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Symptomatic Bradycardia in a Patient with Meningoencephalitis

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Abstract: ***The National Institutes of Health defines bradycardia as a heart rate <60 bpm in adults other than well trained athletes. However, population studies frequently use a lower cutoff of 50 bpm. In physiological cases, bradycardia is asymptomatic. When bradycardia becomes severe or is associated with other pathologies, it may manifest as lightheadedness, presyncope, and syncope, chest pain on exertion, symptoms of heart failure, cognitive slowing, and exercise intolerance. Sinus bradycardia is caused by intrinsic cardiac disorders like sick sinus syndrome or inferiormyocardial infarction, metabolic and environmental causes (such as hypothyroidism and electrolyte disorders), medications (such as beta-blockers and amiodarone), infection (such as myocarditis), increased intracranial pressure, and toxic exposure, while it can sometimes be a normal phenomenon, especially during sleep, in athletes, and during pregnancy. Symptomatic sinus bradycardia should warrant a thorough work-up in order to identify any reversible causes; otherwise, placement of a permanent pacemaker could be needed. We reported a patient where the patient complained of frequent fainting with a pulse rate of 50 beats per minute, there were also complaints of pain in the back of the head, fever accompanied by decreased consciousness and had seizures. On physical examination, there was positive neck stiffness and electrocardiographic examination with the impression of sinus bradycardia. So that the patient was diagnosed with meningoencephalitis with symptomatic bradycardia, atropine sulfate was chosen as therapy for the management of bradycardia in these patients.***

Keywords: Bradycardia, Meningoencephalitis

1. Introduction

Sinus bradycardia is defined as bradycardia with a normal sinus rhythm on an electrocardiogram (ECG). This means that the sinus node is firing impulses at a slower rate than normal. The normal firing rate for the sinus node is around 60 to 100 beats per minute. In an analysis of 4 population studies from the Netherlands, in adults from 20 to 90 years of age, the lowest second percentile for heart rate ranged from 40 to 55 bpm depending on sex and age. Sinus bradycardia can be a normal finding in healthy individuals, as it is responsible for the physiological slowing of heartbeats during sleep. However, it can also occur as a pathological response to other conditions. Common pathological causes of sinus bradycardia include myocardial infarction, obstructive sleep apnea, medications beta-blockers, and infections.1, 2

Meningitis is one of the most common central nervous system infection. In some cases, the inflammatory process occurs in both brain parenchyma and meninges, known as meningoencephalitis. The incidence of meningitis has decreased since the widespread use of vaccinations, however, the mortality remains high. A study in the United States reported that mortality due to meningitis in 2006-2007 was 14.3%, not significantly differ with mortality rate from 1998 to 1999 which was 15.7%. Previous study conducted at Dr. Sardjito General Hospital, Yogyakarta reported that more than a half of meningoencephalitis patients in the neurological wards died.3

Development of symptomatic sinus bradycardia should warrant treatment in order to reduce symptoms as well as a thorough work-up in order to identify any reversible causes of bradycardia and prevent the unnecessary placement of a permanent pacemaker that could be otherwise needed in the case of an intractable and persistent sinus bradycardia.4

We present the case of a patient hospitalized with symptomatic bradycardia due to meningoenchepalitisas an underlying disease.

2. Cases

Patient Mr. WPD, a 38-year-old male, from the Balinese tribe came to the ER on December 26, 2021 with complaints of frequent sensations such as fainting since 5 days before entering the hospital with a duration of between 4-6 seconds. This complaint usually arises when the condition of fatigue and begins with a feeling of heaviness in the back of the head then the vision becomes dark. When unconscious, the patient can still hear the voices of the people around him. The patient is well conscious after fainting without any weakness or paralysis of the limbs. The patient also complained of fever (with measurements of 37.5-37.7oC), fever was felt since 6 days before admission to the hospital, felt intermittent accompanied by nausea and vomiting. Complaints of chest pain, shortness of breath and palpitations were denied. Complaints of hair loss, skin rash and joint pain were also denied.

On the second day of treatment, the patient complained of a constant and stabbing headache. The patient was given painkillers and the patient's complaints improved, but the complaints kept coming back. On the third day of treatment, due to persistent headaches, high fever (with a measurement of 38.8 oC) and the patient's family said that the patient was

often sleepy and difficult to wake up, the patient was consulted to the neurology department with decreased consciousness and cephalgia. At night during the third day of treatment, the patient's family reported that the patient had seizures, seizures throughout the body with a duration of ± 2 minutes.

The patient had sinusitis and had surgery for 1 year ago. The patient had never had these symptoms before. Past medical history such as hypertension, diabetes, kidney disease, congenital heart disease, history of pulmonary TB and epilepsy was denied.

On physical examination, the patient's consciousness decreased, GCS E3V5M4 with blood pressure 100/60 mmHg, pulse rate 50 times per minute, respiratory rate 20 times per minute, temperature 37.8oC and oxygen saturation 97% room air. General status, on head and neck examination found resistance when bending the neck (positive neck stiffness), positive pupillary reflex isocor with pupil size 3mm, no enlarged lymphnodes, JVP ± 2 cmH2O. Examination of the thorax and abdomen was found to be normal. On examination of the extremities there was no lateralization.

Complete blood count, white blood cells 5.6 x103/µL, hemoglobin 16.1 g/dL and platelets 279 x103/µL, hematocrit 45.4%, absolute neutrophils 4.3 x103/µL, absolute lymphocytes 0.9 x103/ L. Blood chemistry Urea N 49 mg/dL, blood creatinine 0.96 mg/dL, ALT 63 U/L, AST 25 U/L, sodium 132.8 mmol/L and potassium 4.96 mmol/L, TSH 0.11 IU/Ml, FT4 16.20 pmol/L with normal effect. HIV testing was found to be non-reactive. Electrocardiographic examination with sinus rhythm, frequency 48 beats per minute, normal axis, P wave 0.04 seconds, PR interval 0.16 seconds, QRS complex 0.08 seconds, ST-T waves normal, with ECG impression of sinus bradycardia. The chest X-ray results were within normal limits.

The patient was diagnosed with Meningoencephalitis et causa bacterial dd virus with symptomatic bradycardia. While being treated, the patient was given infusion of 0.9% NaCl 20 tpm, Ceftriaxon 2x2 g (iv), omeprazole 2x40 mg (iv), ondansetron 3x4 mg (iv), dexamethasone 3x10 mg (iv) with tappering off after 3 days, paracetamol 3x1 g (iv), mecobalamin 2x500 mg (iv), Sulfas Atropine 1 mg (iv) three times every 5 minutes, phenytoin 2x100 mg (po), clobazam 1x10 mg (po).

3. Discussion

The National Institutes of Health defines bradycardia as a heart rate <60 bpm in adults other than well trained athletes. However, population studies frequently use a lower cutoff of 50 bpm. In physiological cases, bradycardia is asymptomatic. When bradycardia becomes severe or is associated with other pathologies, it may manifest as lightheadedness, presyncope, and syncope, chest pain on exertion, symptoms of heart failure, cognitive slowing, and exercise intolerance. Symptoms usually appear or are increased in a situation where an increase in cardiac output is required. For example, during exercise, stress, or during an active infection. It is important to know that, in many cases, patients do not usually present with the complaint of bradycardia rather the underlying condition itself, for instance, myocardial infarction. However, in some cases, patients may develop symptomatic bradycardia that mandates proper and rapid management. Symptomatic bradycardia is defined as “documented bradyarrhythmia that is directly responsible for the development of the clinical manifestations of syncope or presyncope, transient dizziness or lightheadedness, heart failure symptoms, or confusion state, resulting from cerebral hypoperfusion attributable to slow heart rate.[1, 2]

A thorough history and physical examination are required not only to diagnose bradyarrhythmia but also to discover the underlying etiology and comorbid condition of the patient. It is essential to ask about the details of the episode of bradycardia, the frequency, timing, duration, triggers, and alleviating factors. Since medications are a common cause of bradycardia, one must always ask about both prescription and over-the-counter medication usage. Asking about previous episodes and how closely matched those were to the recent episode is also important as it may indicate different pathologies or similar ones. In the end, physicians should end their history with a comprehensive review of systems, family history of comorbid conditions, and cardiovascular risk assessment. During the physical exam, physicians must focus on the examination of the cardiovascular system. However, other systems must also be examined to determine whether a mixed pathology is present or not. After the history and physical examination, the most essential tool for evaluation is a 12-lead ECG. It can detect the abnormality whether it is a conductive or generative error. However, for it to be effective, it must be done during the attack. If the symptoms are resolved, the ECG will not be of value in the diagnosis of bradyarrhythmia as it will show normal heart rhythm in most cases, unless it is due to an AV conduction defect. However, it remains essential to rule out other pathologies that may be found. For example, myocardial infarction and cardiomyopathies. If the ECG reading is normal and the patient reports that symptoms appear or increase in severity with exercise, exercise ECG is indicated to determine the abnormality. However, in most cases, it can be difficult to obtain useful data from exercise ECG because of the comorbid condition of most patients. If suspicion remains high for bradycardia, an ambulatory monitor, such as holter, can be used to diagnose bradyarrhythmia. Other tests that can be done are laboratory testing and invasive testing. These tests should never be done routinely and only when high suspicion of an underlying pathology be present. Laboratory testing may be considered for patients with bradycardia or conduction disorder based on the initial history and physical and should be targeted towards narrowing a specific differential diagnosis. Testing may include thyroid function testing, Lyme titers, electrolytes, and other selected tests as indicated. An example of

invasive testing is the implantable cardiac monitor.2, 5

Bradycardia management depends on multiple factors. These include the severity of the symptoms, the underlying etiology, the reversibility of the condition, the presence of signs indicating unstable condition, and the risk of progression into asystole, also called cardiac flatline. The signs that indicate an unstable patient are signs of shock, syncope, heart failure, and myocardial infarction. Those patients are at risk of cardiac arrest and death and must be treated initially in accordance with the advanced cardiac life support (ACLS) guidelines and principles. In the acute setting, increasing the heart rate is the top priority of the management team. Initial pharmacological therapy is by using atropine sulfate. Atropine is an antimuscarinic drug. It acts by reversing the cholinergic effect on the heart that reduces the heart rate. The recommended dose in adults is 0.5 mg intravenously, repeated 3-5 times to a maximum dose of 3 mg if necessary. In cases where atropine is ineffective, β-adrenergic agonists can be used. Examples of these include dopamine and epinephrine. If the patient remains unstable, temporary pacing should be initiated. There are multiple types of temporary pacing. However, the most commonly used one in the acute setting is transvenous pacing. Furthermore, this type requires sedatives or analgesics as the procedure is painful.2
In our case, from the anamnesis, the patient complained of frequent fainting and during treatment the patient experienced a loss of consciousness, on physical examination the patient's pulse rate was 50 beats per minute (regular, sufficient contents), then on supporting examination, namely electrocardiography with the impression of sinus rhythm Bradycardia. From the anamnesis, physical examination and electrocardiography results, the patient was diagnosed with symptomatic bradycardia. Because the patient has signs of changes in mental status, namely a decrease in consciousness, the patient is given management in the form of atropine sulfate 1 mg every 5 minutes three times with a target pulse rate of 60 times per minute.

Meningitis is defined as inflammation of the meninges. This process may be acute or chronic and may result from infective or non-infective stimuli. A wide range of infective agents have been shown to cause meningitis, including viruses, bacteria, fungi and parasites. Encephalitis is part of the spectrum of inflammatory diseases of the central nervous system, characterised by evidence of an inflammatory process involving brain parenchyma. Encephalitis has over 100 causes, including viral infections (the majority), infection associated with other microorganisms and immune-mediated conditions (including post-infectious inflammatory processes). The time course of disease may be acute (most viral encephalitis), subacute, or chronic. Viral encephalitis is usually acute and is often associated with some elements of meningitis (i.e. meningoencephalitis), although neck stiffness occurs in less than one in three cases. Most studies report that the aetiology of encephalitis is unclear in at least 40% of cases. AUK wide study on the Aetiology of Encephalitis found an infectious cause in 42% of cases most commonly herpes simplex virus (19%), varicella zoster virus (5%) and Mycobacterium tuberculosis (5%). A further 21% of cases had acute immune-mediated encephalitis and 37% were of unknown aetiology. Arboviruses and rabies, are common causes of meningoencephalitis in some parts of the world.

A useful case definition for encephalitis is encephalopathy (altered level of consciousness, cognition, behaviour or personality persisting for more than 24 hours) and two or more of the following:

- Fever or history of fever (≥38°C)
- Seizures and/or focal neurological findings
- CSF pleocytosis (>4 WBC/μL)
- EEG findings compatible with encephalitis
- Abnormal results of neuroimaging (with evidence of brain parenchyma involvement)

Most have fever, headache and changes to behaviour or level of consciousness. Prognosis may depend on early initiation of appropriate treatment and thus the importance of making an aetiological diagnosis cannot be overemphasized. A systematic approach should be followed for initial investigation, although clinical features, season and travel history are vital for formulating the differential diagnosis.6

Although a wide range of viruses have been reported to cause encephalitis, specific antiviral therapy for viral encephalitis is generally limited to disease caused by the herpes viruses, especially herpes simplex virus. Because the earlier that treatment is started for herpes simplex encephalitis, the less likely that death or serious sequelae will result, acyclovir (10 mg/kg intravenously every 8 h in children and adults with normal renal function) should be initiated in all patients with suspected encephalitis as soon as possible, pending results of diagnostic studies. Other empirical antimicrobial agents should be initiated on the basis of specific epidemiologic or clinical factors, including appropriate therapy for presumed bacterial meningitis if clinically indicated. In patients with clinical clues suggestive of rickettsial or ehrlichial infection during the appropriate season, doxycycline should be added to empirical treatment regimens.7

In our case, the patient complained of severe headache, followed by loss of consciousness, fever (38.8°C) and convulsions. On physical examination, a positive nuchal rigidity was found, a lumbar puncture was not performed because the patient was uncooperative and the family refused. So from the results of the examination, the patient was diagnosed with Meningoencephalitis et causa bacterial infection dd viral infection. The patient was given antibiotic therapy because of the suspicion that the cause of meningoencephalitis was bacteria. The patient was also given symptomatic therapy for fever and seizures in the form of paracetamol and anticonvulsants (phenytoin and clobazam).

According to Nicol et al, it is hypothesized that the sinus node dysfunction occurring during encephalitis results from lesions of the central nervous system and not from primary cardiac attempt. Indeed, in the fatal case, necropsy revealed no cardiac structure abnormalities especially in conduction pathways. Likewise, polymorphic Ventricular Tachycardia (VT) secondary to acquired long QT syndrome has been described in a 26-year old woman hospitalized for confirmed herpes encephalitis. VT was never detected again after antiviral therapy. As electrocardiogram, cardiac MRI and genetic studies were negative, the authors suggested that these cardiac manifestations were triggered by cerebral infection. Moreover, asystole or others cardiac arrhythmias have been also reported in case of seizures and ischaemic strokes, especially originating from temporal lobes, and could possibly be related to some unexplained sudden death observed in these situations. Neurological lesions (cortical and subcortical abnormalities including insular region) due to autoimmune mechanisms, such as anti-N-methyl-D-aspartate receptors (NMDAR) encephalitis, associated in many cases with tumors such as ovarian teratoma, have been also found to be associated with sinus node dysfunction, generally reversed by tumor extraction and/or immunosuppressive therapy. It's noteworthy that recent investigations indicate that herpes simplex encephalitis can trigger autoimmunity and then NMDAR encephalitis.

All these data highlight the fact that injury of insular cortex especially in its right side, that is thought to be involved in the control of autonomic system, can result in central sinus node dysfunction, whatever the aetiology. Anterior cingulated cortex seems to play a major role in the central autonomic network, but to our knowledge, lesions of this area have not been proved to be associated with cardiac disturbance. HSV encephalitis is a rare and possibly underestimated cause of reversible sinus node dysfunction. Hence, this association should be recognized when encephalitis is suspected, in order to systematically monitor cardiac rhythm, and to avoid unnecessary permanent cardiac pacing. Antiviral treatment should be promptly initiated, resulting in cardiac recovery and preventing severe neurological complications.8

4. Summary

A 38-year-old male patient has been reported with complaints of frequent fainting, pain in the back of the head, fever accompanied by decreased consciousness and had seizures. On physical examination, positive neck stiffness, low pulse rate and electrocardiographic examination showed sinus bradycardia. Patients diagnosed with meningoencephalitis with symptomatic bradycardia, atropine sulfate was chosen as therapy for the management of bradycardia in these patients. After being given therapy the patient's condition improved.

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