IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 2 Ser.8 (February. 2023), PP 33-37
www.iosrjournals.org

Paroxysmal Vagally Mediated AV Block with Recurrent Syncope: A Case report

Dr.Haless kamal, Dr.Jama Dounia, Pr. Habbal Rachida, Pr.Abdenasser Drighil, Pr. Leila Azzouzi
Service of Cardiology - CHU Ibn Roched of Casablanca-Morocco

Abstract
INTRODUCTION: Vagally mediated atrioventricular (AV) block is defined as a paroxysmal AV block, localized within the AV node, associated with slowing of the sinus rate. All types of second-degree AV block may be present. Vagally mediated AV block is benign, it can be recorded as an asymptomatic or symptomatic event (syncope/pre-syncope).
OBSERVATION: A 30 years old man, referred to our cardiology center after recurrent syncope. No particular medical history; his syncope has not associated with any other symptoms; no prodromal event was noted. On admission, he was conscious; hemodynamically stable. physical examination was typically normal. His basic ECG; Echocardiography and Laboratory examination were all normal. The precise cause of his syncope went undetected until an Holter-ECG revealed transient AV block. we also performed ECG-effort for searching AV block at effort which it was with no particularity. After all we test his autonomic nervous system which showed major vagal hyperactivity. Actually, he is under serious follow up and repeated Holter-ECG recordings which it showed well improvement after our advises in lifestyle modification and other measurements.
DISCUSSION: Vagally mediated atrioventricular (AV) block is defined as a paroxysmal first, second or third degree AV block associated with slowing of the sinus rate. It is therefore extremely likely that the site of vagally mediated AV block is within the AV node. Itis relatively benign, occurs secondary to a surge in parasympathetic activity. In some patients, the cause of vagal over-activity is identifiable, since it occurs during situations characterized by enhanced vagal tone, such as vomiting, coughing. Syncope is main accompanying symptom in approximately 40% patients affected by recent-onset persistent AVB. However, the prevalence of syncope due to paroxysmal AVB is probably under reported. In recent years, newly available long-term ECG monitoring devices have increased the diagnostic yield. Extrinsic vagal AVB (EV-AVB is typically treated with lifestyle modification, isotonic maneuvers, and rarely medication. We believe that, in patients with an asymptomatic vagally mediated AV block, pacemaker implantation is not indicated, since the phenomenon is benign. These patients should only be followed up in order to monitor the possible appearance of symptoms.
CONCUSION: Vagally mediated AV block is benign and the death appears to be an exceptional event and has never been demonstrated with certainty in humans. Nevertheless, vagally mediated AV block can cause syncope, which could be recurrent.

Keywords: *Syncope, Rythmology, Vagally BAV*

Date of Submission: 02-02-2023 Date of Acceptance: 13-02-2023

I. Introduction:

Vagally mediated atrioventricular (AV) block is defined as a paroxysmal AV block, localized within the AV node, associated with slowing of the sinus rate. All types of second-degree AV block, including pseudo-Mobitz II block, and complete AV block, may be present. Vagally mediated AV block is benign, it can be recorded as an asymptomatic or symptomatic event (syncope/pre-syncope). Syncope due to this form of AV block should be diagnosed and managed as neurally mediated syncope.

There is little published information on vagally mediated AV block, and in clinical practice it often goes unrecognized. In this article, we present young man with syncope reveledtransient second AV block.

II. Observation:

A 30 years old man, referred to our cardiology center after recurrent syncope. There were denying history of trauma, transfusion, food or drug allergy and poisoning also there was no special birth history with normal growth and development; his syncope has not associated with other symptoms; no prodromal event was noted. On admission, he was conscious; hemodynamic stable with normal blood pressure was 130/61 mmHg,

Pulse was regular with a heart rate of 61 beats/min. physical examination was typically normal. The response to carotid sinus pressure and Valsalva manoeuvres were tested. Hisbasic ECG and echocardiography also were normal (Fig.N1,Fig.N2) .Laboratory examination included blood routine, liver and kidney function, electrolytes, thyroid hormones (TSH, T3, T4), myocardial enzymes and cTNI (cardiac TroponinI, cTNI) were all normal.

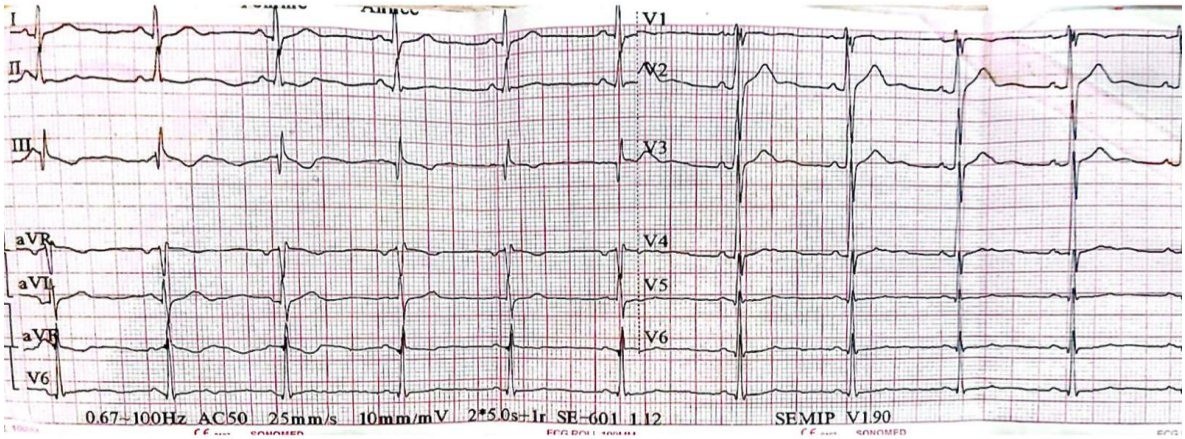

Fig.N1: shows his normal basic ECG

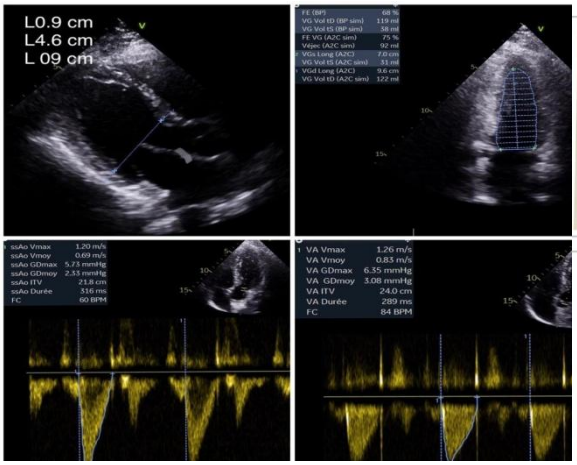

Fig.N2

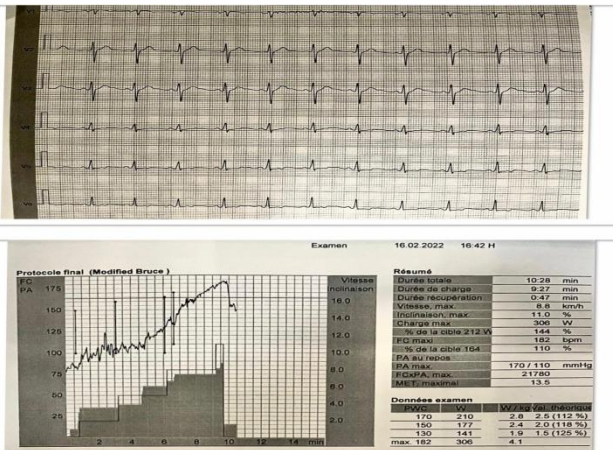

Fig.N3

Fig.N2: shows his normal echocardiography with EF estimated at 68%.
Fig.N3: Effort ECG without anomaly

We performed 24-h dynamic electrocardiogram which showed that P-P interval and RR interval had their own fixed rules, P wave and QRS wave had no fixed relationship, by which paroxysmal second-degree atrio-ventricular block Mobitz-II was diagnosed (Fig.N4). we alsocarried out ECG-effort for searching AV block at effort which it was with no particularity.

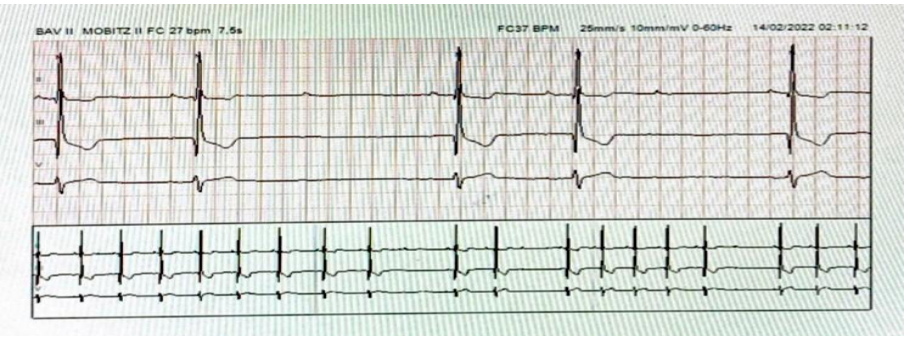

Fig.N4

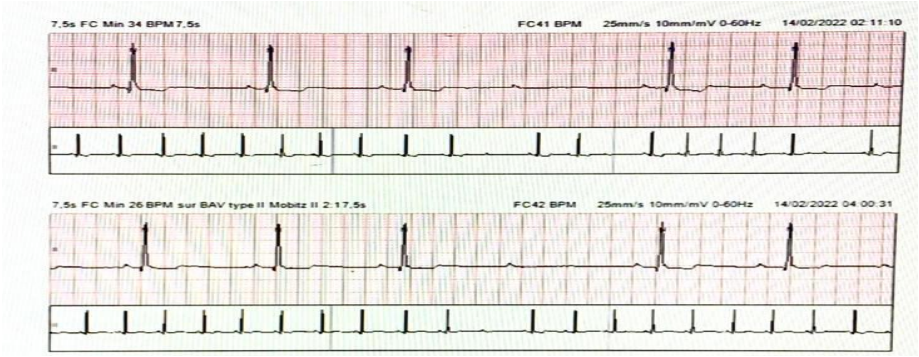

Fig.N5

FigN4-N5: HOLTER-ECG showed two events of second-degree atrioventricular block Mobitz-II associated with simultaneous slowing of sinus rate at 02:00 and 04:00 o'clock in the morning.

Our investigation was continued by testing his autonomic nervous system which showed major vagal hyperactivity. His autonomic profile in favor of:

- Baseline BP + HR is optimal
- vagal hyperactivity moderate to severe (Hand grip + dee breathing) on the ortho-test responsible of his BAV at the night
- Alpha and beta central sympathetic hyperactivity
- Alpha peripheral sympathetic hyperactivity
- No POTS or orthostatic hypotension

We suggest a therapeutic plan composed of lifestyle modification, isotonic maneuvers, salty diet, vitamin-therapy with magnesium, compression socks, but cardiotonic and veinotonic(Chlorhydrateetilefrine) was not prescribed; those measurement showed well improvement with no recurrent of his syncope after 2 months on follow-up. Our rythmopolehas not indicated pacing seen that vagally mediated atrioventricular block is relatively benign. Informed written consent was obtained from the patient.

III. Discussion:

Vagally mediated atrioventricular (AV) block is defined as a paroxysmal first, second or third degree AV block associated with slowing of the sinus rate. A vagal input depresses sinus node function and AV nodal conduction, but does not influence the velocity of conduction in the His-Purkinje system. It is therefore extremely likely that the site of vagally mediated AV block is within the AV node; the results of some electrophysiological studies during which this form of AV block has been reproduced confirm this site (1,2,3). There are no data on the prevalence of vagally mediated second or third degree AV block. Owing to its poor recognition and unpredictability, it is probably under-reported in the literature.

Vagally mediated paroxysmal AV block is relatively benign, occurs secondary to a surge in parasympathetic activity (4). In some patients, the cause of vagal overactivity is identifiable, since it occurs during situations characterised by enhanced vagal tone, such as vomiting, coughing, difficulty in swallowing and hiccups, but in the vast majority of patients the cause of the vagal surge is not evident (1-3).A differential diagnosis must be made between a vagally mediated AV block and an AV block secondary to anatomical involvement of AV conduction that is, an intrinsic AV block.

Syncope is a common symptom accounting for 1% of all admissions in emergency departments (5,6,7); Ant it is the main accompanying symptom in approximately 40% patients affected by recent-onset persistent AVB (9,10,11). However, the prevalence of syncope due to paroxysmal AVB is probably under reported (12). In recent years, newly available long-term ECG monitoring devices have increased the diagnostic yield (13,14). Vagally mediated atrioventricular (AV) block may be associated with identifiable triggers including vomiting, micturition, intense coughing, or phlebotomy, it may be asymptomatic, as noticed on Holter recordings, especially during nighttime (4).The universal underlying mechanism leading to syncope is a global transient cerebral hypoperfusion resulting from severe cardiac rhythm disturbances (bradycardia or tachycardia) and/or intense hypotension (8).Our patient presented with recurrent episodes of syncope, her electrocardiogram between the attacks was normal. The precise cause of syncope went undetected until an Holter-ECG revealed transient AV block.

The carotid sinus massage resulted in normal slowing of sinus node discharge in our case without precipitation of any AV block. Similarly, during Valsalva manoeuvre there was normal slowing of the sinus rate, the retching reflex failed to provoke AV block to our patient. It is well known that the effect of vagal

stimulation depends upon various factors, such as the intensity of stimulation, the method of stimulation, and the resting sympathetic activity (De la Fuente et al., 1969; Moore and Spear, 1976 (15,16). In the laboratory, it may thus be difficult to control all these factors to induce a spontaneous phenomenon, the association of nausea and vomiting with these episodes is intriguing.

Extrinsic vagal AVB (EV-AVB), also known as vasovagal syncope (or neuro-cardiogenic syncope), which is typically treated with lifestyle modification, isotonic maneuvers, and rarely medication (4). In contrast intrinsic AVB (I-AVB) and extrinsic idiopathic paroxysmal atrioventricular block (EI-AVB) treated with pacemaker implantation (22,23).Our patient elucidate this entity of AV block as a cause of her syncope (EV-AVB).The effect of treatment with theophylline in prevention of syncopal recurrences has been investigated, A few small observational studies on patients with reflex syncope treated with theophylline have recorded a recurrence rate ranging between 12% and 22% [17,18,19].We believe that, in patients with an asymptomatic vagally mediated AV block, pacemaker implantation is not indicated, since the phenomenon is benign. These patients should only be followed up in order to monitor the possible appearance of symptoms (20). Our patient did not undergo pacemaker implantation. actually, he is under serious follow up and repeated Holter-ECG recordings which it showed well improvement after our advises in lifestyle modification and other measurements.

IV. Concusion:

Even in the absence of robust evidence, there is a general consensus that vagally mediated AV block is benign because it is localised within the AV node and not in the His-Purkinje system and also, and especially, because it is not an expression of anatomical involvement of AV conduction. Moreover, a vagally mediated death appears to be an exceptional event and has never been demonstrated with certainty in humans (21). Nevertheless, vagally mediated AV block can cause syncope, which could be recurrent.

Les References:

[1]. Baron SC, Huang SK. Cough syncope presenting as Mobitz type II atrioventricular block. An electrophysiologic correlation. Pacing ClinElectrophysiol1987;10:65–9.
[2]. Nakagawa S, Koiwaya Y, Tanaka K. Vagally mediated paroxysmal atrio-ventricular block presenting as 'Mobitz type II' block (Letter). Pacing ClinElectrophysiol 1988;11:471–2.
[3]. Kakuchi H, Sato N, Kawamura J. Swallow syncope associated with complete atrioventricular block and vasovagal syncope. Heart 2000;83:702–4.
[4]. Bansal R, Mahajan A, Rathi C, Mehta A, Lokhandwala Y. What is the mechanism of paroxysmal atrioventricular block in a patient with recurrent syncope? J Arrhythmia. 2019;35:870–872
[5]. Blanc JJ, L'Her C, Touiza A, Garo B, L'Her E, Mansourati J. Prospective evaluation and outcome of patients admitted for syncope over a 1 year period. Eur Heart J 2002;23: 767–8.
[6]. Farwell D, SulkeN. Howdowediagnosesyncope?JCardiovascElectrophysiol 2002;13: 9–13.
[7]. Disertori M, Brignole M, Menozzi C, Raviele A, Rizzon P, Santini M et al. Management of patients with syncope referred urgently to general hospitals. Europace2003;5: 283–91.
[8]. Moya A, Sutton ,Michele Brignole, Angel Moya, Frederik J de Lange, Jean-Claude Deharo, Perry M Elliott, Alessandra Fanciulli,ArturFedorowski, Raffaello Furlan, Rose Anne Kenny, Alfonso Martín , 2018 ESC Guidelines for the diagnosis and management of syncope , *EuropeanHeart Journal*, Volume 39, Issue 21, 01 June 2018, Pages 1883–1948.
[9]. Aste M., Oddone D., Donateo P. Syncope in patients paced for atrioventricular block. *Europace.* 2016;18:1735–1739.
[10]. Langenfeld H., Grimm W., Maisch B. Course of symptoms and spontaneous ECG in pacemaker patients: a 5-year follow-up study. *Pacing ClinElectrophysiol*. 1988;11:2198–2206.
[11]. Proclemer A., Ghidina M., Gregori D. Trend of the main clinical characteristics and pacing modality in patients treated by pacemaker: data from the Italian Pacemaker Registry for the quinquennium 2003–07. *Europace.* 2010;12:202–209.
[12]. Lee S., Wellens J.J., Josephson M. Paroxysmal atrioventricular block. *Heart Rhythm.* 2009;6:1229–1234.
[13]. Brignole M., Vardas P., Hoffmann E. Indications for the use of diagnostic implantable and external ECG loop recorders. *Europace.* 2009;11:671–687.
[14]. Locati E.T., Moya A., Oliveira M. External prolonged electrocardiogram monitoring in unexplained syncope and palpitations: results of the SYNARR-Flash study. *Europace*. 2016;18:1265–1272.
[15]. De la Fuente D, Jedlicka T, Moe CK: Time course of vagal effects on S-A and A-V nodes (abstr.). Fed Proc 28, 269(61) (1969) .
[16]. Moore EN, Spear JF: Effect of autonomic activity on pacemaker function and conduction. In me Conduction System of the Heart. Structure, Function and Clinical Implications. (Eds. Wellens HJJ, Lie KI, Janse MJ). Leiden, He StenfertKroese BV (1976) 100
[17]. Šinkovec M., Grad A., Rakovec P. Role of endogenous adenosine in vaso-vagal syncope. *ClinAuton Res.* 2001;11:155–161
[18]. Benditt D.G., Benson W., Kreitt J. Electrophysiologic effects of theophylline in young patients with recurrent symptomatic bradyarrhythmias. *Am J Cardiol.* 1983;52:1223–1229.
[19]. Brignole M., Gaggioli G., Menozzi C. Adenosine-induced atrioventricular block in patients with unexplained syncope:the diagnostic value of ATP testing. *Circulation.* 1997;96:3921–3927.
[20]. Vardas PE, Auricchio A, Blanc JJ, et al. Guidelines for cardiac pacing and cardiac resynchronization therapy. The Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Developed in collaboration with the Heart Rhythm Association. Eur Heart J 2007;28: 2256–95.
[21]. Alboni P, Alboni M, Gianfranchi L. Simultaneous occurrence of two independent vagal reflexes: a possible cause of vagal sudden death. Heart 2011;97:623–5.
[22]. Lange HW, Ameisen O, Mack R, et al. Prevalence and clinical correlates of non-Wenckebach, narrow-complex second-degree atrioventricular block detected by ambulatory ECG. Am Heart J 1988;115:114–20.
[23]. Barold SS, Hayes DL. Second degree atrioventricular block: a reappraisal. Mayo Clin Proc 2001;76:44–57.

Dr.Haless kamal, et. al. " Paroxysmal Vagally Mediated AV Block with Recurrent Syncope: A Case report." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 22(2), 2023, pp. 33-37.