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HYPERTENSION AND ARRHYTHMIA

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Introduction

Arrhythmia-both atrial and ventricular-is a common comorbidity with hypertension (HT). Underlying mechanisms are many and various, including left ventricular hypertrophy (LVH), myocardial ischemia, impaired left ventricular function and left atrial enlargement. Any form of arrhythmia may be associated with LVH but ventricular arrhythmia is more common as well as being more dangerous.

Atrial arrhythmia

Prevalence

After supraventricular extrasystole, atrial fibrillation (AF) is the next most common form of arrhythmia associated with HT. The relative risk of developing AF in HT is modest compared with other conditions, such as heart failure and valve disease. Nevertheless, HT is the most prevalent, independent, and potentially modifiable risk for AF (1). AF is most common after the age of 65 and in men (2).

Mechanisms

Changes in atrial electrical properties occur early in hypertensive heart disease, preceding the appearance of left ventricular and left atrial enlargement (3).

Enlargement of the left atrium: Enlargement of the left atrium results in stretching of the atrial fibers which is what leads to the creation of arrhythmogenic foci. In the AFFIRM study, ultrasound measured a left atrium of normal size (diameter < 40 mm) in only 33% of patients (1). Left atrial enlargement seems to set in before LVH.

Left ventricular hypertrophy: LVH paves the way for AF by perturbing diastolic function and thereby raising the left atrial pressure (4). In the Framingham cohort, patients with an electrocardiographic diagnosis of LVH had a 3.0 to 3.8-fold increased risk of developing AF (5). Verdecchia et al. found that, in hypertensive subjects with sinus rhythm and no major predisposing conditions, the risk of AF increases with age and left ventricular mass whereas increased left atrial size predisposes to chronicization of AF (6).

Abnormal blood potassium levels: Blood potassium imbalance, especially hypokalemia (iatrogenic or secondary to hyperaldosteronism) can lead to the development of supraventricular arrhythmia.

Diagnosis and prognosis of atrial arrhythmia

Whenever a hypertensive patient complains of palpitations, the possibility of arrhythmia-supraventricular or ventricular-should be considered. Definitive diagnosis depends on resting ECG or ambulatory heart rate measurement over a period of 24-48 hours. Identifying causes may require cardiac Doppler ultrasound examination (to detect LVH, impairment of left ventricular function, left atrial enlargement or valve disease) and blood tests (potassium levels and high-sensitivity TSH test).

AF has many consequences. The most dangerous is arterial embolism, with stroke being four to five times more common in patients with AF (7, 8). Otherwise, AF can lead to cardiomyopathy and may exacerbate pre-existing impairment of left ventricular function (9). The onset of AF may trigger an episode of congestive heart failure, especially if the ventricular response is rapid or if there is some underlying problem with left ventricular function (either systolic or diastolic) (10). AF can also cause episodes of dizziness or even syncope. Finally, in the Framingham study, a correlation was observed between AF and mortality in both sexes, and this independently of other variables (11).

Treatment of atrial arrhythmia

Preventing AF in hypertensive subjects depends on controlling blood pressure in order to reduce the risk of hypertensive cardiomyopathy (or at least mitigating the consequences thereof). Antihypertensive therapy has been shown to reverse some of the structural cardiac changes caused by HT, including LVH and atrial enlargement (12, 13). ACE inhibitors and angiotensin receptor blockers may directly reduce the chance of the recurrence of AF.

Any potassium imbalance must be corrected. Moreover, anticoagulant therapy is essential in patients with AF. In contrast, the value of anti-arrhythmic drugs is more controversial. In practice, some physicians prefer to reduce the arrhythmia and then maintain a sinus rhythm, whereas others choose to work with the AF by controlling the heart rate (to between 60 and 90 beats per minute). Radiofrequency ablation of AF is a technique that will probably become more widespread in the near future.

Ventricular arrhythmia

Ventricular arrhythmia is usually triggered by simple or complex ventricular extrasystole whereas the mechanism whereby tachycardia is perpetuated more usually involves a re-entry circuit.

Arrhythmogenic factors

Left ventricular hypertrophy: Ventricular premature complex is more common in hypertensive subjects when there is concomitant LVH (14, 15). The most dangerous forms of ventricular arrhythmia (tachycardia and ventricular fibrillation) are still rare (16). Both the incidence and seriousness of these forms correlate with the severity of the LVH, as measured by ECG and ultrasound (17). Asymmetric septal and eccentric hypertrophy seem to be associated more often with ventricular arrhythmia than concentric LVH (18). That LVH is involved in the pathogenesis of ventricular arrhythmia is demonstrated by the fact that the incidence of the latter drops once the former has been reversed (19).

Myocardial ischemia: Myocardial ischemia is the most common arrhythmogenic factor, and this is also true in hypertensive subjects. This comorbidity increases the risk of sudden death. The ischemia may be secondary to atherosclerosis of the major epicardial coronary arteries, or due to problems in the myocardial capillary system. In the hypertensive subject, there is a link between the frequency and severity of arrhythmia, and myocardial ischemia (be the episodes symptomatic or subclinical) (20).

Impaired left ventricular function: The risk of arrhythmia in hypertensive patients is likewise exacerbated by impaired left ventricular function (systolic or diastolic) as a result of electrical asynchronism. This risk is further increased if the left ventricle is enlarged. As a general rule, at least two of the above-mentioned risk factors (LVH, myocardial ischemia or impaired ventricular function) need to be present for onset of the most dangerous forms of ventricular

arrhythmia in hypertensive subjects.
Other factors: Circadian variations and sudden increases in blood pressure can trigger arrhythmia as a result of associated changes in pre- and post-charge (21). Similarly, the sympathetic irritability which commonly accompanies HT can lead to ventricular arrhythmia (22). Whether or not variations in blood electrolyte levels (notably of potassium) also constitute an arrhythmogenic factor is more controversial (16, 23).

Diagnosis and prognosis of ventricular arrhythmia

Positive diagnosis depends on resting ECG and ambulatory heart rate measurement over a period of 24-48 hours. Amplified ECG (to detect late ventricular potentials) and programmed ventricular stimulation need not be performed on a systematic basis. Identifying underlying mechanisms will involve carrying out examinations to look for LVH (by ECG or ultrasound), myocardial ischemia (ECG or ultrasound stress testing, myocardial scintigraphy, Holter monitoring), heart failure or some underlying metabolic problem.

HT is associated with an increased risk of sudden death, essentially due to ventricular arrhythmia (24). In patients with LVH, global mortality is increased if there is complex or frequent ventricular extrasystole, even if this is asymptomatic (25).

Treatment of ventricular arrhythmia

If there is no myocardial ischemia, only the more severe forms of ventricular arrhythmia need positive management. However, if myocardial ischemia is present, this needs to be corrected as do frequent ventricular extrasystoles, ventricular doublets and salvoes. Blood potassium abnormalities should always be treated.

Beta-blockers and amiodarone are the drugs of choice in ventricular arrhythmia although calcium-channel blockers and angiotensin converting enzyme inhibitors have been shown to be effective against ventricular arrhythmia by virtue of their action against LVH (19, 23). Spironolactone may also be prescribed, not only to reverse hypokalemia but also for its antifibrotic activity in the ventricular myocardium. In patients with either severe ventricular arrhythmia, which has proven refractory to pharmacological treatment, or profoundly impaired ventricular function, an automatic implantable cardioverter defibrillator should be considered.

Conclusion

Both ventricular and atrial forms of arrhythmia are common in patients with HT. The underlying mechanisms are many and various, and the most useful diagnostic information comes from ambulatory heart rate monitoring. Arrhythmia needs to be treated on a case-by-case basis with objective criteria in sight.

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