



Kasuistika | Case report

Pitfalls in rate and rhythm control: Severe concomitant orthostatic hypotension unmasks after conversion to sinus rhythm

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SOUHRN

U pacientů s recidivující fibrilací síní představuje úprava srdeční frekvence atraktivní léčebnou strategii; zpravidla je jednodušší než úprava srdečního rytmu. Po dosažení optimální úpravy komorové frekvence zůstávají pacienti s dlouhodobě trvající fibrilací síní běžně v dobrém klinickém stavu a není třeba je následně opět hospitalizovat či měnit předepsanou medikaci a její dávky. Popisujeme případ neúspěšného použití strategie poměrně dlouhodobě účinné úpravy srdeční frekvence v důsledku převedení fibrilace síní na atypický flutter síní. Následná spontánní konverze na sinusový rytmus sice vedla ke zlepšení hemodynamických poměrů pacienta, odhalila však současně přítomnou závažnou ortostatickou hypotenzi s významnými důsledky pro léčbu pacienta.

Učební cíl: Transformace ve flutter síní může být příčinou neúspěchu strategie, jejímž cílem je úprava srdečního rytmu, a to dokonce i u stabilních pacientů s dlouhodobě perzistentní fibrilací síní. Spontánní konverze na sinusový rytmus může odhalit ortostatickou hypotenzi. Léčba ortostatické hypotenze jako přidruženého onemocnění může být náročná, protože hypotenze může být závažná a může mít výrazný vliv na stav pacientů.

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ABSTRACT

Rate control is an attractive strategy in management of patients with recurrent atrial fibrillation. Typically, it is more simple approach than rhythm control. Once optimal ventricular rate control is achieved patients with long-lasting atrial fibrillation commonly remain in good clinical status and do not require subsequent readmissions and change of prescribed drugs and their doses. We report a case of effective rate control strategy failure after relatively long period due to transformation of atrial fibrillation into atypical atrial flutter. Subsequent spontaneous conversion to sinus rhythm improved patient's hemodynamic but unmasked concomitant orthostatic hypotension that was severe and had significant impact on treatment of patient.

Learning objective: Transformation to atrial flutter may cause failure of rate control strategy even in previously stable patients with long time persistent atrial fibrillation. Spontaneous conversion to sinus rhythm can unmask orthostatic hypotension. Management of comorbid orthostatic hypotension might be challenging as it could be severe and have significant impact on patients' condition.

Keywords:

Atrial fibrillation and flutter

Orthostatic hypotension

Rate control

Spontaneous conversion

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Introduction

Among the most important decisions that must be made by a patient and care provider when choosing a treatment plan for atrial fibrillation (AFib) is the choice between rate and rhythm control. Typically, rate control is more simple strategy than rhythm control, involving the use of generally less toxic medications and fewer medical procedures. Stability is one of the most attractive features of rate control strategy. Once optimal ventricular rate control is achieved patients with long-lasting AFib commonly remain in good clinical status and do not require subsequent readmissions and change of prescribed drugs and their doses [1]. In our case effective rate control strategy failed after relatively long period of a 9-month AFib persistence due to transformation into atypical atrial flutter (AFL). Subsequent spontaneous conversion to sinus rhythm unmasked concomitant orthostatic hypotension (OH) that was severe and had impact on treatment of patient.

Case report

78-year-old Caucasian male admitted due to shortness of breath. His medical history is remarkable for hypertension and ischemic stroke in 2004. He had a history of paroxysmal AFib since 2005. In 2014 he was hospitalized due to recurrent episodes of AFib and received dual-chamber pacemaker for symptomatic ventricular asystole (up to 7.3 s). After rhythm-control strategy failed beta-blocker therapy was initiated. Patient was discharged on bisoprolol

2.5 mg OD with good ventricular rate control and within next 9 months he remained asymptomatic. Device follow-up showed permanent AFib and pacemaker was switched to VVI mode. There were no signs of device malfunction. Gradually increasing dyspnea developed 2 weeks ago.

Clinical investigation revealed hypervolemia (Ht 36.4%), pulmonary congestion and right-sided hydrothorax due to conversion of AFib into atypical AFL (mean ventricular rate 122 bpm). Torasemide 10 mg OD was initiated leading to 4 kg weight loss within 5 days. Bisoprolol dose gradually increased up to 5 mg BID but no rate control was achieved and digoxin 0.25 mg OD was started. Three days later patient spontaneously restored sinus rhythm, bisoprolol dose was reduced to 2.5 mg OD, digoxin and torasemide were discontinued. Within next 2 days patient lost additional 2 kg. Clinical investigation showed decreased hypervolemia (Ht 39.5%) and resolution of pulmonary congestion. Despite regression of dyspnea and hemodynamic improvement patient complained on progressive weakness. Two syncopes occurred next day. Active standing test confirmed severe OH (Fig. 1). Bisoprolol was discontinued and supine arterial hypertension was noted but orthostatic syncope recurred. Patient education, dietary modification and elastic compression of legs prevented syncope and decreased degree of supine arterial hypertension but severe OH persisted.

Discussion

A clinical interrelationship between AFib and AFL has long been appreciated. Patients who primarily manifest AFib

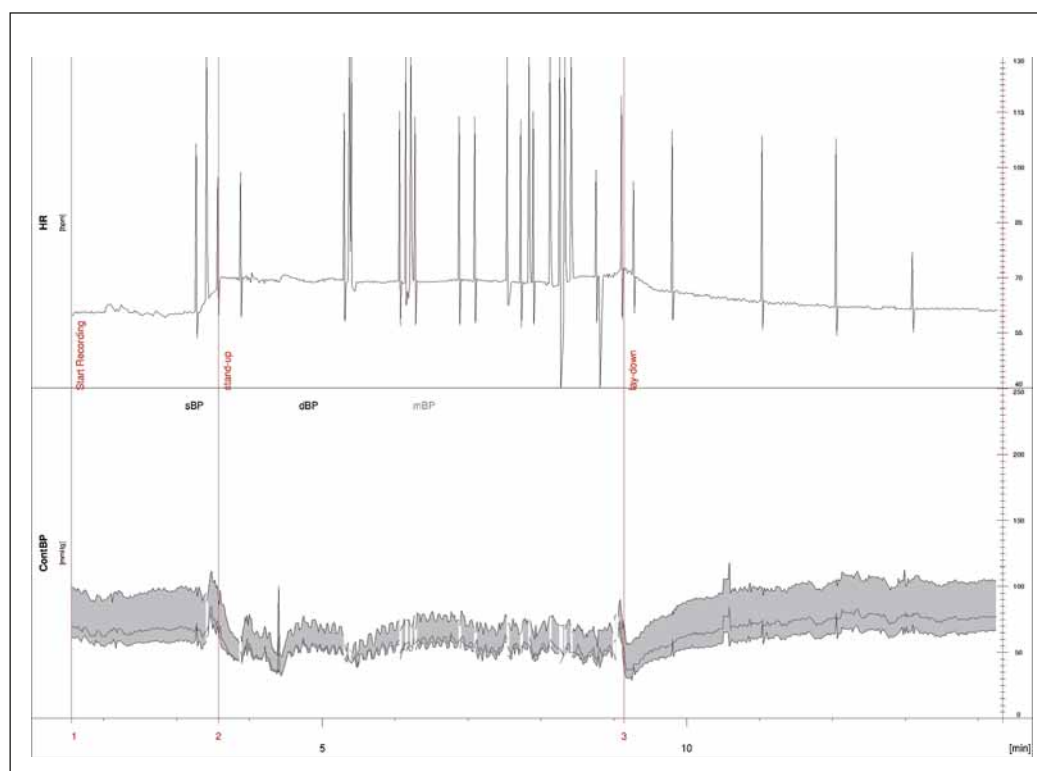


Fig. 1 – Continuous blood pressure (contBP) and heart rate (HR) monitoring during active standing test demonstrates severe orthostatic hypotension. Increase in heart rate during active standing excludes vasovagal syncope. sBP, dBP, mBP – systolic, diastolic and mean blood pressure.

commonly also manifest AFib and vice versa. These clinical associations are not coincidental but reflect linkage of their pathophysiologies and mechanisms [2]. Transformation to AFib is a known cause of patients' condition deterioration as patients with that type of arrhythmia usually have more rapid ventricular rate and worse response to atrioventricular (AV) nodal blocking agents than patients with AFib. However, conversion to AFib typically observed in recent-onset, not long-lasting AFib and commonly facilitated by antiarrhythmic drugs with sodium-channel-blocking properties prescribed for rhythm control not by beta-blockers used for rate control [3].

Spontaneous conversion to sinus rhythm is always possible therefore term "permanent" AFib usually represents a tactical decision to retain from sinus rhythm restoration and maintenance made by physician and patient, rather than a real pathophysiologic condition [1]. There is a trend that the longer the duration of current AFib episode, the less patient is likely to restore sinus rhythm [4]. Recent study of 356 patients with pacemaker devices capable of continuous atrial rhythm monitoring demonstrates that though many patients with AFib will have persistent episodes, the majority will have a paroxysmal, as opposed to persistent, form of the arrhythmia [5]. Despite numerous clinical trials enrolling thousands of patients with AFib data on spontaneous conversion of long-lasting AFib remains sparse and limited to case reports and small observational studies.

Previous publications postulate that spontaneous termination of long-lasting AFib predicts poor prognosis as it usually manifests progression of underlying heart disease and represents almost complete substitution of left atrial myocardium by fibrous tissue and frequently associated with severe AV and intraventricular conduction disturbances [6]. However, there are reports of spontaneous sinus rhythm restoration with preserved mechanical atrial function as indirect beneficial result of therapeutic interventions [7]. Such controversy may partly be explained by changes in concomitant patient's characteristics (nowadays severe rheumatic mitral valve disease became significantly less common in developed countries) and by advances in management of heart failure (use of anti-fibrotic drugs and endovascular and surgical interventions).

In our case patient restored sinus rhythm soon after initiation of digoxin. In 1981 Gardner and Dunn reported 4 cases of spontaneous termination of permanent AFib in patients, who received digitoxin [8]. Lately performed randomized placebo-controlled trial showed that digitalization does not affect the likelihood of reversion to sinus rhythm [9]. Though the drug could possibly contribute to sinus rhythm restoration, we consider it unlikely, because conversion occurred only 3 days after initiation of treatment and plasma level of digoxin was subtherapeutic (0.61 ng/ml).

Conversion to sinus rhythm improved hemodynamic and led to excessive fluid excretion and resolution of pulmonary congestion (patient lost additional 2 kg of weight despite withdrawal of torasemide and digoxin). Echocardiography showed preserved left atrium contractile function and no transient left ventricular systolic dysfunction that sometimes occurs after cardioversion of

persistent AFib. Despite improvement of hemodynamic, patient developed severe orthostatic hypotension (OH) with syncope.

Thorough clinical investigation revealed no endocrine, neurologic and metabolic disorders that are known to cause orthostatic hypotension. Administration of volume-depleting agent (torasemide) was unlikely to trigger OH in our case because it was used in standard therapeutic dose, it was discontinued before the onset of OH and clinical investigation demonstrated no signs of diuretic-induced dehydration. Bisoprolol was unlikely to cause OH since after conversion to sinus rhythm its' dose was decreased to 2.5 mg OD and patient had no symptoms of OH while taking that dose when he had AFib in previous 9 months. Furthermore, orthostatic syncope recurred after subsequent discontinuation of bisoprolol.

Orthostatic hypotension is a common cardiovascular disorder. Its' prevalence is age dependent, ranging from 5% in patients younger 50 years of age to 30% in those who are older 70 years of age. Orthostatic hypotension may cause disabling symptoms, faints, traumatic injuries and substantially reduce quality of life. Presence of OH independently increases mortality and the incidence of myocardial infarction, stroke, heart failure and atrial fibrillation [10].

Atrial fibrillation is the most common sustained arrhythmia. Its' prevalence increases with age up to 9% in patients older than 80 years [1]. Taking into consideration high prevalence of both conditions there is no surprise that they may coexist in one patient. However, data on clinical course, manifestation, prognostic value and therapeutic implications of such comorbidity remain sparse. OH is reported to be manifestation of AFib [10], though in our case persistent AFib concealed symptoms of concomitant OH.

Supine arterial hypertension represents common problem and therapeutic challenge in management of patients with OH since it is associated with increased cardiovascular complications and mortality and almost all drugs that are used to prevent orthostatic syncope exacerbate arterial hypertension [10]. Luckily, in our case non-pharmacologic approaches were sufficient to prevent recurrence of orthostatic syncope and to decrease the degree of supine hypertension.

Conflict of interest

None declared.

Funding body

None.

Ethical statement

Authors state that the research was conducted according to ethical standards.

Informed consent

Informed consent was obtained from the patient participating in this study.

Appendix A – Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:10.1016/j.crvasa.2016.08.005.

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