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EDITORIAL COMMENT

Knowing the true nature of the cardiovascular effects of androgen deprivation therapy in prostate cancer: We still have a way to go. . .

Complicações cardiovasculares da terapêutica de privação androgénica no cancro da próstata: ainda temos um caminho a percorrer para o seu correto diagnóstico. . .

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The cornerstone of systemic treatment for prostate cancer is pharmacological or surgical androgen deprivation therapy (ADT):[1]

- For patients with metastatic prostate cancer, ADT is the primary method of treatment.[2]
- ADT is also used in combination with local treatment modalities to achieve better cancer control or as an effective salvage therapy in patients who do not respond to local therapy.[2]
- As many as 46% of men with prostate cancer receive ADT at some point during their treatment.[2]

Pharmacological ADT refers to treatment with a gonadotropin-releasing hormone (GnRH) agonist or GnRH antagonist.[1] Suppression of androgen signaling can also be accomplished with androgen receptor inhibitors or cytochrome P450 17A1 inhibitors.[1]

Although ADT provides a means of effective cancer control, studies have raised concerns regarding the adverse effects of ADT on metabolism and subsequent cardiovascular risk.[2] The mechanisms through which ADT has been suspected to increase cardiovascular risk include increased fat mass, increased low-density lipoprotein and total cholesterol, increased triglycerides, and increased insulin resistance.[2]

Cardiovascular disease represents the most common comorbidity and cause of death among patients with prostate cancer.[3] ADT had positive associations with cardiovascular events, cardiovascular death, and myocardial infarction.[1]

A number of studies have demonstrated that ADT is associated with fatal and nonfatal cardiovascular disease events that encompass ischemic heart disease, myocardial infarction, sudden cardiac death or ventricular arrhythmias, cerebrovascular accidents, peripheral artery disease, and venous thromboembolism.[3] Increased risk seems to occur regardless of whether the patient is administered short- or long-term ADT.[3]

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Epstein et al. reported that death from ischemic heart disease is the most common noncancer death in patients with prostate cancer on ADT.[3]

The authors present a case that seems to be an example of acute myocardial infarction in an octogenarian patient on ADT with goserelin and bicalutamide. Goserelin is a GnRH agonist and bicalutamide an androgen receptor inhibitor.

They show other lesser-known manifestations of ADT that include QT interval prolongation and changes in repolarization presenting in a patient with chest pain.

Repolarization abnormalities and QT interval prolongation have been described with goserelin administration[4] and chest pain has been documented with bicalutamide.[4]

However, the question remains, despite the absence of coronary disease, as to whether the chest pain and inversion of T waves would correspond to ischemia or not.

The clinicians in this case decided to discontinue treatment with the drugs that caused the androgen deprivation and cardiac semiology regressed. However, we do not know what the subsequent attitude was. Did they switch to a GnRH antagonist?

QT prolongation is a drug class effect due to androgen deprivation.[5] Prolongation of QT interval represents a delay in ventricular repolarization, which may lead to ventricular tachyarrhythmias, including torsade de pointes (TdP). TdP can degenerate into ventricular fibrillation, leading to sudden death.[5]

Looking at this clinical case of cardiovascular effects of ADT makes us think that there is still a way to go in the search for knowledge.

Conflicts of interest

The author has no conflicts of interest to declare.

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