

Diltiazem: A Reversible Cause of Atrioventricular Block – Until Proven Otherwise

Konstantinos C. Koskinas*, Leonidas Lillis and Antonios Ziakas

Cardiology Department, AHEPA Hospital, Aristotle University of Thessaloniki, Greece

Drugs are considered a common reversible cause of rhythm conduction disorders. Atrio-ventricular (AV) block occurs more commonly following β -blocker or digitalis administration, but calcium channel blockers have also been linked to AV block [1,2]. The cornerstone of management in drug-related AV block is to determine whether the AV block is truly reversible upon withdrawal of the culprit drug, hence implantation of a permanent pacemaker can be avoided.

Brenes and colleagues report an interesting case of complete AV block in an elderly patient who received diltiazem [3]. The patient's deteriorating renal function and his advanced age were considered as possible precipitating factors of diltiazem-induced AV conduction abnormality. The AV block resolved following intravenous calcium administration. While the temporal sequence of drug interruption, calcium administration, and sinus rhythm restoration in this case³ indeed strongly suggests a cause-and-effect relation between diltiazem and AV block, some caution is still required. Previous evidence from large case series indicates that the majority of patients for whom drug discontinuation leads to resolution of AV block have recurrence of AV block in the absence of therapy [4,5]. Conversely, drug discontinuation is followed by resolution of AV block in 41% of cases, but spontaneous improvement of AV conduction also occurs in 23% of patients who have AV block in the absence of drugs [4], suggesting that resolution of AV block upon drug interruption does not necessarily prove that the AV block represents an exclusively adverse drug effect of the AV blocker. In fact, it has been reported that AV block is truly caused by drugs in only 15% of patients with 2nd 3rd degree AV block during therapy with beta-blockers, verapamil or diltiazem.⁶ In most patients receiving AV blockers, AV block may actually be triggered due to underlying AV conduction disease [6]. As a consequence of the high risk of AV block recurrence despite the discontinuation of the

suspected culprit drug, about half of patients who receive AV blockers and develop AV block ultimately require implantation of a permanent pacemaker [6].

In conclusion, in patients receiving drugs that affect AV conduction AV block is very likely to be merely drug-related rather than truly drug-induced. Therefore, AV blocking drugs should be considered as reversible causes of benign, intermittent AV block, but only until proven otherwise.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflicts of interest.

ACKNOWLEDGEMENTS

None declared.

REFERENCES

- [1] Hayes DL, Zipes DP. Cardiac pacemakers and cardioverter defibrillators. In: Braunwald E, Libby P, Bonow RO, Mann DL, Zipes DP Eds. Heart Disease: A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia, PA: WB Saunders Company 2008; p. 832.
- [2] Mangrum JM, DiMarco JP. The evaluation and management of bradycardia. *N Engl J Med* 2000; 342: 703-9
- [3] Brenes JA, Cha YM. Diltiazem-induced transient complete atrioventricular block in an elderly patient with acute on chronic renal failure. *Open Cardiovasc Med J* 2013; 7: 23-7
- [4] Zeltser D, Justo D, Halkin A, *et al.* Drug-induced atrioventricular block: Prognosis after discontinuation of the culprit drug. *J Am Coll Cardiol* 2004; 44: 105-8
- [5] Shohat-Zabarski R, Iakobishvili Z, Kusniec J, Mazur A, Strasberg B. Paroxysmal atrioventricular block: Clinical experience with 20 patients. *Int J Cardiol* 2004; 97: 399-405
- [6] Osmonov D, Erdinler I, Ozcan KS, *et al.* Management of patients with drug-induced atrioventricular block. *Pacing Clin Electro-Physiol* 2012; 35: 804-10

Received: May 09, 2013

Accepted: May 10, 2013

© Koskinas *et al.*; Licensee Bentham Open.

This is an open access article licensed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted, non-commercial use, distribution and reproduction in any medium, provided the work is properly cited.

*Address correspondence to this author at the Cardiology Department, AHEPA Hospital, Aristotle University of Thessaloniki, 1 St Kyriakidi Street, 54636 Thessaloniki, Greece; Tel/Fax: +30 2310994837; E-mail: kckoskinas@gmail.com