Reversible left ventricular dysfunction due to endless loop tachycardia in patient with dual chamber pacemaker- A case report

Vikrant Khese a, Chandrakant Chavan b, *, Rajesh Badani c

a Bharati Hospital and Research Centre, Pune, India
b Dept. of Cardiology Bharati Hospital and Research Centre, Pune, India
c Dept. of Cardiology Aditya Birla Hospital, Chinchwad, Pune, India

A 60 years male patient underwent permanent pacemaker [DDDR -with dual chamber pacing (D) with dual chamber sensing (D) with dual mode of response (D) with rate responsive pacing(R) -St Jude's medical (Abbott- Endurity 2160)] implantation for complete heart block (CHB). After 4 months patient was admitted for congestive heart failure. 12 Lead electrocardiograms (ECG) was suggestive of tachycardia at 130 beats per minute (regular rhythm), with ventricular complexes preceded by pacing spikes and maintenance of 1:1 atrio-ventricular relationship. Echocardiography showed global hypokinesia of left ventricular (LV) myocardium with reduced LV ejection fraction. LV dysfunction and heart failure were attributed to tachy-cardiomyopathy. Pacemaker telemetry data demonstrated that the tachycardia was likely to be pacemaker-mediated endless loop tachycardia (ELT). ELT in this case was perpetuated secondary to shortening of post ventricular atrial refractory period (PVARP), intact retrograde ventriculo-atrial (VA) conduction and addition of antiarrhythmic drugs prolonging retrograde VA conduction. Rate response (Dynamic) PVARP was reprogrammed allowing PVARP extension. Following this ELT was terminated. LV ejection fraction was normalized on subsequent follow up visit after seven days.

1. Case report

A 60 years old male patient with history of diabetes mellitus, systemic hypertension and implanted permanent pacemaker (DDDR St Jude's medical-Abbott Endurity 2160) for complete heart block (4 months back), presented with symptoms of shortness of breath (New York Heart Association class IV dyspnea), palpitations, and fatigue for 2 months. In view of tachycardia patient was started on betablocker and Sotalol for last 2 months at local care center. Chest X ray was suggestive of pulmonary venous congestion. ECG was suggestive of rapidly paced ventricular rhythm (pacing spike preceded the QRS complex) and small bleb seen before T wave, likely retrograde P wave with intact 1:1 VA conduction (Fig. 1A). Echocardiography showed global hypokinesia of left ventricular (LV) wall with reduced left ventricular ejection fraction (25–30 %). Differential diagnosis of atrial tachycardia and pacemaker mediated tachycardia (PMT) was made. Further device interrogation was suggestive tachycardia at heart rate of 130 beats per minute. There was regular VA relationship with atrial complexes seen after PVARP. This retrogradely conducted P wave is sensed by an atrial channel initiates atrio-ventricular (AV) interval resulting in ventricular pacing. This creates an endless loop: forward from atrium and paced ventricle, then backward from ventricle to atrium. Each retrograde P-wave triggers a ventricular paced event. This is possible in setting of CHB when there is intact retrograde VA conduction This loop recurs in cyclical fashion resulting in endless loop tachycardia (ELT) (Figs. 1B and 2). In this patient this was further increased due to addition of anti-arrhythmic like betablocker.

In St Jude’s Pacemaker (Abbott- Endurity 2160) rate responsive PVARP was reprogrammed at low setting (allowing PVARP extension). Simultaneously antiarrhythmics were stopped. Patient was treated with small dose diuretic. After 7 days patient became asymptomatic. Left ventricular ejection fraction was normalized. There was improvement of symptoms after reprogramming PVARP setting and termination of ELT. LV systolic dysfunction in this
patient was related to tachycardiomyopathy due to pacemaker-mediated endless loop tachycardia.

In this pacemaker, St Jude’s medical (Abbott-Endevity 2160) with rate responsive PVARP ventricular refractory period can be programmed Low, Medium and High setting. The rate responsive PVARP feature automatically adjusts PVARP settings in a way that guarantees a certain atrial alert period during increases and decreases in heart rate. The guaranteed atrial alert period is a percentage of the higher programmed value of either the Maximum Tracking Rate (MTR) or Maximum Sensing Rate (MSR). Rate responsive PVARP becomes active when the intrinsic rate or pacing rate exceeds 60 beats per min, whichever is higher. As the rate increases, the algorithm shortens PVARP until the MSR, MTR, or the programmed shortest PVARP is reached. This device allows timing reserve of 10% (Low), 20% (Medium), 30% (High) at MTR or MSR if rate responsive sensor is programmed for atrial alert period. There will be linear decrease of refractory period from base rate or 60 bpm, whichever is higher.

In present case, changing rate responsive PVARP and stopping anti-arrhythmic has eliminated tachycardia which favors diagnosis of PMT over atrial tachycardia. In contrast, a VP-AR-AS-VS/VP (depending on AV conduction) response will be expected in an atrial tachycardia. The LV systolic function and NYHA class improved on tachycardia elimination. LV systolic dysfunction was related to tachycardiomyopathy in this patient.

2. Discussion

Pacemaker-mediated tachycardia, commonly known as endless-loop tachycardia, is seen in patients with repetitive retrograde VA conduction. PMT can only occur in DDD or VDD pacing modes (P-synchronous ventricular pacing) and has also been referred to as pacemaker circus movement tachycardia, repetitive re-entrant pacemaker VA synchrony, re-entrant VA pacemaker tachycardia, ELT or antidromic re-entrant dual chamber pacemaker tachycardia [1]. It is a tachycardia resulting from the sensing of retrograde P-waves by the pacemaker, consequently triggering ventricular pacing beyond the programmed PVARP. PMT can be induced by retrograde P waves due to ectopic ventricular beats, ectopic atrial beats, atrial sensing failure, atrial pacing failure, and long AV delay.

The incidence of retrograde VA conduction was reported in 80% of Sick Sinus Syndrome cases and in 35% of AV block cases [1]. Another study reported that the incidence of VA conduction was 32% in second degree AV block, and 14% in complete AV block [2]. Even in complete AV block, some patients who were implanted with a physiological pacemaker have a risk of ELT [3]. The rate of arrhythmia depends on the VA conduction time, the programmed

![Image](image_url)
MTR and the AV interval. It typically occurs at the MTR of the device when the sum of VA interval and AV interval is less than the upper rate interval. The device extends the AV interval to follow the MTR, dynamic AV delay. These changing intervals are called as dynamic interval.

In current pacemakers, a heart rate or SIR (sensor indicated rate) determined dynamic PVARP adjustment may be enabled. In tracking pacing modes, PVARP is extended during lower heart rates in order to protect against pacemaker-mediated tachycardia, while it is shortened at higher rates to allow P-synchronous ventricular pacing at faster rates (allows the programming of a higher MTR) and to reduce the likelihood of competitive atrial pacing. In non-tracking pacing modes, PVARP is extended to prevent inhibition of atrial pacing by an atrial event early during the VA interval, and shortened at high SIR to reduce the likelihood of competitive atrial pacing.

These changes in interval occur due to various sensing features in pacemaker such as sudden change in native heart rate, change in thoracic impedance etc. Due to this dynamic function, sometimes PVARP may shorten and retrogradely conducted P wave may fall outside this dynamic PVARP and PMT may be initiated.

In present case possible mechanism for initiation of tachycardia could be due to combination of atrial premature complex arising closure to atrioventricular (AV) node and shortening of PVARP in next beat due to rate responsive function (Fig. 3 A & B). In this tracing 8th atrial event could be atrial premature complex close to AV node which takes some time for it to be sensed by atrial electrode in right atrial appendage. This means atrial premature beat has occurred few milliseconds before actual sensing of P wave by right atrial lead in right atrial appendage. However, by this time ventricular paced event occurs and AV node may allow retrograde conduction leading to retrograde activation of atrium (eighth to eleventh intracardiac electrocardiograms are similar to each other but different from sinus P wave electrocardiogram i.e., narrow complex with deep S wave suggesting retrogradely depolarized atrial beat). Here due to retrograde VA conduction, the P wave is seen 280 milliseconds after paced ventricular event and it is sensed (as rate responsive PVARP would decrease with increasing atrial rate), which initiates next AV Delay (Sensed AV delay was programmed 180 ms at this time) resulting in endless loop tachycardia.

In this case, patient had sinus rhythm, maximum tracking rate is 130 beats per minute rate response PVARP is Programmed at High, so 30 % will be the alert period of MTR interval. PVARP shortening starts at 60 bpm or the programmed base rate whichever is higher. Cycle length at MTR is 462 milliseconds (i.e., 60000/130). Sensed AV Delay is 180 milliseconds. As 30 % of maximum tracking rate is 139 milliseconds. The minimum PVARP will be 462-180=143 milliseconds of PVARP at MTR.

In ELT, VA conduction works as the retrograde limb and consequent atrial tracking by the pacemaker as the antegrade limb of the macro-reentrant circuit [4,5]. Therefore, an atrial sensing ventricular pacing sequence will be observed (Fig. 3 B). Any condition which separates the P wave from the QRS complex (paced or spontaneous) in a patient with retrograde VA conduction may initiate PMT [1,6]. Once established, this re-entrant mechanism continues until it is interrupted or until the retrograde limb of the circuit is exhausted. The ventricular pacing rate in response to sensed atrial activity cannot violate the programmed maximum tracking rate. If the VA conduction time is sufficiently long, PMT could occur below the MTR. PMT may be asymptomatic if the programmed MTR is low; however, symptoms may be significant if the MTR is programmed relatively high.

Preventive strategy for PMT: The facility to grant an adequate post ventricular atrial refractory period after a ventricular event is
the key to the prevention of endless loop tachycardia by ensuring that retrograde atrial activation will not be sensed. One of the oldest preventive strategy is PVARP extension after premature ventricular complex (PVC), since they are the most common triggers. Extension of the PVARP after a PVC prevents tracking of retrograde P wave and so P wave will fall within the PVARP. Although still PMT could also initiate despite PVARP extension after a PVC, due to ineffective atrial pacing soon after retrograde P wave (during the atrial repolarization period—Atrial Refractory Period), followed by ventricular pacing with retrograde VA conduction, initiating PMT.

It is important to address that if the triggering mechanism is atrial loss of capture, atrial under sensing or oversensing, premature atrial complex or magnet removal then the “PVARP extension” strategy will fail, and it will initiate a PMT. Secondly, there is way to diagnose and treat PMT rather than preventing it. Pacemaker assumes atrial sensed ventricular paced events at MTR as PMT. Further after diagnosing either inhibition of ventricular paced rhythm leading to failure of retrograde VA conduction or extension of PVARP leading to failure of sensing retrograde P wave. In St Jude’s pacemaker, PMT is confirmed if ventricle paced — atrial sensed interval remains stable (within 16 milliseconds of prior eight ventricle paced — atrial sensed intervals) despite shortening or increasing AV interval and if atrial sensed—ventricle paced interval is greater or shorter than 100 milliseconds, respectively. PMT is terminated after ventricular pacing is withheld, restoring AV synchrony with atrial pacing 330 ms after the last atrial-sensed event. Successful atrial capture breaks the cycle and prevents retrograde conduction after the next ventricular-paced complex. Table 1 shows various Algorithms to prevent, identify, and terminate
pacemaker-mediated tachycardia.

3. Conclusion

Endless loop tachycardia causing cardiomyopathy as an initial presentation is unusual. Pacemaker can be directly involved in initiating or sustaining various forms of arrhythmias. Appropriate modification in pacemaker parameters during device interrogation is essential to prevent or cure these arrhythmias.

Source of support

Nil.

Declaration of competing interest

Dr Vikrant Khese. Dr Chandrakant Chavan, Dr. Rajesh Badani declare that they have no conflict of interest. The manuscript has been read and approved by all the authors, that the requirements for authorship as stated earlier in this document have been met, and that each author believes that the manuscript represents honest work.

References

[1] Serge Barold S. Repetitive reentrant and non-reentrant ventriculoatrial synchrony in dual chamber pacing. Sep Clin Cardiol 1991;14(9):754–63.
[2] Klementowicz P. Ausubel K. Furman S. The dynamic nature of ventriculoatrial conduction: dynamique de la Conduction ventriculo-auriculaire. Nov Pacing Clin Electrophysiol 1986;9(6):1050–4.
[3] Duray GZ, Israel CW, Wegener FT, Hohnloser SH. Tachycardia after pacemaker implantation in a patient with complete atrioventricular block. Oct 1 Europace 2007;9(10):900–3.
[4] Doppalapudi H. Timing cycles of implantable devices. Jan 1 In: Clinical cardiac pacing, defibrillation and resynchronization therapy. Elsevier; 2017. p. 961–1030.
[5] Robledo-Nolasco RO, Ortiz-Avalos MA, Rodriguez-Diez GE, Castro-Villacorta HJ. Catheter ablation of accessory pathway in the treatment of pacemaker-mediated tachycardia. Apr Pacing Clin Electrophysiol 2012;35(4):e84–6.
[6] Monteil B, Ploux S, Eschalier R, Ritter P, Haissaguerre M, Koneru JN, Ellenbogen KA, Bordachar P. Pacemaker-mediated tachycardia: manufacturer specifics and spectrum of cases. Dec Pacing Clin Electrophysiol 2015;38(12):1489–98.

Table 1

Algorithms to prevent, identify, and terminate pacemaker-mediated tachycardia.

| ALGORITHM       | TRIGGER                                      | INTERVENTION                                      | PREVENT PMT | DIAGNOSE AND TREAT PMT |
|-----------------|----------------------------------------------|---------------------------------------------------|-------------|------------------------|
| PVC RESPONSE    | “PVC” beat (R without a preceding P wave)    | Programmed PVARP is extended immediately after sensed PVC beat | Yes         | No                     |
| PMT TERMINATION | 16 successive VP at MTR following AS events. VA interval stability: PMT is declared if all the 15 VA intervals are <32 ms longer or shorter than first VA interval | Extend PVARP to fixed 500 ms for one cardiac cycle to break PMT | No          | Yes                    |
| PMT INTERVENTION| Nine consecutive VP events of <400 ms that end with an AS event. On eighth consecutive VA interval, activity sensor is checked (assess-exercise-related tachycardia); PMT is declared if sensor-indicated rate less than pacing rate and intervention will occur | Forces a 400-ms PVARP extension after the ninth VP event | No          | Yes                    |
| PMT PROTECTION  | Eight consecutive VP events in which atrial events lie within a programmed PMT VA criterion (default 350 ms). PMT is confirmed by stable VA interval after decreasing MTR by 10 bpm or shortening AVI to next programmable length by 10 bpm | PVARP extension by measured V–V interval plus 50 ms | No          | Yes                    |
| PMT RESPONSE    | Eight consecutive VP–AS events above PMT detection rate. PMT is confirmed if VP–AS interval remains stable (within 16 ms of prior eight VP–AS intervals) despite shortening or increasing AVI by 50 ms if AS–VP interval is greater or shorter than 100 ms, respectively | Suspends VP event and delivers atrial pulse at 330 ms after detected retrograde P-wave | No          | Yes                    |

AS, atrial-sensed event; AV, atrioventricular; AVI, AV interval; PVC, premature ventricular contraction; SIR, sensor-indicated rate; VP, ventricular paced event.