

The Hidden Burden of Premature Cardiovascular Disease in a National Leader: Mustafa Kemal Atatürk's Familial Risk, Lifestyle, and Occupational Stress

To the Editor,

The health of national leaders has always attracted public attention. In contemporary times, advances in medicine and preventive care have enabled many heads of state to live long lives despite stressful demands. However, the disclosure of a leader's health problems remains a sensitive issue, as it may be perceived as a vulnerability with implications for national security and political stability.

Mustafa Kemal Atatürk's health has long been a subject of speculation and historical debate.¹ As a young officer, he managed to lead a defeated country to independence and established a powerful republic under extremely challenging conditions. However, his extraordinary success was accompanied by an extraordinary workload and an unhealthy lifestyle—all of which contributed to a high risk for cardiovascular disease (CVD).

In their recent article published in *Anadolu Kardiyoloji Dergisi*, Köken AH and colleagues² provided a valuable historical–cardiological analysis of Atatürk's CVD, combining archival materials with modern diagnostic frameworks. Building on their contribution, I'd like to add further insights—particularly concerning the early onset CVD—based on additional historical sources, especially Eren Akçiçek's comprehensive book *Atatürk'ün Sağlığı, Hastalıkları ve Ölümü* (Atatürk's Health, Diseases, and Death),³ and aides' diaries,⁴ both of which may allow for a more detailed reconstruction of his clinical course.

While many consultations were documented,³⁻⁴ Atatürk's official medical records were never fully disclosed. His long-time personal physician, Dr. Neşet Ömer İrdelp, left no personal notes and likely destroyed those of other doctors.³ He consistently attributed Atatürk's cardiac symptoms to "fatigue."^{2-3,5-6} This may have been an attempt to conceal the health problems of a national leader, or it may reflect a diagnostic oversight. Either way, the truth remains unknown. Caring for a national leader is rarely straightforward; transparency is often sacrificed for the sake of political continuity. Moreover, physicians themselves may experience ethical dilemmas or professional pressure, at times hesitating to implement what they believe to be the most appropriate medical approach.

A careful review of contemporaneous documents suggests that Mustafa Kemal Atatürk experienced at least 5 coronary episodes between 1923 and 1927.³ Table 1 summarizes his cardiovascular risk profile, clinical course, and therapeutic strategies during each episode.

Accordingly, the first cardiac episode occurred on November 11, 1923, when Atatürk was 42 years old. Only 2 weeks had passed since the proclamation of the Republic on October 29, 1923, and the stressful transition from empire to republic was ongoing. During lunch, Atatürk developed sudden retrosternal chest pain lasting approximately 20 minutes, radiating to the left arm, and accompanied by diaphoresis and marked fatigue.^{2,3} Dr. Refik Saydam, administered morphine,

SPECIAL LETTER TO THE EDITOR

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Table 1. Cardiovascular Risk Factors and Documented Cardiac Episodes of Mustafa Kemal Atatürk (1923–1927)

Predisposing factors					
Family history					
Father, Ali Rıza Efendi, died at age ~46–47, reportedly of a sudden illness (possibly intestinal tuberculosis with CVD)					
Mother, Zübeyde Hanım, died at age 66 from heart failure, likely secondary to chronic aortitis and hypertension					
Life-style & occupational strain					
<ul style="list-style-type: none">• Heavy smoking (30–40 cigarettes/day)• Excessive coffee intake (15–20 cups of Turkish coffee/day)• Chronic sleep deprivation and irregular rest• Prolonged sedentary periods (24–30 hours of desk work)• Intense cognitive workload (mental exertion)• Nighttime eating, often skipping daytime meals• Severe malaria during military service• Chronic pyelitis					
Chronic infections					
Traditional risk factors					
<ul style="list-style-type: none">• Other risk factors (e.g., dyslipidemia, diabetes) not documented• BP readings often >140/90 mmHg, but no formal diagnosis of hypertension					
Cardiovascular Episodes					
Episode No.	Date & age	Circumstances / Triggers	Symptoms & Signs	Likely Modern Diagnosis	Treatment
1	11 Nov 1923 (42 y/o)	Newly elected president; intense political transition just two weeks after the Republic's proclamation	Retrosternal chest pain (~20 min) radiating to left arm, diaphoresis, fatigue BP: 140/90 mm Hg Extrasystoles every 20–40 beats	Acute coronary syndrome (likely NSTEMI)	Morphine for pain relief Low dose iodine therapy for 2 months
2	13 Nov 1923 (42 y/o)	Following lunch & coffee during walking in the garden	Profound fatigue, syncope, shortness of breath.	Post-MI angina possibly accompanied by arrhythmia	Bed rest followed by 50-day convalescence in İzmir; Abstinence from caffeine & tobacco.
3	22–23 May 1927 (46 y/o)	During intense preparation of <i>Nutuk</i> ; ≥24–30 hours of uninterrupted work	Severe chest pain radiating to left arm, diaphoresis, pallor, nausea at night	Acute coronary syndrome (likely STEMI)	Bed rest & diet Morphine for pain relief
4	24 May 1927 (46 y/o)	Two days after the 3 rd episode	Recurrent angina with left arm radiation, sweating, nausea	Post MI angina or reinfarct	Morphine for pain relief Strict bed rest, morphine, prohibition of smoking and alcohol, milk–vegetable diet. Within days, reduced smoking was permitted again.
5	25 May 2027 (46 y/o)	Three days after the 3 rd episode	Lower-intensity but prolonged chest pain BP: 145/90 mm Hg Frequent extrasystoles	Post MI angina ± arrhythmia/pericarditis/PE*	Continued bed rest, Smoking permitted again shortly after
* Pulmonary embolism could not be ruled out due to prolonged immobilization BP, Blood pressure; MI, Myocardial Infarction; NSTEMI, Non–ST–Elevation Myocardial Infarction; STEMI, ST–Elevation Myocardial Infarction; PE, Pulmonary Embolism; y/o, years old					

* Pulmonary embolism could not be ruled out due to prolonged immobilization
BP, Blood pressure; MI, Myocardial Infarction; NSTEMI, Non–ST–Elevation Myocardial Infarction; STEMI, ST–Elevation Myocardial Infarction; PE, Pulmonary Embolism; y/o, years old

which relieved the pain. Physical examination revealed a blood pressure (BP) of 140/90 mm Hg and extrasystoles every 20–40 beats.

Two days later, on November 13, 1923, Atatürk experienced another episode while walking in the garden after lunch and coffee. He reported profound fatigue, followed by syncope and shortness of breath. Eyewitnesses described him as pale and weak, requiring assistance. This event likely indicated a second ischemic episode, possibly accompanied by arrhythmia.

On November 14, 1923, Dr. Neşet Ömer İrdelp examined Atatürk and attributed the episodes to “elemi asabi” (nervous exhaustion), rather than CVD. He recommended strict rest and a change of climate, leading to more than 50 days in İzmir under close observation. Tobacco and coffee were prohibited, and a milk–vege diet was implemented. Atatürk recovered well and returned to near-normal function. However, he soon resumed smoking, coffee intake, and irregular working hours. During this period, he also received a low-dose iodine treatment for 2 months, a now-abandoned practice once believed beneficial for CVD.³

At the age of 46, Atatürk experienced his most severe cardiac event on the night of May 22 and May 23 1927, during the intense preparation of his historical Great Speech, Nutuk. He developed severe retrosternal pain radiating to the left arm, accompanied by diaphoresis, pallor, nausea, and distress while at rest. In the following days, he suffered 2 additional anginal episodes—reportedly less intense but longer in duration—again with retrosternal pain and progressive breathlessness. Witnesses recalled him shouting: “Take this pain away from me!” He was treated with morphine. His BP was 145/90 mm Hg, with frequent extrasystoles.^{2–6}

In June 1927, German professors Friedrich Kraus (Berlin) and Ernst von Romberg (Munich) were invited to examine Atatürk. Their report noted a normal cardiac exam and a negative Wassermann test (used to exclude syphilitic aortitis, a routine consideration in CVD at the time),³ and they offered a diagnosis of “tobacco-induced angina.” Despite the cautious language, the overall clinical picture strongly supported post-infarction angina with underlying myocardial damage. Treatment remained empirical: bed rest,



Figure 1. Chronological photographs of Mustafa Kemal Atatürk from 1923 to 1930, reflecting changes in his physical appearance following myocardial infarctions. (A) Photo taken prior to his first cardiac event (24.04.1923). (B, C, D, E) Images captured after his first attack in November 1923, spanning 1924–1926. (F) Photograph taken shortly after his third and most severe cardiac event in May 1927. (G, H, I) Images from 1928–1930, reflecting the progressive physical decline. As can be seen, Atatürk’s abdominal obesity gradually increases, and his overall healthy appearance visibly deteriorates following the third myocardial infarction.

morphine, and a milk–vege diet.³⁻⁵ Tobacco was initially prohibited, but he was later permitted to resume smoking in reduced amounts—reflecting both medical uncertainty and Atatürk's strong dependency.

Atatürk's cardiovascular history probably exemplifies the multifactorial nature of premature CVD, shaped by a combination of genetic, infectious, and lifestyle factors. His father⁷ died young at age 46-47, and his mother reportedly suffered from high BP and died of heart failure⁸—probably suggesting familial predisposition (Table 1). He also had severe chronic infections, including malaria and pyelitis,⁹ both of which are associated with systemic inflammation and endothelial damage. His personal habits further increased the risk: an unbalanced diet, consumption of 10-15 cups of coffee and 30-40 (50) cigarettes per day, minimal and irregular sleep, and extreme work hours, sometimes exceeding 30 hours without rest.²⁻⁶

Without access to aspirin, anticoagulants, β -blockers, statins, cholesterol testing, or revascularization therapies, his treatment was restricted to bed rest and symptomatic relief. Unfortunately, prolonged immobilization carried its own dangers—particularly deep vein thrombosis and pulmonary embolism—which may have contributed to his coronary episodes and also progression of a probable myocardial dysfunction after 1927. Eyewitnesses and photographs from this time suggest worsening dyspnea, edema, abdominal obesity (distension), and declining functional capacity (Figure 1).^{3,10}

Chronic passive hepatic congestion from right-sided heart failure may have contributed to the cirrhosis recognized in 1938, which was diagnosed quite late, despite long-standing symptoms such as severe pruritus and recurrent nasal bleeding. Although often attributed solely to alcohol,¹¹ the etiology of his cirrhosis may have been multifactorial, including heart failure and malaria and/or anti-malaria drugs. Regardless, his clinical course underscores both the therapeutic limitations of early 20th-century cardiology and the complex

politics surrounding the care and disclosure of health conditions in high-profile leaders.

Atatürk's ability to lead through and beyond multiple myocardial infarctions is a remarkable testament to his resilience. That he remained so successful despite life-threatening illness and progressive decline invites further exploration—possibly rooted in traits such as intense drive, hyperfocus, hyperactivity, and an unshakable sense of duty.

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