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Almost Gone in 70 Seconds

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ABSTRACT: We will present the case of a 63-year-old male patient who underwent extensive neurological and cardiological treatment over a period of several years due to a history of unresolved syncopes. It was only after the installation of the loop recorder that the cardiologic etiology of the syncope was established in the form of a recorded pause of 70 seconds with rhythm disturbances in the form of non-sustained ventricular tachycardia. After an ICD device (implantable cardioverter defibrillator) was installed, the patient's symptoms completely resolved.

KEYWORDS: asystole, ICD, syncope

INTRODUCTION

Syncope is a sudden and temporary loss of consciousness caused by reduced blood flow to the brain. This condition is characterized by rapid, short-term loss of consciousness and postural collapse, usually lasting a few seconds to minutes. Syncope can result from a variety of causes, including cardiovascular, neurological, metabolic, and psychogenic factors. A sudden cessation of cerebral blood flow for as short as 6–8 s can cause symptoms of syncope (1). A systolic BP of 50–60 mmHg at heart level, i.e., 30–45 mmHg at brain level in the upright position, will also cause a syncope (2,3). Physicians can distinguish syncope from other types of total loss of consciousness in about 60% of cases by using a thorough clinical history (4). Depending on the patient's age and the clinical environment, syncope prevalence varies. Syncope is thought to occur in 3% of men and 3.5% of women during the course of their lifetimes (5). In individuals aged 35 to 44, the prevalence of syncope is estimated to be 0.7%, whereas in patients aged 75 and beyond, it ranges from 4% to 6%. In the Framingham research, males aged 70 to 79 had a syncope incidence of 11.1 per 1,000 events person-years, up from 5.7 per 1,000 events person-years in men aged 60 to 69. The underlying etiology of syncope affects the prognosis overall. There is a substantial risk of overall mortality and sudden cardiac death in patients with syncope who also have structural heart disease and primary electrical disease. On the other hand, reflex syncope in young patients has a very good prognosis. Recurrence of episodes and physical harm are linked to morbidity in syncope patients. In population studies, one-third of patients experience a recurrence of syncope within three years. The rate of recurrence appears to be influenced by the number of prior episodes, but not by gender, the findings of a tilt test, severity, or the presence of heart disease. In comparison to individuals with 3 syncope episodes, the estimated recurrence in 1 to 2 years for patients with 1 or 2 syncope episodes is 36% to 45%. Minor trauma accounts for 29.1% of syncope patients who report to the emergency room, major trauma for 4.7%, and major trauma for 43% of older patients with carotid disease. Morbidity is particularly prevalent in the elderly and is linked to institutionalization, depression, fractures, and feelings of inadequacy (6). Prospective investigations have revealed that the majority of syncopal occurrences (between 38% and 56%) are caused by neurally driven factors. Smaller percentages of cases are attributable to cardiovascular reasons, which are further divided into syncope owing to orthostasis (2% to 24%) and structural heart disease (11% to 23%). In 14% to 18% of cases, the cause of the syncope is unknown. Compared to the previous ones, 2018. ESC (European society of cardiology) guidelines emphasize the need for prolonged monitoring of patients with the installation of a loop recorder in selected cases and prolonged monitoring via Holter ECG telemetry to diagnose the cardiac cause of syncope.

CASE REPORT

A 63-year-old man presents himself for a cardiological examination because he has had a history of syncope for the past 2 years, which has occurred on three occasions. According to the wife, the syncope lasted on average between 5 and 10 seconds with prodromal symptoms such as buzzing in the head, sweating, and temporal headache. Disturbances were related to mood swings,

during the 22-hour bus ride and after getting up from the chair in the office. In family there was no history of sudden cardiac death in a relative. As a child, he had febrile convulsions and inflammation of the parotid glands. There is a history of prior stomach ulcer, which was successfully repaired with a stable blood count and chronic gastritis. No history of prior diabetes or known hypertension. Anamnesis is negative for alcohol intake, smoking and consumption of psychoactive substances and drugs. There is a normal neurological and cardiological status without vascular murmurs on the precordium and on the carotid arteries.

METHODS

Transcranial doppler (TCD) indicated a reduced flow rate in the posterior cerebral artery with signs of increased circulatory resistance and regular flow in the other arteries, while TCD of the vertebral arteries indicated slowed flow and increased circulatory resistance. The EEG (electroencephalogram) was slightly dysrhythmically altered without focal changes. Brain magnetic resonance (MR) showed several non-specific T2-type hypodensities without other pathology. The tilt table test indicated slight adrenergic and cardiovagal damage, while it was negative in the provocation with a slight drop in pressure on the left carotid sinus, so the possibility of hypersensitivity of the carotid sinus was not excluded. Color doppler of the carotid arteries indicated arterial tree with minimal, stable peripheral ACI plaques. 24-hour Holter pressure: average pressure 142/75 mmHg, daily average systole values 143 mmHg (96-210), diastole 79 mmHg (48-115), night average 140 mmHg systole (127-155) with 68 in diastole (61-75) and dipping status 13% with introduction of ramipril 1.25 mg in therapy. Isolated laboratory findings: hemoglobin 140, leukocytes 4.8, platelets 181, D dimers 255 ug/L, cholesterol 5.55 mmol/l, LDL 3.71 mmol/l, triglycerides 1.9 mmol/l, creatinine 70 umol/l, potassium 3.6 mmol/l, sodium 134 mmol/l, TSH 1,065 mIU/L. Polysomnography: nocturnal epilepsy was ruled out and sleep apnea was verified on 6 occasions. Psychological testing indicated above-average intellectual abilities without certain indicators of organic deficits in cognitive functions, as well as significant psychopathological deviations, except for latent anxiety as part of the experience of current disturbances. Cardiac echo indicated a regular left ventricular ejection fraction with concentric hypertrophy of the walls with competent valves and initial diastolic dysfunction(I/IV) and present dyskinesia of the interventricular septum consistent with left bundle branch block. Coronary angiography showed non-obstructive calcifications of the left main trunk of left coronary artery and LAD without significant stenosis. EKG showed normal sinus rhythm with first degree AV block and left bundle branch block without significant pauses (Picture 1). 24 hour and 48-hour Holter EKG without significant pauses with a total of 111,726 QRS complexes with basic sinus rhythm with permanent left bundle branch block. Maximum heart rate 130/min, minimum 49/min with an average of 80/min. There were no pauses longer than 2 seconds with one VES (ventricular extrasystole) and 3 individual SVES (supraventricular extrasystole). No atrial fibrillation or malignant heart rhythm disorders were recorded. Examination by an otolaryngologist indicated a reduction of hearing on both sides at 2000 Hz. The Dix-Hallpike test was negative, and the two-hot link caloric test indicated uniform excitability of both labyrinths. Ocular vestibular-evoked myogenic potential (oVEMP) was normal as well as cervical vestibular evoked myogenic potential (cVEMP) test. Video head impulse test showed slight deviation of 3% to the right.). Loop recorder (REVEAL LINQ LNQ11) is on device reading 13.06. 2019 indicated a pause from 30.5. 2019. of 70 seconds type of total AV block and sinus arrest (Picture 2,3,4) with 5 episodes of ventricular tachycardia.

DISCUSSION

The path to elucidating the cause of syncope is often difficult and not always successful, so we decided to present the case of our patient, who for a period of almost four years had occasional non-specific syncope without a clear diagnosis. Disturbing loss of consciousness was the cause of anxiety and fear of sudden death, which was verified during a psychological examination. Most frequent causes of cardiac syncope are arrhythmias. They reduce cardiac output, which results in hemodynamic dysfunction (7). Among these are bradyarrhythmias, tachyarrhythmias, arrhythmias brought on by drugs and abnormal electrolyte levels. Syncope can be caused by both severe tachycardia (from either a supraventricular or ventricular origin) and considerable bradycardia (from either a supraventricular or ventricular origin). The initial cardiological tests indicated permanent left bundle branch block and AV block of the 1st degree with hypertrophy of the walls of the left ventricle, which indicated a structurally altered heart, so further extensive cardiological treatment was indicated. Because of the risk factor age, male gender, newly discovered arterial hypertension, dyslipidemia and an unclear history of syncope, coronary angiography was performed on two occasions, which indicated non-obstructive coronary disease. No significant pauses or malignant rhythm disturbances were recorded on the Holter ECG in 24 and 48-hour period, while the ultrasound of the heart indicated a preserved ejection fraction of the left ventricles without valvular stenosis, so the initial tests indicated a non-cardiogenic mechanism of syncope. 24-hour Holter pressure indicated initial hypertension without a significant drop in arterial pressure that would explain the syncope. Typically, syncope in intrinsic sick sinus syndrome develops as a result of prolonged pauses brought on by sinus arrest or sinoatrial block which will only be clarified later in our case. Basic management of syncope includes a neurological examination due to the large number of neurally mediated syncope. An extensive neurological work-up was performed, which indicated well-patented carotid arteries without significant stenoses. TCD indicated a reduced flow rate in the posterior cerebral artery with signs of increased circulatory resistance and regular flow in the other arteries, while TCD of the vertebral arteries indicated slowed flow and increased circulatory resistance which is a non-specific finding and cannot explain the present syncope. Polysomnography and EEG ruled out epilepsy as the cause of syncope, while sleep

apnea was verified, which can cause the development of arterial hypertension due to increased sympathetic stimulation. Tilt table test indicated slight adrenergic and cardiovagal damage, while it was negative in the provocation with a slight drop in pressure on the left carotid sinus, so the possibility of hypersensitivity of the carotid sinus was not excluded and it was misleading. Color doppler of the carotid arteries showed minimal stable peripheral ACI plaques, which did not support the cause of syncope, but only represents a risk factor for cardiovascular diseases. The otolaryngologist's treatment ruled out inner ear disease as the cause of the disturbances, and hypoacusis was established, which is in accordance with the patient's age. Laboratory findings ruled out anemia and metabolic causes such as uremia as the cause of loss of consciousness, with regular findings of D dimer, which nullify the possibility of repeated pulmonary emboli as the cause of syncope. After the patient's insistence on further cardiac treatment due to the continuation and intensification of syncope, we decided to install a loop recorder. Loop recorder (REVEAL LINQ LNQ11) is on device reading 13.06. 2019 indicated a pause from 30.5. 2019. of 70 seconds type of total AV block and sinus arrest (picture attached) with 5 episodes of ventricular tachycardia. ICD device with pacing capability was installed, which solved the patient's problems and since then there has been no syncope. Picture 3 clearly shows the moment when the patient activates the loop recorder with a blow during a long pause and during presyncope, as advised during device installation. At further controls, the patient became dependent on electrostimulation with the planned replacement of the pacemaker generator due to permanent ventricular stimulation. No complex ventricular rhythm disorders were detected in subsequent follow-ups, so the cause of ventricular tachycardia was asystole.

CONCLUSIONS

We can conclude that a thorough and persistent approach is needed in resolving syncope in elderly and middle-aged patients due to possible structural heart disease. Our treatment coincides with the recent ESC guidelines of prolonged monitoring of patients with unresolved syncope. In this way, it would be possible to reduce the possibility of developing sudden cardiac death in a part of the patients, which in the case of our patient would be inevitable.

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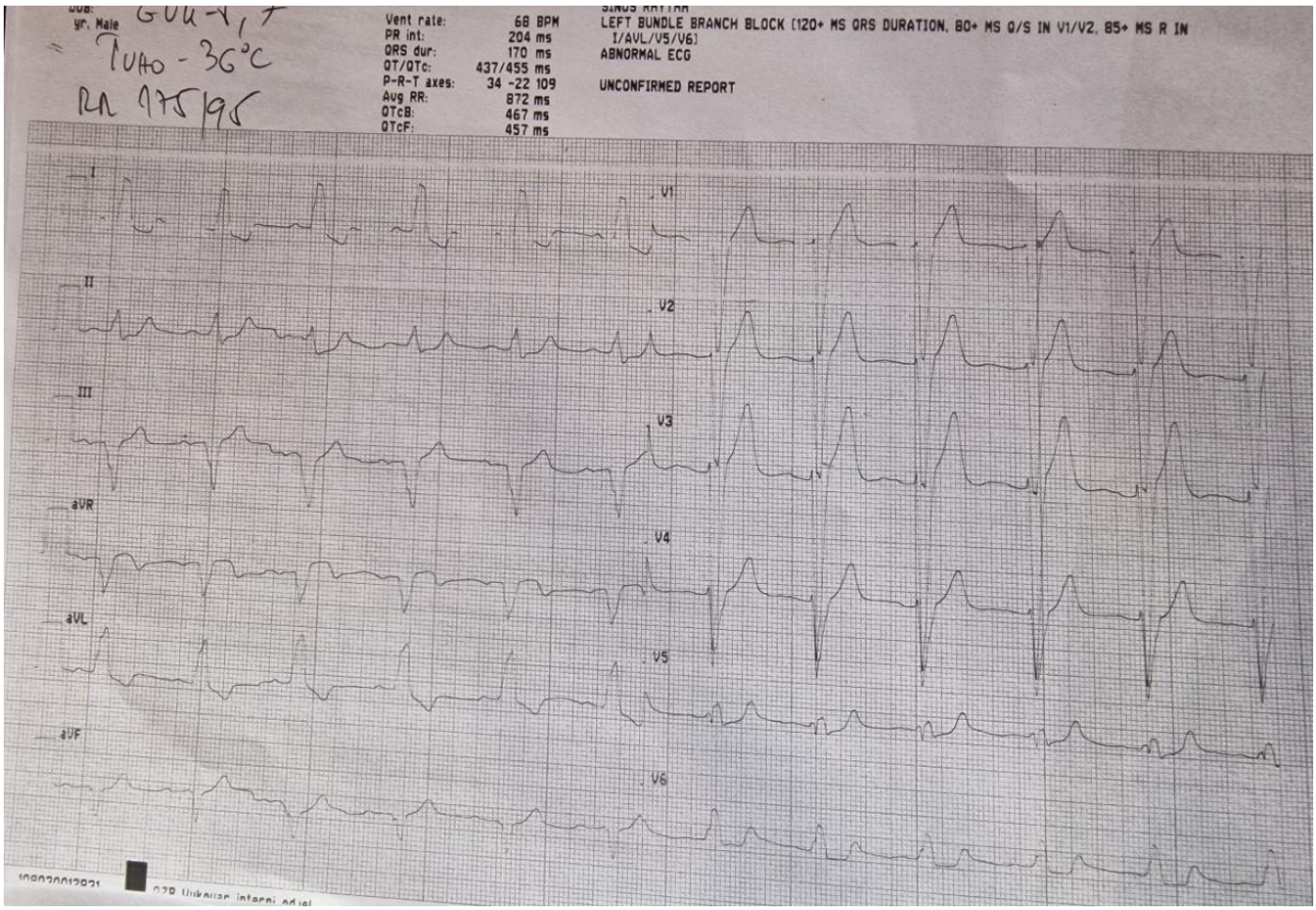

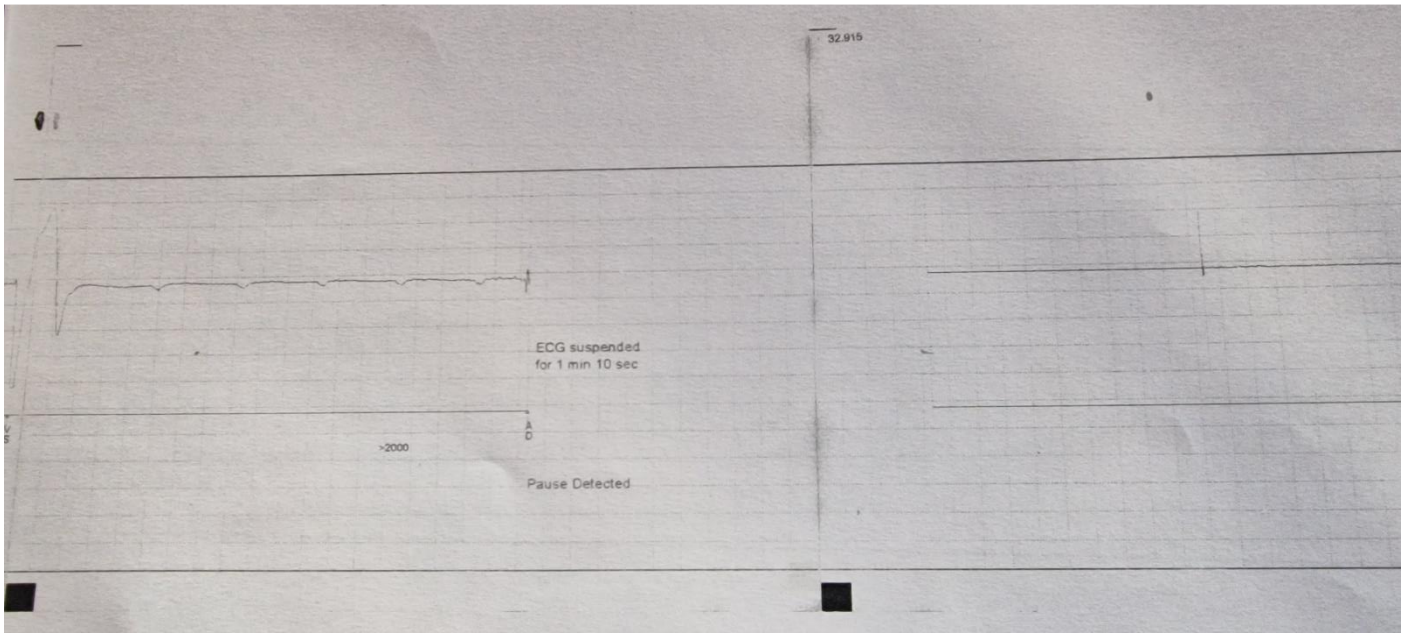

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