Ege Journal of Medicine / *Ege Tıp Dergisi* 2014;53(1):45-48

Adams-Stokes attacks: Imitator of epilepsy

Adams-Stokes atakları: Epilepsi taklitçisi

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Summary

Stokes-Adams Syndrome is commonly seen in elderly patients and does not cause any symptoms in most cases. However, it may have convulsions that mimic epileptic seizures during periods of cerebral ischemia. These symptoms are often intermittent, changeable and unpredictable. The most common cause of this syndrome is cardiac arrhythmias. Despite numerous investigations for this syndrome, most diagnoses are made by 12-lead electrocardiography. Cardiac pacemaker implantation is the most powerful therapy.

A patient who had stroke history, was admitted to the emergency department with repetitive convulsions that mimic epileptic seizures. Also these convulsions included loss of consciousness and tonic-clonic jerks. The convulsions were associated with cardiac arrhythmia and increased with antiepileptic therapy. The aim of this paper is to review the Stokes-Adams attack and its similarity to epileptic seizure with a case report.

Key Words: Stokes-Adams syndrome, epileptic seizures, sick sinus syndrome, convulsive syncopes.

Özet

Stokes-Adams sendromu sıklıkla yaşlı hastalarda görülür ve çoğu olguda herhangi bir semptoma neden olmaz. Bununla birlikte, Stokes-Adams sendromunda serebral iskemi periyodları sırasında hastalarda epileptik nöbetleri taklit eden konvülsiyonlar da görülebilir. Çoğu zaman bu semptomlar ani, değişken ve beklenmedik bir şekilde ortaya çıkar. Bu sendromun en sık nedeni kardiak aritmilerdir. Yapılan çoğu tetkike rağmen tanı 12 kanallı elektrokardiyografi ile konur. Kardiak pacemaker implantasyonu ise en etkili tedavi seçeneğidir. Daha öncesinde inme öyküsü olan hasta epileptik nöbetleri taklit eden tekrarlayan konvülsiyonlar ile acil servise başvurdu. Hastanın tonik-klonik kasılmalarına bilinç kaybı eşlik etmekteydi. Sonrasında kardiak ritm bozukluğundan kaynaklandığı anlaşılan nöbetler anti epileptik tedavi ile artış göstermişti. Bu makalede olgu sunumu eşliğinde Stokes-Adams ataklarının epileptik nöbetlere olan benzerliği tartışıldı.

Anahtar Sözcükler: *Stokes-Adams sendromu, epileptik nöbet, hasta sinüs sendromu, konvülsif senkop.*

Introduction

Stokes-Adams syndrome (SAS) is defined as an abrupt, transient loss of consciousness due to a sudden decrease in cardiac output that is caused by a sudden change in the heart rate or rhythm. This definition does not include vasovagal syncope and epilepsy, nevertheless during this episode some patients may have seizures caused by cerebral ischemia.

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Received: 10.12.2012 Accepted: 13.06.2013

These partial or complete heart blocks are usually presented as asymptomatic and some of these arrhythmias can produce syncopal episodes which are also called “arrhythmia induced syncope” (1). SAS was first observed by Adams in 1827 and Stokes in 1854. Huchard gave it the name of these two clinicians in 1893 (2).

Sick sinus syndrome (SSS) is characterized by various arrhythmias such as sinus bradycardia related to the sinus node degeneration, sinus arrest or sinoatrial block, regular or irregular atrial tachycardia, tachycardia-bradycardia attacks or slow ventricle responding atrial fibrillation (3,4). Frequently, most sick sinus syndromes are idiopathic and the cause can be multifactorial. Degenerative fibrosis of nodal tissue is the most

common cause of intrinsic changes in the sinoatrial node that leads to SSS. The choice of treatment for symptomatic bradyarrhythmias in patients with SSS is the placement of a pacemaker. In addition, artificial pacemakers are well tolerated in elderly patients (5).

The patient with stroke history was admitted to the emergency department with repetitive convulsions that mimic epileptic seizures. Also these convulsions had included loss of consciousness, tonic-clonic jerks and increased with antiepileptic treatment that was caused by cardiac rhythm disturbance. The aim of this paper is to review the Stokes-Adams attack and its similarity to epileptic seizure, with a case report.

Case Report

An 80-year old patient admitted to the emergency service with complaints of sudden loss of consciousness once or twice a week, with her eyes turning upward and contractions of arms and legs continuing for one-two minutes. Her complaints had repeated 7-8 times within the last two days. She had no urea-stool incontinence but she had a past medical history of an ischemic stroke 5 years previous. In her neurological examination, she had 4/5 level muscle strength on the left arm and leg. She had sequelae of left central facial palsy and positive Babinski sign on the left side. Other examination findings were normal. Her brain tomography showed an encephalomalacia in the right parietal lobe of the brain. She had a newly diagnosed of atrial fibrillation on Electrocardiography (ECG) screening, Laboratory tests including hemogram, electrolytes (Na, K, Ca, Mg), and thyroid hormones were also normal.

The patient was followed up with cardiac monitoring in the intensive care unit in order to investigate the cause of a sudden loss of consciousness, seizure and also the possible cardiac arrhythmias. Phenytoin 3x100 mg per a day was given to the patient because of the encephalomalacia which could be a reason for the epileptic seizures. Suddenly, she developed bradycardia-tachycardia attacks, loss of consciousness with convulsions and also sinus bradycardia, sinus pauses on her ECG, after phenytoin was given (Figure-1). All of these findings showed that the seizures of the patient had been a result of cardiac arrhythmia.

In this case, arrhythmia which was triggered by phenytoin was the highlighter for the diagnosis. Although the medication with phenytoin and other anti-arrhythmic drugs were stopped, bradycardia tachycardia attacks, sudden loss of consciousness and seizures had continued repeatedly. After the permanent pace maker implantation, the temporary loss of consciousness attacks and seizures ceased (Figure-2).

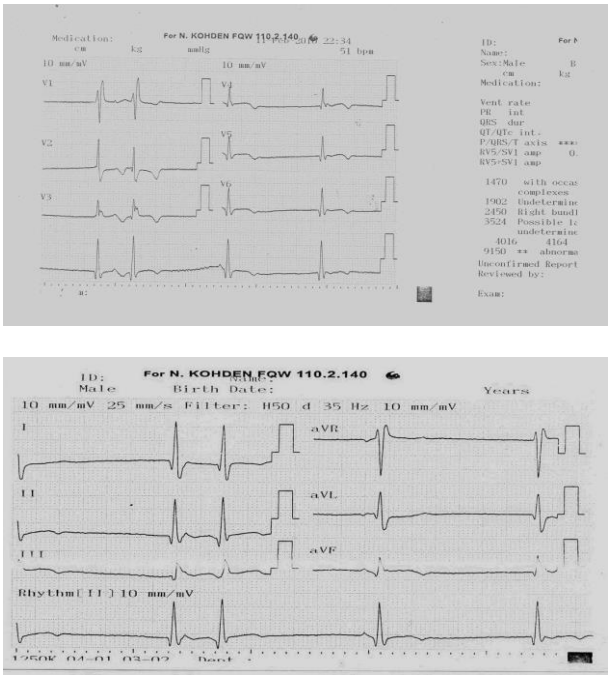

Figure-1. Examples of episodes of AV block monitored by electrocardiogram. Before pacemaker implantation.

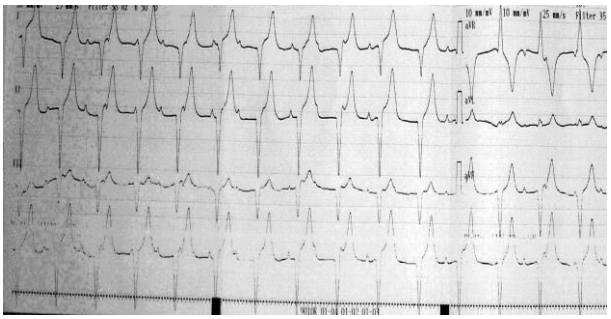

Figure-2. The ECG of after pacemaker implantation.

Discussion

SSS was first described in 1968 and it has some various manifestations of sinoatrial rhythm disorders which are serious, persistent, otherwise inexplicable sinus bradycardias, and paroxysmal or chronic atrial fibrillation due to sinus arrest (6). The syndrome generally occurs in the 6^{th}-7^{th} decade of life and it has 0.3% prevalence in society (3,4). Of those, 20% to 30% SSS patients have had symptoms of paroxysmal or intermittent atrial fibrillation as part of the sinus node disorder. This entity is also called bradycardia-tachycardia syndrome. An irregular and powerless atrial movement, such as atrial fibrillation, leads to blood stasis within the atria and their appendages, forming the basis for thromboembolism (6). Patients with sick sinus syndrome who have bradycardia-tachycardia syndrome or chronic atrial fibrillation are at risk for embolic stroke (5). In this case, the possibility of the thromboembolism may be a reason for stroke history.

The classical description of a Stokes Adams attack is of collapse without warning, associated with loss of consciousness lasting a few seconds. The affected individual is pale at first, but can become flushed on recovery from the episode. However, this flushing episode does not always occur and the seizure-like activity can occur if the attack is prolonged. The disorder is typically associated with complete heart block, but has also been described in other diseases such as tachy-brady syndrome (7). Sinoatrial rhythm disorders, such as SSS, and their cardiac rhythm disturbance are causes of SAS. A literature review showed that, the original Stokes-Adams concept of cardiac arrhythmias resulting in syncope was described in 1829 and Levine reported sinoatrial block and paroxsymal atrial fibrillation in 1916, then Short described symptomatic brady-tachycardia in 1954 (8,9).

The symptoms of epileptic seizures are varied and there are many imitators, ranging from convulsive syncope through to psychogenic events. The phenomenon most commonly mistaken for a convulsive seizure is syncope (10). Symptoms of arrhythmia induced syncope can vary from slight faintness to complete loss of consciousness, with or without convulsions (1). Generally, in clinical practice, the differential diagnosis between epilepsy and syncope is not easy. Although the aetiologies of the two entities are completely different, their clinical presentation may be quite similar and indistinctive when based on clinical history taking. For that reason, the typical clinical presentation can be confusingly similar. Ozkara et al. present a patient with reflex syncopal episodes that mimic seizures by using video-EEG recordings. When the patient presented, she lost consciousness and her heart beating stopped and diffuse generalized slow delta waves with high amplitude appeared on the EEG, followed by a complete suppression of cerebral activity (11). The clinical appearance is similar but also etiology entirely different, that caused by cardiac arrhythmia.

Occasionally, patients who have syncopes can be treated with antiepileptic drugs. Gelisse et al. (12) reported on a patient whose eye compression produced a typical vasovagal syncope, which had been treated for five years with sodium valproate because of a misdiagnosis of epilepsy. We can see the most common convulsive movements in these cardiac fluctuations as; myoclonus, multifocal myoclonic jerks, head turns, oral automatisms and righting movements and tonic extensor spasms (11). Our patient presented with repetitive convulsions, loss of consciousness, generalized tonic and clonic spasms that mimic epileptic seizure.

However, if we begin treating patients who have cardiac syncope with a misdiagnosis of epilepsy, diphenylhydantoin can be a bad choice since diphenylhydantoin or lidocaine may also depress the ectopic pacemaker of the heart (1). When we used diphenylhydantoin in our case, it increased all symptoms and we observed a sinusoidal pause, bradycardia and tachycardia attacks in her ECG.

It is an unfortunate mistake that Stokes-Adams syndrome can be evaluated as a generalized epileptic seizure. If a convulsive seizure of only seconds duration with an abrupt return of consciousness implies syncope not epilepsy. Repeated convulsive syncopes, without provocation, suggest cardiac syncope (13). Furthermore, a 12-lead ECG obtained in a patient with recurrent seizure-like episodes is necessary and important. In some of the patients, an interictal ECG can be normal and inclusion of ECG and EEG analysis can be helpful in this situation (14). The interpretation of seizure semiology in such paroxysmal events may also be misleading. For that reason, other parameters, such as ECG, must be added to investigation of seizure semiology. When there is doubt, the neurologist should not hesitate to call for further cardiological investigation (11). Syncope is much more common than epilepsy and may present with symptoms akin to the latter. This fact is not well appreciated and often leads to misdiagnosis (15).

In conclusion, cardiac arrhythmia can be a reason for the patients who have stroke history and some limitations in central nervous system perfusion, thus temporary loss of consciousness with accompanying convulsions can occur. It can be distinguished by using long-time cardiac rhythm monitoring whether or not these convulsions were caused by epileptic seizure. As a result, if the convulsions increase with exclusively antiepileptic medication in elderly patients, cardiac arrhythmia and Stokes-Adam Syndrome should be considered to avoid the misdiagnosis of epilepsy.

References

1. O'Rourke RA. Clinical cardiology: The Stokes-Adams syndrome-definition and etiology; mechanisms and treatment. Calif Med 1972;117(1):96-9.
2. Gordon HS. Adams-Stokes syndrome, with report of two cases. Cal State J Med 1905;3(7):210.
3. Brignole M. Sick sinus syndrome. Clin Geriatr Med 2002;18(2):211-27.
4. Lilly LS (ed). Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 5th ed. Philadelphia; Elsevier: 1997.
5. Adan V, Crown LA. Diagnosis and treatment of sick sinus syndrome. Am Fam Physician 2003;67(8):1725-32.

6. Alt E, Lehmann G. Stroke and atrial fibrillation in sick sinus syndrome. Heart 1997;77(6):495-7.
7. Harbison J, Newton JL, Seifer C, Kenny RA. Stokes-Adams attacks and cardiovascular syncope. Lancet 2002;359(9301):158-60.
8. Hart WJ. The neurological presentation of sick sinus syndrome. Can Fam Physician 1979;25:1233-41.
9. Short DS. The syndrome of alternating bradycardia and tachycardia. Br Heart J 1954;16(2):208-14.
10. Chadwick D, Smith D. The misdiagnosis of epilepsy. BMJ 2002;324(7336):495-6.
11. Ozkara C, Metin B, Kucukoglu S. Convulsive syncope: A condition to be differentiated from epilepsy. Epileptic Disord 2009;11(4):315-9.
12. Gelisse P, Serafini A, Davy JM, Balbe M, Coubes P, Crespel A. Video-EEG in syncopal attack due to ocular compression in an adolescent mistreated for epilepsy. Epileptic Disord 2007;9(2):174-8.
13. You CF, Chong CF, Wang TL, Hung TY, Chen CC. Unrecognized paroxysmal ventricular standstill masquerading as epilepsy: A Stokes-Adams attack. Epileptic Disord 2007;9(2):179-81.
14. Diaz-Castro O, Orizaola P, Vazquez S, Gonzalez-Rios C, Pardo M, Fernandez-Lopez JA, et al. Images in cardiovascular medicine. "Stokes-Adams epilepsy": sometimes we need the electroencephalogram. Circulation 2005;112(8):101-2.
15. Petkar S, Cooper P, Fitzpatrick AP. How to avoid a misdiagnosis in patients presenting with transient loss of consciousness. Postgrad Med J 2006; 82(972): 630-41.