

Editöre Mektup

Spontaneous right coronary artery dissection possibly associated with clonidine transdermal patch

Dear Editor,

I read with interest the case presented by Çilingiroğlu et al. recently published in your journal.^[1] They presented a case of spontaneous coronary artery dissection (SCAD) in a patient with normal coronary arteries on coronary angiography one week prior to presentation. Since the patient had been on clonidine transdermal 0.3-mg patch weekly for one month to control her hypertension and had recurrent vomiting, fatigue, and lightheadedness with substernal chest pressure that began in the morning of presentation after a long hot shower, the authors concluded that SCAD could be due to clonidine overdose. However, common clinical manifestations of clonidine toxicity include ataxia, dizziness, palor, dry mouth, lethargy, coma, respiratory depression, hemodynamic instability, miosis, hyporeflexia, hypotonia, bradycardia, hypotension, sinus bradycardia, complete heart block, and supraventricular tachycardia.^[2] Even if a clonidine patch is swallowed and all its content is absorbed, the signs of its overdose include obtundation and profound sinus bradycardia.^[3] Therefore, none of the patient's signs and symptoms could be due to clonidine overdose. Although it has been shown that increased absorption of clonidine is associated with increased cutaneous temperature,^[4] certainly, increased absorption cannot be the definite indicator of overdose. Even if it is, a cause-effect relationship between the clonidine overdose and SCAD cannot be confirmed. Also, none of the explanations given by the authors to show the role of clonidine overdose in SCAD is confirmatory of such a role. For instance, hypertension due to clonidine overdose has only been demonstrated in high-dose oral ingestion of clonidine^[5] or in cases with renal failure^[6] or the vasoconstriction of the peripheral vessels has only been shown in the intravenous administration of clonidine.^[7] Reflex coronary artery vasoconstriction secondary to alpha-receptor-mediated action has also been reported only in patients with coronary artery disease,^[8,9] which was not detected in this case. In

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my view, the best explanation for the occurrence of SCAD in this patient may be that it might have an idiopathic origin, which has been most commonly described in middle-aged, healthy women without any significant atherosclerosis or apparent risk factors for atherosclerosis.^[10] However, it should not be forgotten that the patient had stage III hypertension, representing a higher risk for SCAD.^[10]

Sincerely,

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The authors' reply

Dear Editor,

First of all, we thank our colleague for his letter to the editor regarding this case presentation. Several issues are to be discussed: the title of the case reads as "Spontaneous right coronary artery dissection possibly associated with clonidine transdermal patch". So any association between the use of clonidine patch in this patient and spontaneous coronary dissection (SCD) is potential rather than definite, which was discussed clearly in our case presentation throughout the text. However, it should also be kept in mind that previous literature well describes potential associations between reflex vasoconstriction due to other vasoactive substances and SCD. Thus, clonidine as being a centrally acting agent could be a potential cause of SCD in our patient because of the reasons discussed in the case. The gold standard for any causal relationship in a disease entity is the Koch's Postulate as defined by Dr. Robert Koch in 1884, which is beyond the scope of this case presentation. Even in today's medicine, we do call certain disease states as spontaneous even though there is clearly a tendency for a certain population or groups to have that association such as SCD in healthy middle-aged women. Even though we call it SCD, there are potential pathological pathways leading to them, as it is widely described in the literature, and there is a reason why so called SCD is seen in females rather than males, and why its incidence is increased in middle-aged pregnant women. So, basically indeed it is not spontaneous, there is certainly a reason, but we have not yet clearly defined it as it is in our case.

Regarding the presence of signs and symptoms of clonidine overdose in our patient, I think any potential association is most likely to be an effect-relationship rather than a full-blown clonidine overdose or toxicity. Also, as anything else in medicine that we have learned, not all the effects of clonidine overdose should be present in our patient, since it is most likely a clonidine effect-relationship rather than overdose.

Last but not least, SCD is usually seen in middle-aged females with no potential risk factors for CAD and also in those with healthy coronary arteries. In our patient, there was minimal CAD as reported by coronary angiography despite no significant plaque burden by IVUS. Of course, it was not a surprise for us since she had a long-standing report of significant hypertension.

While we thank the author for his interest and bringing up important points regarding this case presentation, we believe our case brings up an important discussion regarding the potential association of SCD with use of clonidine in daily clinical practice.

Sincerely,

On behalf of the authors,

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Dislodgement of a sirolimus-eluting stent in the circumflex artery and its successful deployment with a small-balloon technique.

Sayın Editör,

Türk Kardiyoloji Derneği Arşivi'nin 2011 yılı 5. sayısında yayımlanan ve sirkumfleks artere uygulanan PKG işlemi sırasında ilaç kaplı stentin proksimal bölümde takılması ve stentin daha düşük profilli bir

balonla şişirilerek hedef lezyona başarılı bir şekilde yerleştirilmesini bildiren olgu sunumunu^[1] ilgiyle okudum.

Bu tip olgularda, stent hedef lezyonun proksimal bölgesinde takıldığı taktirde, yöntem olarak stent ya düşük profilli bir balonla hafif basınçla şişirilerek hedef lezyona ulaştırılmaya çalışılır; stent ilerlemeyorsa ya geri çekilir ya da bulunduğu bölgede şişirilir. Bizim de bir olgumuzda, dominant sağ koroner