

Robert E. Lee's Right Ear and the Relation of Earlobe Crease to Coronary Artery Disease



Richard A. Reinhart, MD*

Relation of a diagonal earlobe crease (ELC) to coronary artery disease (CAD) was first reported by Sanders Frank in 1973 more than 100 years after the death of Robert E. Lee. Lee provides a rich description of his symptoms in his letters. Lee lived in a period when scientific medicine was just beginning to develop. CAD was not commonly recognized as a clinical entity by practicing physicians during Lee's lifetime. Numerous studies since Frank's original report have shown how an ELC can add to the predictive probability of CAD in the individual patient. This report provides brief context of the state of medical knowledge of CAD and clinical practice in the 19th century to provide better understanding of Lee's medical history. Photographs of Lee showing diagonal ELC are provided. A review of the medical reports shows the relation of ELC to CAD. Lee's description of his symptoms added to the presence of ELC offer compelling evidence that Lee suffered from ischemic heart disease. In conclusion, a modern observation, the value of ELC in the prediction of CAD, applied to a historical figure provides an example of the importance the observation of an ELC in the evaluation of a patient with suspected CAD and may provide evidence for evaluation of the medical history of other historical figures as well as for our current patients. (Am J Cardiol 2017;120:327–330)

The medical history of historical figures can be difficult to sort out particularly if the person lived before the advent of scientific medicine. Evidence of medical illness often can be gathered only indirectly about a person who writes a description of symptoms but rarely are there objective physical findings available to aid in diagnosis. One such case is that of General Robert E. Lee (1807 to 1870) who lived in what has been described by historians as a time of emergence from the medical Middle Ages when understanding of heart disease was just beginning and when scientific principles were just emerging in the education and practice of physicians.¹ In this study, I present a case report of the medical history of Robert E. Lee derived from the description of symptoms he experienced and from observations made by others that provide evidence supporting the diagnosis of ischemic heart disease beginning when he was 56 years and progressed until his death at age 63 years. Details of Lee's medical history are previously reported.^{2,3} In the case of Lee, there is 1 objective piece of evidence, 1 physical finding, which supports the diagnosis of coronary heart disease and that is 2 photographs of his right profile showing a clear picture of his right earlobe. The state of heart disease and medical practice in the 19th century is reviewed for context.

Case Report

Robert E. Lee died at age 63 years in 1870 while serving as the President of Washington College in Lexington

Department of Medicine, Brody School of Medicine, East Carolina University, Greenville, North Carolina. Manuscript received January 24, 2017; revised manuscript received and accepted April 12, 2017.

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*Corresponding author: Tel: (715) 218-8107; fax: (715) 358-6108.

E-mail address: reinhart@ecu.edu (R.A. Reinhart).

Virginia. Lee first reported cardiac symptoms at the age of 56 years in a letter to his wife, March 27, 1863, "The troops are not encamped near me & I have felt so unwell since my return [Lee arrived from Petersburg March 19, 1863] as not to be able to go anywhere."⁴ "He had not been sleeping well and he contracted a serious throat infection which settled into what seemed to be a pericarditis. His arm, chest, and back were attacked with sharp paroxysms of pain that suggest even the possibility of angina."⁵ Symptoms continued and in a letter to his wife on April 19, 1863, Lee notes, "I am feeble & worthless & can do but little."⁶ After Gettysburg, Lee wrote to Jefferson Davis asking to be relieved of command on August 8, 1863, "I sensibly feel the growing failure of my bodily strength. I have not yet recovered from the attack I experienced last spring. I am becoming more and more incapable of exertion... Everything, therefore points to the advantages to be derived from a new commander..."⁷

These symptoms persisted throughout the war and gradually progressed after Lee's return to civilian life. After the war, Lee moved to Lexington Virginia where he was appointed as the President of Washington College. In a letter to a friend, General Chilton, April 7, 1870, Lee reported progressive symptoms, "I am suffering from an aggravation of the attack I had in '63, just before the Battle of Chancellorsville ...",⁸ and in another letter on April 11, "... I feel no change in the stricture of my chest. If I attempt to walk beyond a very slow gait, the pain is always there."⁹ His pain progressed to rest angina and on April 18, 1870, he wrote, "... I still have the pain in my chest whenever I walk. I have felt it occasionally of late when quiescent, but not badly, which is new."¹⁰

On September 28, 1870, Lee became acutely ill, and he gradually declined and died October 12. His physicians

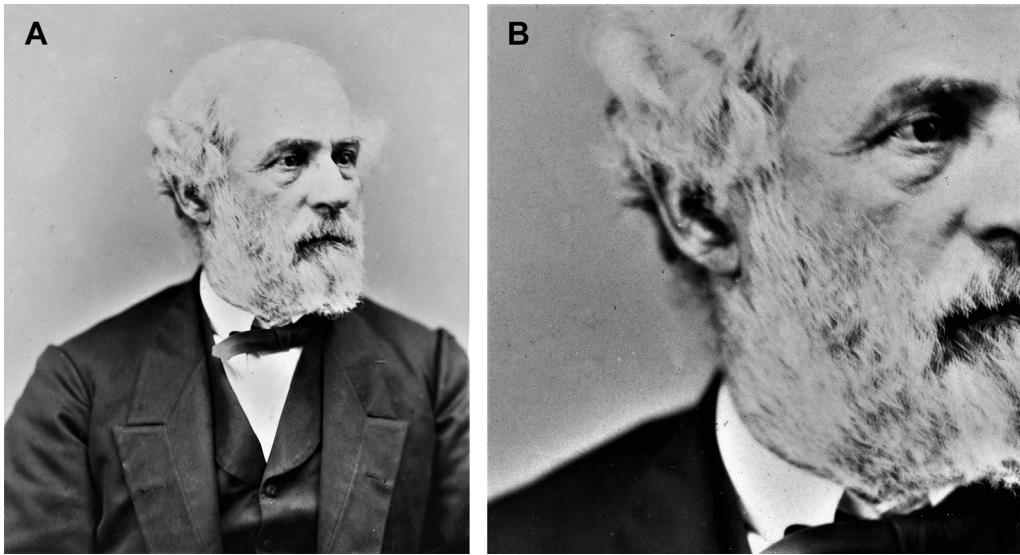


Figure 1. (A) Right profile of Lee 1870 (photograph courtesy of the Library of Congress). (B) Close-up right profile of Lee 1870 (photograph courtesy of the Library of Congress).

described in detail his final illness which included impaired consciousness, accelerated and weak pulse, cool extremities, hurried respirations, and deepening unconsciousness. It was said by his physicians, “General Lee died of a broken heart, and its strings were snapped at Appomattox!”¹¹

Risk factors for coronary artery disease (CAD) were not recognized by practitioners during Lee’s lifetime, but some facts can be derived from historical accounts. There is no account of family history of heart disease. Lee did not use tobacco and drank little alcohol. His lifestyle was vigorous and not sedentary. Diabetes mellitus was recognized in medical practice but was never ascribed to Lee’s medical history.¹²

There is little to report from the standpoint of physical examination during his entire illness aside from occasional mention of his pulse, skin temperature, and respirations. Auscultation of the heart and lungs was a well-established part of physical examination during Lee’s lifetime,¹³ but there are no available reports of such for Lee.

The only evidence on physical examination is contained in 2 photographs (Figures 1 and 2) of Lee’s right profile taken by Boude and Miley in 1869 and 1870, probably one of the last of Lee before his death. Both photographs show a diagonal earlobe crease (ELC).

Discussion

Heart disease in the 19th century was in an early stage of understanding. No tests or technology existed to assist in the diagnosis of CAD. This report demonstrates how new findings can be applied to the diagnosis of historical figures and how this finding is important in the contemporary practice of medicine. Coronary atherosclerosis and myocardial infarction was not recognized as a clinical entity during Lee’s life.¹⁴ William Heberden’s 1772 classic description of angina as a cause of chest pain remains accurate today, but he did not associate angina with disease of the coronary arteries.¹⁵ In 1779, Parry¹⁶ reported the

connection between angina pectoris and CAD. Despite this earlier report, Flint¹⁷ did not make the connection between angina pectoris and CAD in his textbook of diseases of the heart published in 1859. This connection was not made for Lee. Drugs available for the treatment of angina were just being discovered. In 1867, Brunton¹⁸ described the use of amyl nitrite, a vasodilator, for use in angina pectoris. Nitroglycerine was first synthesized in 1846, but its efficacy in angina was not recognized until 1879.¹⁹ Neither of these drugs was made available for Lee.

There is evidence that Lee suffered from ischemic heart disease that had its onset as an acute myocardial infarction in the March 1863. His initial symptoms were atypical, but symptoms of “cold or flu” are often not recognized as an acute myocardial infarction by patients and may lead to delayed treatment.²⁰ The symptoms that Lee describes in the last few years of his life are typical for angina. He remarks that these typical symptoms of angina he experienced later in life were the same that he had at the onset of his illness in 1863 when his symptoms were not as typical. This helps to confirm the diagnosis of symptomatic ischemic heart disease at the onset of his illness in 1863. Typical symptoms of angina in a male are highly predictive of the presence of CAD.²¹ Lee’s symptoms progressed to unstable angina. His final illness likely was recurrent myocardial infarction leading to cardiogenic shock and death.

There is one physical finding Lee possessed that adds to the evidence that he had ischemic heart disease. In 2 photographs obtained close to the time of his death, there is a diagonal ELC. Since Frank’s initial report,²² approximately 120 studies appeared in the medical reports confirmed by a MEDLINE search addressing the relation of ELCs to ischemic heart disease. Most studies confirm the relation, but the mechanism linking the 2 has not been determined. ELCs appear to be an acquired characteristic without any genetic marker ascribed to the diagonal ELC.²³

Shmilovich et al²⁴ evaluated whether incorporating diagonal ELC into the predictive model improved the



Figure 2. (A). Right profile of Lee 1869 (photograph courtesy of the Virginia Historical Society). (B) Close-up right profile Lee 1869 (photograph courtesy of the Virginia Historical Society).

ability of the Diamond-Forrester algorithm to estimate the likelihood of significant CAD. Coronary computed tomographic angiography was used in 430 patients to determine the relation of ELC to CAD. The investigators found that a combined variable of the Diamond-Forrester algorithm plus ELC adds to the ability to detect coronary artery stenoses with greater than or equal to 50% narrowing (odds ratio 5.6, 95% CI 1.6 to 19.8, $p = 0.007$).

Rodriguez-Lopez et al²⁵ studied ELC shapes and cardiovascular events in 1,000 patients. Diagonal creases were defined as those extending from the tragus posterolaterally or inferiorly from the lobe. The medical histories of patient were examined. Multivariate analysis showed an association between ELCs and cardiovascular events. Lee's ELCs match the description of a linear crease starting at the tragus and running posterolaterally and somewhat inferiorly. It is not certain whether Lee has bilateral ELCs because both poses show only the right profile.

A report by Wang et al²⁶ examined the relation between diagonal ELCs and CAD as determined by coronary arteriography in a total of 588 consecutive patients. The authors' conclusion of the results of their study indicated that diagonal ELCs are a readily available means of identifying coronary heart disease. A receiver-operating curve analysis of the ELCs with respect to the detection of CAD showed the sensitivity of the sign was 78%; the specificity was 61%; and the positive predictive value was 89%.

In a meta-analysis by Lucenteforte et al²⁷ included PubMed database search resulting in 120 studies from which 37 studies covering 31,100 subjects meeting their criteria for inclusion in the study. The investigators conclude that ELC could be a marker for CAD. In this study, the combined sensitivity was 0.62 and specificity was 0.67. The investigators cautioned that further study was needed to define the histologic and molecular mechanisms of the relation.

There have been several studies showing no convincing evidence of the relation of ELC to CAD^{28–30} Negative studies appear to be in the minority. It seems reasonable to use the ELC as additive evidence in the daily practice of medicine to help in the prediction of CAD in the individual.

Compared with many cardiac methods, looking for the presence of ELC is inexpensive, noninvasive, and low risk. In the "patient" under consideration in the case report, the addition of the physical sign of ELC to his diagnostic symptoms of angina pectoris lends compelling evidence that Lee experienced symptoms from CAD from the age of 56 years until his death at age 63 years. The clinical course of Lee's symptoms and ultimately his death are characteristics of the natural history of CAD further confirming the diagnosis.

Disclosures

The author has no conflicts of interest to disclose.

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