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Amplatzer ductal occluder should be preferred as a better and up-to-date choice of treatment in such cases.

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Author reply

Dear Editor,

We thank authors for their comments and concerns of our case report entitled "Pulmonary artery coil migration after management of patent ductus arteriosus in a 65-year-old female patient". It was published in June 2009 issue of Anatolian Journal of Cardiology.

There are several factors that determine a successful coil closure for PDA. The PDA size, PDA shape, and the degree of left-to right shunt and younger age may influence the results of coil occlusion of PDA (1). The complication occurred in this particular patient for two reasons. Firstly, the coil used for PDA closure was too small for the patient. Secondly, the aortic ampulla was large which made the coil unstable. 8 mm was the largest Cook coil. A second attempt of the coil of 8 mm in diameter was tried again. But unfortunately the coil seemed to move towards the pulmonary artery. The procedure of closure was stopped because of coil position was unstable. In conclusion, any interventionist who undertakes coil occlusion of the PDA should be familiar with the problem of migration, thoroughly equipped for foreign body removal, and skilled in the use of all types of equipment necessary to withdraw a foreign body from a pulmonary artery branch. As claimed by the authors, Amplatzer duct occluder may be considered for moderate to large PDAs (2).

Sincerely,

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Do bone morphogenic protein-4 antagonists have any role in the treatment of human hypertension?

Kemik morfojenik protein-4 antagonistleri insanlarda hipertansiyonun tedavisinde kullanılabilir mi?

To the Editor,

Bone morphogenic protein-4 (BMP-4) is originally identified as a regulator of cartilage and bone formation (1). However, BMP-4 transcripts are detected in the mesoderm around the developing gut and the myocardium (2). BMP-4 is found in calcified atherosclerotic plaques and it plays a role in calcification involving medial smooth muscle cells (3). BMP-4 might have a novel role in vascular inflammation in an endothelium-dependent manner.

Chronic BMP-4 infusion was showed to impair endothelium dependent vasodilatation in mice. It stimulated vascular NADPH oxidase activity and superoxide production which decreased endothelial nitric oxide (NO) bioavailability and led to hypertension in mice (4). 'Noggin' is the recombinant human BMP-4 antagonist that prevented the BMP-4 induced hypertension with mice. BMP4's role on the activation of NADPH oxidases and impairment of vasorelaxation has also been demonstrated along with the prevention of hypertension by apocynin treatment (Apocynin is the inhibition of the NADPH oxidases) (4).

The actual increase in arterial blood pressure is caused by an increase in systemic vascular resistance (SVR). Systemic vascular resistance refers to the resistance blood flow offered by all of the systemic vasculature in vascular beds. Mechanisms that cause vasoconstriction increase SVR and those mechanisms that cause vasodilatation decrease SVR.

Endothelial dysfunction with decreased NO production is known to be related to hypertension. The vascular NADPH oxidase contributes to endothelial dysfunction and high blood pressure in the spontaneously hypertensive rat by enhancing superoxide production (5).

Briefly, chronic BMP-4 infusion activates arterial NADPH oxidases and that this in turn leads to endothelial dysfunction and hypertension. As BMP-4 is a novel mediator of endothelial dysfunction and hypertension, Noggin, could prevent its effect in mice. To the best of our knowledge, there is no study whether BMP-4 antagonists could be an effective treatment in cases of human hypertension. Knowing the fact of required sophisticated studies to evaluate the role on blood pressure, on the current background, we would like to speculate that BMP-4 antagonists might propose a critical role as an effective antihypertensive medication by potential mechanisms of the suppression of the raised the systemic vascular resistance in human hypertension.

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Atriyal fibrilasyon ile komplike Tako-tsubo sendromu

Tako-tsubo syndrome complicating with atrial fibrillation

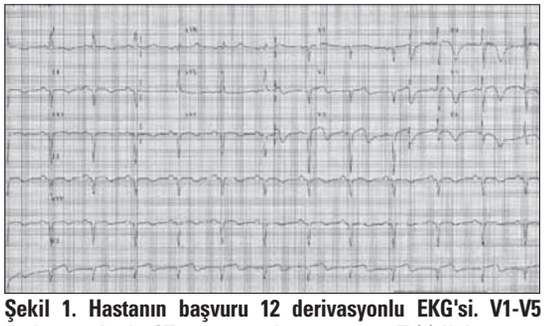
Sayın Editör,

Tako-tsubo sendromu tarif edildikten sonra bildirilen vaka sayılarında belirgin artış olmuştur. Çoğunlukla psikojenik stres sonrasında göğüs ağrısı, EKG değişikliği, koroner arter patolojisi olmaması ve tipik apikal balonlaşma görüntüsü ile tanı konur. Maksimum 6 hafta içerisinde normale döner. Ritm bozukluğu nadiren eşlik edebilir.

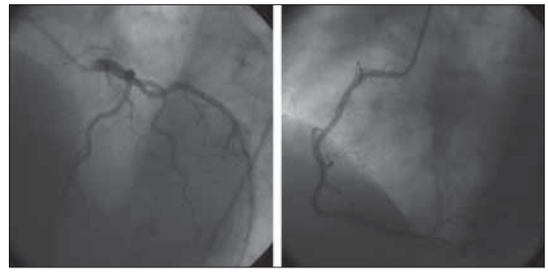
Atriyal fibrilasyon gelişen Tako-tsubo sendromlu bir hastamızı sunuyoruz. Altmış dokuz yaşında kadın hasta, bir hafta kadar önce hasarsız bir trafik kazası geçirmiş. Bu olaydan çok etkilenmiş. Olayın 3. gününde göğsünde baskı tarzında ağrı, soğuk terleme, nefes darlığı şikâyetleri başlamış ve birkaç gün sonra acil servise başvurmuş. İstirahat elektrokardiyografisinde (EKG) V2-V3 de ST elevasyonu, V3-V4-V5-V6'da derin T(-)'liği mevcuttu (Şekil 1). Subakut anteroseptal miyokard infarktüsü ve kompanze kalp yetersizliği bulgularıyla koroner yoğun bakıma alındı. Parenteral nitrat, diüretik, anjiyotensin dönüştürücü enzim inhibitörü, asetil salisilik asit, klopidogrel ve düşük molekül ağırlıklı heparin tedavisiyle stabilleşti. Troponin I hafif yüksek (0.86 ng/ml) ve kreatin-kinaz-MB normal sınırlarda idi, takiplerde yükselme olmadı. Ertesi gün koroner anjiyografi yapıldı. Koroner anjiyografi normaldi (Şekil-2) ve ventrikülografide tipik sistolde apikal balonlaşma görüldü (Şekil 3, 4). Tako-tsubo sendromu tanısıyla tedavisi yeniden düzenlendi. Koroner anjiyografiden 12 saat sonra hastada hızlı ventrikül cevaplı atriyal fibrilasyon gelişti (Şekil 5), Kordaron infüzyonu ile sinüzal ritme döndü. Ekokardiyografik kontrollerinde sol ventrikül sistolik fonksiyonlarında belirgin düzelme tespit edildi. On gün sonra miyokardiyal nekroz bulgularını ekarte etmek için miyokard perfüzyon sintigrafisi yapıldı ve iskemi/infarkt saptanmadı. Hipertiroidi yönünden tiroid fonksiyon testleri istendi ve normal geldi. Bir ay sonra poliklinik kontrolünde, EKG ve ekokardiyografi tamamen normaldi.

Tako-tsubo sendromu ilk olarak 1991 yılında tarif edilmiştir (1). Diastolde ventrikülografi görüntüsü ahtapot yakalama kabına benzediğinden bu isim verilmiştir. Çoğunlukla postmenopozal kadın, psikojenik stres sonrasında aşırı sempatik aktivite ile koroner mikrovasküler vasospazmın sorumlu olduğu öne sürülmektedir (2). Bizim hastamız da 69 yaşında bayan hasta olup, hasarsız bir trafik kazası sonrasında bu klinik tablo gelişmişti. Ayrıca daha genç bayanlarda ve erkek hastalarda da bildirilmektedir.

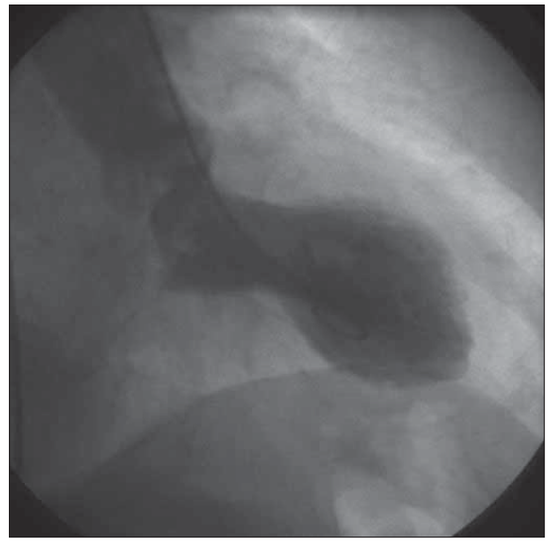
Literatüre bakıldığında çoğunlukla vaka raporları sunulmakta, cerrahi veya psikojenik stes, atriyoventrikül nod ablasyonu, cinsel ilişki ve hatta ilaç intoksikasyonu sonrasında bu sendrom rapor edilmektedir (3). Ayrıca kalp yetersizliği, kardiyojenik şok ile de komplike olabilmektedir. Ancak bu hastalığın akut döneminde veya seyri sürecinde başlıca ventriküler olmak



Şekil 1. Hastanın başvuru 12 derivasyonlu EKG'si. V1-V5 derivasyonlarda ST segment elevasyonu ve T (-) liği mevcut
EKG -elektrokardiyogram



Şekil 2. A) Hastanın sol sistemin koroner anjiyografisi normal görünümde B) Hastanın sağ koroner anjiyografisi



Şekil 3. Hastanın sol ventrikülografisi: Sistolde tipik apikal balonlaşma görüntüsü