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Review

Acute myocardial infarction — Historical notes

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ABSTRACT

For the time being, acute myocardial infarction represents a history of success concerning diagnose, management and treatment, whereas it was considered a fatal disease in the beginning of the 1900s. The present paper is aimed at reviewing the landmarks of acute myocardial infarction, as key historical concepts are an important tool for understanding disease management, the daily dilemmas and future perspectives.

1. Introduction

Although description of clinical cases compatible with an acute myocardial infarction (AMI) was reported by William Harvey in the XXVII century literature, the initial recognition of AMI in a living patient was later attributed to Dock, in his paper, "Notes on the coronary arteries", published in 1896 [1]. In 1899, an American pathologist, Ludwing Hektoen, also contributed to the understanding of this pathology by stating "… while cardiac infarction may be caused by embolism, it caused much more frequently by thrombosis, and thrombosis again is usually secondary to sclerotic changes in the coronaries" [1]. Another pathologist, Osler, has, for the first time published a correlation between clinical and pathological findings of coronary thrombosis [2]. Based upon a number of cases from his private practice, he gave a thorough discussion of practically every aspect of angina pectoris reporting several cases of coronary thrombosis. He concluded that a blockage of one of the coronaries by a thrombus or an embolus led to a condition which was known as anemic necrosis, or white infarct [2].

In the beginning of the twentieth century, acute coronary artery thrombosis was not considered as a major health problem even though it was always fatal when it occurred [3]. The whole work of Krehl in 1901, the reports of Obrastzov and Strazhesko in 1910 and the ones of James Herrick in 1912 challenged the paradigm of the inevitable fatality of AMI [4–6]. Their observations described the relationship of symptoms with the sudden or gradual arterial occlusion, the identification of complications of an AMI, such as ventricular aneurysm formation and myocardial rupture, and also established AMI as a clinical entity distinct from angina pectoris [3].

Joseph Wearn, from the Peter Bent Brigham Hospital, Boston Massachusetts, published the first series of 19 patients with a clinical diagnosis of AMI, confirmed at necropsy. After an exhaustive clinical and etiological description, Wearn concluded that an AMI was caused, most likely, by a thrombus in an atherosclerotic left anterior descending coronary artery, mostly affecting fifty-year-old male patients, with sudden chest pain in the heart region. The attack was typically severe and could be associated with other symptoms, such as acute dyspnea. Physical examination usually revealed "signs of the failing circulation, such as pale and cold skin, lung crackles, enlarged heart, pericardial rub and irregular rhythm". Regarding management, a critical concept, noted by Wearn, was that the outcome of the patient would depend upon the extent of the damage in the cardiac muscle and in the conduction system. Treatment should be based upon absolute bed rest, in order to spare the heart and prevent sudden cardiac death. It was also emphasized that in patients with pulmonary rales, fluid intake should be restricted and digitalis given, as this appeared to result in an improvement in symptoms, perfusion, and diuresis. Among the 19 patients, in no case did nitrates relieved chest pain, and usually large doses of morphine were required. Also, he added: "… in very critical times, when blood pressure dropped and patients showed other signs of collapse" caffeine and camphor should be used, as they seemed to provide an almost immediate relief [2].

In 1929, Samuel Levine, from Boston, published another important clinical series of AMI patients. In his book, entitled "Coronary thrombosis: its various clinical features", he presented the concept of cardiac risk factors, namely heredity, male gender, obesity, diabetes and hypertension, as they predisposed to coronary thrombosis. Levine also drew attention to the adverse impact of cardiac arrhythmias during

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an AMI, addressing, probably for the first time, the concept of patient monitoring. He suggested that trained nurses should carefully follow the rate and rhythm of the apex beat, so as to identify and promptly treat arrhythmias. He also noted the use of quinidine for ventricular tachycardia, and adrenaline for AV block [1].

2. Angina pectoris

It is believed that the first description of angina pectoris dates back to 1550BC. In the Ebers Papyrus, Egyptians reported a classic and dramatic description of coronary ischemia: "if thou examinest a man for illness in his cardia and he has pains in his arms, and in his breast and in one side of his cardia… it is death threatening him" — Fig. 1 [7]. More than 3000 years later, in 1768, William Heberden, an English physician, published a clinical description of angina pectoris associated with adverse prognosis and sudden death. The pathophysiologic correlation between angina and coronary artery disease was established only a few years later, in 1786, by Edward Jenner, who witnessed three patients' autopsies with history of anginal attacks, and reported "…a kind of firm fleshy tube, formed within the coronary vessel, with a considerable quantity of ossific matter dispersed irregularly through it…" [1]. Almost a century later, in 1856, Rudolph Virchow, Professor of Pathology at the University of Berlin, was the first to describe the characteristics of the coronary vessel wall in patients with atherosclerosis. He proposed that injury to the inner wall of the blood vessel, possibly caused by fat, might lead to inflammation and secondary plaque formation, a very close notion to present day theory [8]. In 1867, a Scottish physician, Thomas Brunton, introduced the use of nitrates (inhaled amyl nitrate) for the treatment of the angina crises, for he believed that angina was caused by a derangement of the vasomotor system [1].

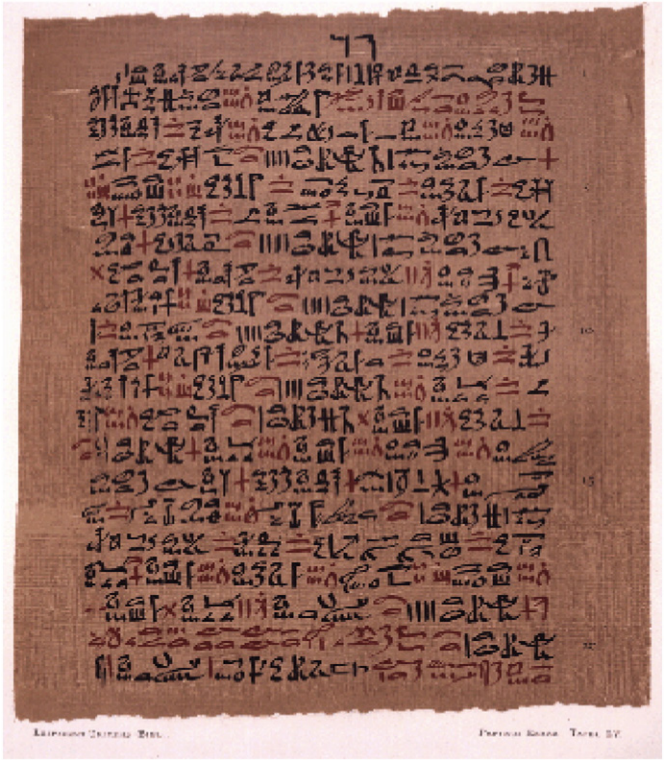

Fig. 1. The Ebers papyrus. In 1862, the Ebers Papyrus was purchased by Edwin Smith, an American adventurer living in Cairo. He kept the papyrus in his possession until 1869 when he placed it on sale. In 1872, the papyrus was purchased by the Egyptologist Georg Ebers, for whom the papyrus is named. The papyrus is 110 pages long, making it the largest medical papyrus discovered ever. The exact date when the papyrus was composed is open to some debate but it probably dates back to around 1534 BCE. The papyrus refers to a myriad of medical spells, treatments, surgeries and diseases that afflicted the ancient Egyptians. In: A small dose of toxicology. The Health effects of common chemicals. Steven Gilbert, 2nd edition, Healthy World Press, 2012 [151].

3. Clinical and electrocardiographic correlations

The origin of modern cardiac electrophysiology dates back to 1840. At that time, an Italian physicist, Carlo Matteucci identified an electrical current that accompanied each cardiac contraction. A few years later, Emil DuBois, a German physiologist, confirmed the discovery of electrical activity in the frog heart. French physicist Gabriel Lippmann devised a capillary electrometer in the early 1870s, that the following British physiologists, John Burdon Sanderson and Frederick Page, used to record the heart's electrical current. In 1878, they reported that each cardiac contraction was accompanied by a two-phase electrical variation, which was the very first description of ventricular depolarization and repolarization. A few years later, Burdon Sanderson and Page published several tracings of the heart's electrical activity recorded with a capillary electrometer. The undulations they registered were later termed as the QRS complex and the T wave. Right about this time, Augustus Waller began a series of experiments, at St. Mary's Hospital Medical School, in London, which culminated in his recording of the first human electrocardiogram. By connecting electrodes attached to the front and back of a man's chest to a capillary electrometer, Waller showed that each heartbeat was "accompanied by an electrical variation" [9].

After attending an international physiology meeting, Einthoven began to explore the use of the capillary electrometer to record minute electrical currents and, in 1895, he was able to detect recognizable waves, which he labeled "P, Q, R, S, and T". Probably one of the most important advances made by Einthoven was the invention of the string galvanometer, in 1903. This device was much more sensitive than both the capillary electrometer previously used by Waller and the string galvanometer that had been invented separately, in 1897, by the French engineer Clément Ader. Rather than using today's self-adhesive electrodes, Einthoven's subjects would immerse each of their limbs into containers of salt solutions from which the electrocardiogram was recorded. With this new technique, he standardized the tracings and formulated the concept of "Einthoven's triangle" by mathematically relating the three leads (Lead III = Lead II − Lead I). He described bigeminy, complete heart block, "P mitrale," right and left and ventricular hypertrophy, atrial fibrillation and flutter, the U wave, and examples of various heart diseases [9,10].

A physician from Chicago, Fred Smith, documented the electrocardiographic changes associated with coronary artery ligation in dogs, and Bousfield described, in 1918, the electrocardiogram during an episode of angina [11]. These findings set the stage for other clinicians, such as Harold Pardee in New York, by the 1920s, to describe the electrocardiographic changes associated with AMI in human beings. He gave rise to the entity of ST-segment elevation myocardial infarction, by describing the electrocardiographic changes of acute coronary occlusion as the takeoff of the T-wave from the descending R-wave [12] — Fig. 2. Thus, by the early of the 20th century, physicians were armed with knowledge on the fundamental clinical and electrocardiographic features of myocardial infarction (MI) and with the belief that sudden coronary thrombosis was the most common immediate precipitant [13].

4. The "Boston chair" treatment of AMI

Until the mid-1950s absolute bed rest deemed essential. Care was largely palliative: to relieve chest pain, to prevent deep vein thrombosis and to ease the breathlessness and edema caused by the falling heart. Patients were confined to strict bed rest for four to six weeks; not allowed to turn from side to side without assistance, and they were even isolated, as well as deprived from trivial activities such as listening to the radio or reading a newspaper. The concept was based upon the