscript{10} in a study of 268 necropsied cases with aneurysm, found multiple aneurysms (chiefly of the aorta, but occasionally an aortic aneurysm associated with aneurysm of one of the aortic branches) in 53 (19.7 per cent). In a more detailed study,\textsuperscript{3} but where the data given do not permit exact analysis, they indicate that at least one patient had three or more aneurysms. The figures of Colt,\textsuperscript{11} which may be weighted because they were based in large part on published case reports, reveal an incidence of 57 cases (10 per cent) of multiple aneurysms among 575 patients with thoracic saccular aortic aneurysm, two aneurysms being present in 43 cases, three in seven, four aneurysms in four, and more than four in three patients. Kampmeier\textsuperscript{4} found 23 cases (3.6 per cent) with two or more sacs among 633 patients with aneurysm of the thoracic aorta, and at autopsy found five cases with two sacs and three with three aneurysms.

According to Stokes and associates,\textsuperscript{12} small sacculations and finger aneurysms result from localized bulging at spots weakened by fibrosis due to inflammatory changes around the vasa vasorum. In cases of aneurysm, the systolic blood pressure tends to be low and the diastolic high. An intact aortic valve, by permitting a constant and even pressure on the aortic wall, favors the production of aneurysmal dilatation, while the development of aortic regurgitation tends to protect against the formation of aneurysm. Kampmeier\textsuperscript{4} states that hypertension, in the absence of aortic insufficiency, may aggravate the tendency to saccular dilatation.

The present report describes a patient who died of acute asphyxia when an aortic aneurysm suddenly occluded the tracheal
lumen. This occlusion was observed bronchoscopically, yet at necropsy the tracheal lumen, though narrowed, was not occluded, suggesting that the tracheal obstruction during life was due to dynamic dilatation of the aneurysmal sac. An incidental finding was the presence of three smaller aneurysms of the thoracic aorta.

CASE REPORT

A 56 year old farmer entered the Veterans Administration Hospital, Aspinwall, Pennsylvania, on the evening of August 6, 1947. He complained of dyspnea of one year’s duration, most intense in the last three months, associated with progressively severe hoarseness of six months’ duration and a marked cough. He slept on two pillows and had experienced several episodes of nocturnal paroxysmal dyspnea. His weight had declined from 135 lbs. to 110 lbs. There was no chest or back pain, and no edema or palpitation. Syphilis was denied by name and symptoms.

On examination, the patient revealed evidence of recent weight loss. He was slightly dyspneic and orthopneic at rest and extremely hoarse. His cough was of metallic, rasping quality. The neck veins were slightly distended. The pupils were irregular and unequal, but reacted to light. The fundi could not be examined. The trachea lay in the midline. The point of maximum cardiac impulse was felt in the sixth interspace within the midclavicular line. Dullness to percussion extended beyond the sternum in the left second interspace. Grade IV systolic and diastolic, high pitched aortic murmurs were heard over the entire precordium. A Durozey murmur and pistol shot sound were heard over the femoral artery. Marked capillary pulsation was present in the nail beds. The blood pressure was 120/40. No pulsus alternans or gallop rhythm was present. The lungs were hyperresonant and coarse bronchial rales were heard bilaterally. The liver extended one fingerbreadth below the costal margin but was nontender. The peripheral vessels appeared thickened and tortuous. The knee jerks and ankle jerks could not be elicited. No penile scar was apparent. There was no edema.

Laboratory Data: The blood Kahn and Wassermann tests were positive. The blood count revealed 3,190,000 red blood cells and 18,400 white blood cells per cu. mm. (neutrophils 84 per cent, lymphocytes 16 per cent); hemoglobin 10 gm. per 100 cc. The urine contained a faint trace of albumin, frequent white blood cells, one to two red blood cells per h.p.f., and rare granular casts; the specific gravity was 1.021.

On the day following admission, while drinking through a straw, the patient suddenly gagged or choked. Following this, he became extremely cyanotic, but the pulse remained of good volume at a rate of 80 per minute. There was suprasternal retraction, and the patient was using his accessory muscles of respiration. The breath sounds were markedly depressed in all lung fields. The initial clinical impression was tracheal obstruction. An endotracheal tube was inserted, and suction yielded considerable quantities of clear mucus without foreign material. The vocal cords appeared grossly normal. One hundred per cent oxygen was administered by mask with slight decrease in cyanosis. The blood pressure was 240/69. The endotracheal tube remained in situ for approximately one-half hour during which time the patient appeared somewhat less dyspneic. After removal of the tube, intensification of the cyanosis ensued. Questioning of the patient at this point revealed that he had
experienced one previous similar episode. A chest x-ray film showed cardiac enlargement, and dilatation of the aorta compatible with aneurysm. The respiratory obstruction progressed relentlessly as evidenced by increasing sternal retraction and the use of all accessory muscles of respiration. The patient appeared to be in extremis, and emergency bronchoscopic examination was performed. The trachea was seen to be markedly deviated to the right and almost completely occluded at its lower end by an extrinsic pulsating mass, believed to be an aneurysm. Marked pulsation of the aorta was transmitted to the bronchoscope. The patient became semicomatose and remained so until his demise three hours after the onset of the acute episode. No morphine had been administered.

**Necropsy Findings:** The necropsy was performed five hours after death. The relevant findings were as follows: The pupils were round, but unequal, the right measuring three mm., the left four mm. in diameter. The trachea appeared slightly deviated to the right on palpation. The skull and its contents appeared normal. Both lungs were wet, the left weighing 550 gm., the right 600 gm. The left lower and right upper lobes presented irregular purplish-red areas of doughy consistency. There was no evidence of foreign matter in the tracheobronchial tree.

The heart was hypertrophied, weighing 500 gm. The left ventricular wall measured 20-22 mm. in thickness, the right five mm. The aortic valve revealed slight cord-like thickening of the free edge of the posterior cusp, the commissures being widened to two mm. The valve circumferences were as follows: T.V. 13 cm., P.V. 7 cm., M.V. 10 cm., A.V. 8 cm.

**FIGURE 1:** The aneurysm impinging on the trachea lies opposite the origin of the great vessels. The arrow on the right lies in the opening of a second sac. The other arrow points to the tiny third sac. The fourth aneurysm lies to the right of the upper arrow.
Thickened, puckered intima of the sinuses of Valsalva produced moderate narrowing of the coronary ostia. The branches of the coronary arteries were widely patent, however; the left anterior descending branch showed a few small, yellow intimal plaques.

The thoracic aorta was thickened, inelastic, and widened, measuring 8 cm. in diameter in its ascending portion. The intima of the aorta down to the level of the renal artery was roughened by innumerable flat, yellow and grey-white plaques, between which the surface showed pitting and prominent linear and transverse wrinkling, giving a characteristic tree bark appearance. A remarkable feature of the aorta was the presence of four saccular aneurysms, three juxtaposed in the transverse arch, and one lying in the upper descending aorta (Fig. 1). The most proximal was situated on the posterior surface of the aorta opposite the origin of the left subclavian artery. The opening presented a rolled edge and measured 2.0 x 2.0 cm. in diameter. The sac ballooned to a maximal diameter of 4.0 cm. and a depth of 3.0 cm., and was lined by laminated clot. This aneurysm was firmly adherent to the left bronchus and the left anterolateral surface of the trachea, into which it bulged about 0.6 cm. A second and smaller aneurysm lay adjacent to and slightly distal to the first sac, their orifices being separated by only 0.2 cm. of ragged intima. This sac measured 1.5 x 1.5 cm. in diameter, 0.8 cm. in depth, and did not balloon. Its wall consisted merely of clot and thin adventitia. Above, about 0.4 cm. anterior to, and at a level between the first two aneurysms, there was a tiny third aneurysm, measuring 0.4 x 0.4 cm. in diameter and 0.5 cm. in depth; its base was composed of clot. In the lateral aspect of the upper descending aorta, a fourth saccular aneurysm was found, slightly adherent to the anterolateral surface of the esophagus. The opening measured 1.0 x 1.0 cm. in diameter, and the sac ballooned to a maximal diameter of 2.0 cm., and a depth of 0.7 cm. Its base consisted only of laminated clot. No hemorrhage had occurred from any of the aneurysms.

The liver showed moderate congestion. Both kidneys presented adherent capsules and cortical scarring. The second to sixth thoracic vertebrae, inclusive, were eroded along their left anterior surfaces. This erosion bore no close relation to any of the aneurysms and could not be attributed to them.

Microscopic Findings:

**Lung:** There was patchy bronchopneumonia.

**Trachea:** There was partial ulceration of the epithelium and diffuse infiltration of the immediately underlying lamina propria by many polymorphonuclear leukocytes. The interstitial tissue of the mucous glands was heavily infiltrated by great numbers of plasma cells and lymphocytes, and a small circumscribed area of subepithelial necrosis was evident. The cartilage bars were partially ossified, and their outer surfaces broken. Multinucleated giant cells, large numbers of plasma cells and lymphocytes surrounded these fragments. Beyond the cartilage, an organizing clot lay upon the necrotic and partially fibrosed wall of the adherent aorta. The small arteries of the tracheal wall showed fairly marked intimal thickening.

**Aorta:** The media showed marked disorganization and presented areas of necrosis with disruption of the elastic fibers. Near these areas and throughout the media and adventitia the vasa vasorum were surrounded by masses of plasma cells and lymphocytes. Several of these vessels pene-
trated to the intima, which was irregularly thickened by hyaline fibrosis in which were areas of calcification and atheronecrosis.

**Kidney:** The picture was that of arterial nephrosclerosis.


**Comment**

Since at necropsy the tracheal lumen appeared adequate as an airway, while bronchoscopic examination had revealed the trachea to be almost entirely obstructed by a pulsating mass, the obstruction would seem to have been due to dynamic dilatation of the largest aneurysm during life. One can merely speculate as to the cause of the sudden dilatation. The acute episode began while the patient was drinking through a straw, and it is conceivable that he might have aspirated some liquid, initiating asphyxia, and that the resultant rise in blood pressure produced dilatation of the aneurysm with further asphyxia, and thereby a vicious circle. The elevation in blood pressure and absence of tachycardia shortly after the onset of acute respiratory distress are characteristic of the first phase of asphyxia. The slight relief that was afforded by use of the endotracheal tube and administration of oxygen must have been due principally to aspiration of secretions, since the tube did not pass the site of obstruction.

The presence of four aneurysms in association with aortic regurgitation is unusual. The fact that three of the aneurysms were small was perhaps due to the aortic regurgitation, since enlargement of the aneurysms would not tend to occur in the presence of a lowered diastolic pressure.

**SUMMARY**

Aneurysms of the thoracic aorta commonly press upon the trachea or bronchi with resultant respiratory difficulty. Acute asphyxiation as the cause of death in patients with such aneurysms is rare. The present report describes a patient who died of acute asphyxia when an aortic aneurysm suddenly obstructed the tracheal lumen. This occlusion was observed bronchoscopically, yet at necropsy the tracheal lumen, though narrowed, was not occluded, suggesting that the tracheal obstruction during life was due to dynamic dilatation of the aneurysmal sac. An incidental finding was the presence of three smaller aneurysms of the thoracic aorta of syphilitic origin.
RESUMEN

Los aneurismas de la aorta torácica generalmente causan presión sobre la tráquea o los bronquios con la resultante dificultad respiratoria. Es muy raro que la asfixia aguda sea la causa de muerte en pacientes con tales aneurismas. En este informe se describe un paciente que murió de asfixia aguda cuando un aneurisma aórtico repentinamente obstruyó la luz traqueal. Se observó esta oclusión broncoscópicamente; sin embargo, en la autopsia, aunque la tráquea estaba angostada no estaba obstruida, lo que sugiere que la obstrucción traqueal durante la vida se debió a dilatación dinámica del saco del aneurisma. Un hallazgo comitante fue la presencia de tres aneurismas más pequeños de la aorta torácica de origen sífilítico.

REFERENCES