



Official journal of
Cyprus Turkish Medical Association

CYPRUS JOURNAL OF MEDICAL SCIENCES

VOLUME 2 • ISSUE 2 • AUGUST 2017



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Publication Type
Local periodical

Printed Date
August 2017

Printed at
Share Ajans, Şehit
Fevait Ali Sok. Dük.
No: 4 C, Sönmezler Apt,
Göçmenköy, Nicosia,
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Books:

Chapter in a book: Sherry S. Detection of thrombi. In: Strauss HE, Pitt B, James AE, editors. *Cardiovascular Medicine*. St Louis: Mosby; 1974. p. 273-85.

Personal author(s): Cohn PF. *Silent myocardial ischemia and infarction*. 3rd ed. New York: Marcel Dekker; 1993.

Editor (s), compiler(s) as author: Norman IJ, Redfern SJ, editors. *Mental health care for elderly people*. New York: Churchill Livingstone; 1996.

Conference paper: Bengissson S, Sothemin BG. Enforcement of data protection, privacy and security in medical informatics. In: Lun KC, Degoulet P, Piemme TE, Rienhoff O, editors. *MEDINFO 92. Proceedings of the 7th World Congress on Medical Informatics*; 1992 Sept 6-10; Geneva, Switzerland. Amsterdam: North-Holland; 1992. P. 1561-5.

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access and utilization (dissertation). St. Louis (MO): Washington Univ. 1995.

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Pınar Gelener

Current Treatment of Diabetic Foot Infections and the Effect of Dermobor

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Cite this article as: Büke Ç. Current Treatment of Diabetic Foot Infections and the Effect of Dermobor. *Cyprus J Med Sci* 2017; 2: 29-34

One of the most serious chronic complications of diabetes is diabetic foot infections. Neuropathy, peripheral vascular disease and trauma are among the leading factors to development of diabetic foot ulcers and predispose to progress the diseases to diabetic foot infections. Delay of diagnosis and treatment and poor antibiotic treatment of diabetic foot infections can result in amputation. Diabetic foot ulcers and infections are the main causes of foot amputation around the world. The five-year survival rate of patients underwent foot amputation due to diabetic foot infections is low. With this review it's aimed to give information about the current epidemiology, risk factors, diagnosis and antimicrobial therapy of diabetic foot infections. In addition it's intended to give the results of Dermobor treatment in small number of cases with diabetic foot ulcers and infections.

Keywords: Diabetic foot ulcer, diabetic foot infection, antibiotic treatment, Dermobor

INTRODUCTION

Diabetes mellitus (DM) remains one of the major public health problems worldwide. The prevalence of both type 1 and 2 DM is increasing. Type 2 DM is responsible for almost 90% of diabetes patients. Obesity, inactivity, family history, age, gestation, high blood pressure, abnormal cholesterol and triglyceride levels and polycystic ovary syndrome play crucial role to get type 2 DM (1). The number of people living with diabetes is growing fast. In 2013 the number of patients with type 2 DM was 382 million. With this speed, it's estimated that, there will be 592 million people by 2035 (2). Turkey has the highest rate of prevalence among the European countries with 13.7% although the global prevalence is 8.5% (3). Diabetes is considered one of the devastating diseases with high mortality rate. In 2015, of the 56.4 million deaths worldwide, 1.6 million were due to DM. It remains the 7th leading cause of death. Diabetes is also very costly both for the individual and for the health systems of the countries. In 2013, the cost for diabetes and related diseases reached \$ 548 billion all over the world (4).

Diabetic foot infections (DFIs) are one of the most serious chronic complications of DM. Diabetic foot ulcers complicate the disease. They occur more than 15% of diabetic patients during their lifetime (5, 6). Treatment of diabetic foot ulcers (DFUs) and subsequent infections is difficult. Multidisciplinary approach is needed for the management of DFUs and DFIs. The most dramatic end result of DFUs and DFIs are foot amputation. The number has been reaching one in every 30 second in the world. The annual number of patient, undergoing foot amputation is around 12,000 in Turkey, and mostly due to DFIs (7-9).

The aim of this review is to review the current empiric and definitive antimicrobial therapy and to discuss the effectiveness of Dermobor gel (Genbor Biyosidal Yaşam Ürünleri San.Tic.Ltd.Şti, İstanbul, Turkey) in the treatment of DFUs and DFIs. It's believed that this review may be useful to primary care physicians, infectious diseases physicians, vascular surgeons, orthopedics, nurses and podiatrists.

RISK FACTORS AND PATHOGENESIS OF DIABETIC FOOT INFECTIONS

Most diabetic foot infections begin with a wound and once an infection occurs, the risk of amputation increases significantly. The prevalence of DFUs are around 4-10% depending on age and duration of DM. The lifetime prevalence reaches 15%. Most of the DFUs (60-80%) heal, while 10-15% of them remain unhealed. The healing of DFUs also depend on the characteristics of the wounds. For example neuropathic wounds are more likely to heal over a period of 4-5 months while

neuro-ischemic ulcers take longer period and generally result in amputation. Indeed 10-30% of patients with DFUs progress to amputation within a period of 6-18 months after the first evaluation. Among amputated patients five years mortality is around 50-60% (10, 11).

Multiple risk factors can play a role in the occurrence of DFUs such as; neuropathy, peripheral vascular disease, traumas, poor glycemic control and cigarette smoking. Among these neuropathy and peripheral vascular diseases are the most important factors. Sensory neuropathy can allow diminished perception of pain, pressure and heat, thus patients cannot distinguish well an injury or temperature to their feet. Motor neuropathy can cause foot deformities by muscle weakness, atrophy and paresis, which it becomes open pressure-induced soft tissue damage. In addition, autonomic neuropathy causes dry cracked skin by diminishing sweat secretion, resulting in a disruption of skin integrity. The deficiency of blood flow due to peripheral arterial diseases can results in the development of the wound and gangrene. In this setting the entry of microorganisms into the deep skin structures and the development of microbial infection become easy (12, 13).

CLASSIFICATION OF DIABETIC FOOT ULCERS

Diabetic foot ulcers generally classified as neuropathic, ischemic or both. Neuropathic DFUs is characterized with the presence of peripheral neuropathy without ischemia while ischemic DFUs is defined with the existence of symptoms related to peripheral artery disease with no peripheral neuropathy. In neuro-ischemic DFUs neuropathy and ischemia coexist (14). In describing the extent and the severity of lesions classification of DFUs is important. There are various classification schemes including; Wagner-Meggitt, PEDIS classification, Kings College Hospital classifications, and University of Texas classification. The Wagner-Meggitt and University of Texas are the most well accepted systems (Table I). The most common possible causative mi-

croorganisms responsible from DFIs are; *Staphylococcus aureus*, *Staphylococcus agalactiae*, coagulase negative staphylococcus (CNS), *Streptococcus pyogenes*, *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus spp.*, *Enterobacter spp.*, *Enterococcus spp.*, *Pseudomonas aeruginosa* and *Acinetobacter baumannii*.

CLINICAL SIGNS OF DIABETIC FOOT INFECTIONS

Diabetic foot infections are often accompanied by the cardinal manifestations of inflammation such as; erythema, warmth, swelling, tenderness and presence of pus in an ulcer or sinus tract. But, these signs may not be evident in all cases, especially in the presence of severe ischemia. Patients with sensory neuropathy may have diminished sensation in the involved area and may not complain of tenderness, even in the setting of infection. In such patients, infection may progress to involve deeper tissues (15). Presence of gangrene, severe ischemia, or tissue necrosis may remind the existence of a limb threatening infection. Systemic signs such as fever, chills, hypotension, and tachycardia may accompany local signs of infection and indicate an increased severity of infection, like sepsis and septic shock. Osteomyelitis can occur in the setting of a diabetic foot wound with or without evidence of local soft tissue infection. It should be diagnosed and aggressively treated as soon as possible.

DIAGNOSIS OF DIABETIC FOOT INFECTIONS

The diagnosis of DFIs depends on the presence of two or more cardinal manifestations of inflammation such as; erythema, warmth, tenderness, swelling and induration or the presence of pus (15). For definitive diagnosis the growth of the microorganism in culture DFIs is essential. In the absence of clinical signs and symptoms the growth of the microorganism should be assessed with caution. This situation is generally due to sample taken by superficial swabs. This method is not reliable for predicting the definitive pathogens. For the accurate diagnosis samples for culture should be aspirated from an abscess or curettage from the ulcer base following superficial debride-

TABLE I. Wagner-Meggitt and University of Texas Classifications of DFUs and causative microorganisms for diabetic foot infections

Grade	Wagner-Meggitt	University of Texas	Causative microorganisms
0	Healed or pre-ulcerative wound, pain on the foot only	Superficial ulcer, healed or pre- or post-ulcerative wound ^{A-D}	<i>S. aureus</i> <i>S. agalactiae</i> CNS <i>S. pyogenes</i>
1	Superficial ulcer, without reaching to the deeper layers	Full-thickness ulcer not involving tendon, capsule, or bone and without abscess formation ^{A-D}	<i>S. aureus</i> <i>S. agalactiae</i> CNS <i>S. pyogenes</i>
2	Deeper ulcer and penetrating tendon, bone or joint capsule	Tendon or capsular involvement without bone palpable ^{A-D}	+ Generally polymicrobial Enterobacteriaceae <i>Enterococcus spp.</i> <i>Pseudomonas aeruginosa</i> Anaerobes
3	Deep ulcer with bone, tendon involvement and there is abscess formation	Abscess formation and bone involvement ^{A-D}	+ Anaerobic <i>Streptococci</i> <i>Bacteroides spp.</i> <i>Clostridium spp.</i>
4	Gangrene on the part of the foot		
5	Gangrene on the whole foot		

A: not infected nor ischemic; B: infected; C: ischemic; D: ischemic and necrotic; CNS: Coagulase negative staphylococcus

ment of necrotic tissue (15, 16). Definitive diagnosis of osteomyelitis generally depends on isolation of bacteria from a sterilely obtained bone biopsy sample with histologic evidence. But bone biopsy is not always routinely available or practical. In such instances, the presumptive diagnosis is based on clinical and radiological assessment. The following factors increase the likelihood of osteomyelitis; grossly visible bone or ability to probe to bone, ulcer size larger than 2 cm², ulcer duration longer than one to two weeks, and erythrocyte sedimentation rate (ESR) >70 mm/h. On the presence of one or more findings the suitable changes in conventional radiograph can be helpful in making the diagnosis of osteomyelitis. Magnetic resonance imaging (MRI) is highly sensitive and specific for the diagnosis of osteomyelitis. If bone is grossly visible, radiographic examination is not necessary (17).

ANTIMICROBIAL TREATMENT OF DIABETIC FOOT INFECTIONS

For the success of the treatment of DFIs, multidisciplinary approach plays a key role. Management of DFIs requires attentive wound care, glycemic control, good nutrition, supply of fluid and electrolyte balance and appropriate antimicrobial therapy (18, 19). Patients with ulcerations that are not infected should not receive antibiotic therapy. In this situation local wound care and reducing the pressure on the foot is adequate. The selection of empiric antibiotic therapy should be considered based on the severity of infection and the likelihood of involvement of resistant organisms. If it is needed empiric therapy can be changed to definitive antibiotic treatment depending on culture and susceptibility results.

EMPIRIC ANTIBIOTIC THERAPY OF DIABETIC FOOT INFECTIONS

Clinical signs, epidemiological data and antimicrobial susceptibility results should be taken into the consideration for the choice of antibiotics in the empirical treatment of DFIs. Mild diabetic foot infections, manifesting with cellulitis or erythema extends ≤ 2 cm around the ulcer and without systemic signs of infection, can be treated as in outpatient. Oral single antibiotic therapy is convenient for mild DFIs. If there is no history of antibiotic use in the last one month empiric therapy should cover the activity against staphylococci and streptococci. In patients with previous hospitalization and prior antibiotic use, methicillin-resistant *Staphylococcus aureus* (MRSA) should be taken into the consideration. In patients with moderate DFIs, in which cellulitis or erythema extends >2 cm around the ulcer and infection with abscess, involving deep tissue such as muscle, tendon, joint and bone but without systemic signs of infection, antibiotics should include activity against staphylococci (including MRSA if risk factors are present), streptococci, aerobic gram-negative bacilli and anaerobes. In patients with deep ulcer, involving only fascia, antibiotics can be administered by oral route while patients presenting with extensive infections that involve deep tissues like joint and bone, should receive intravenous treatment and combination therapy should be given as point out in severe infections. In severe, limb-threatening diabetic foot infections and those that are associated with systemic toxicity combined broad-spectrum parenteral antibiotic therapy should be given. Surgical debridement is also necessary in most of these cases. Streptococci, MRSA, aerobic gram-negative bacilli such as; *E. coli*, *K. pneumonia*, *P.*

TABLE 2. Empiric antibiotic choice for diabetic foot infections

Severity	Choice of antibiotics	Dosages
Mild	Amoxicillin-clavulanate	875/125 mg twice a day PO or
	Co-trimoxazole	160/800 mg twice a day PO or
	Clindamycin	600 mg three times a day PO or
	Doxycycline	100 mg twice a day PO
	Fucidic acid*	500 mg three times a day PO or
	Linezolid*	600 mg twice a day PO
Moderate	Co-trimoxazole +	160/800 mg twice a day PO +
	Amoxicillin-clavulanate	875/125 mg twice a day PO
	or	or
	Clindamycin +	450 mg every 8 h PO +
	- Ciprofloxacin	- 750 mg twice a day PO or
	- Levofloxacin	- 750 mg once a day PO or
Severe	- Moxifloxacin	- 400 mg once a day
	Fucidic acid*	500 mg three times a day PO or
	Linezolid*	600 mg twice a day PO
	- Ampicillin-sulbactam	- 3 g every 6 h IV or
	- Piperacillin-tazobactam	- 4.5 g every 6-8 h IV or
	- Imipenem-cilastatin	- 500 mg every 6 h IV or
- Meropenem	- 1 g every 8 hours IV or	
- Ertapenem	- 1 g every 24 hours IV or	
- Moxifloxacin	- 400 mg every 24 h IV	
+	+	
- Vancomycin	- 1g every 12 h IV or	
- Linezolid	- 600 mg every 12 h IV or	
- Daptomycin	- 4-6 mg/kg every 24 h IV	

* In case of MRSA infection; PO: Peroral; IV: Intravenous

aeruginosa, and anaerobes should be covered by empiric antibiotic therapy (15, 20, 21). In patients with life threatened DFIs, long term chronic wound, prior antibiotic use, and exudative wounds, *P. aeruginosa* should be considered and covered in empiric antibiotic therapy (22). The choice of empiric antibiotic therapy is summarized in Table 2.

DURATION OF ANTIMICROBIAL THERAPY

Patients with mild infection oral antibiotic therapy should be given for about one to two weeks. Antibiotics do not need to be given until wound closure. Patients with moderate or severe infection, requiring surgical debridement, intravenous antibiotic therapy is usually adequate for two to four weeks without osteomyelitis. If there is a good response to parenteral therapy, oral agents can be used to complete the course of treatment. In patients with osteomyelitis, surgical resection is generally beneficial. In some studies it is demonstrated that antibiotic therapy for longer period without resection succeed the healing about 60 to 90%, which is comparable to those reported with surgery. The optimal duration is uncertain. But four to six weeks is an appropriate course if there is residual infected bone following debridement of necrotic bone. However, if necrotic bone remains, clinical cure may require several months with antibiotic therapy (15, 20).



FIGURE 1. a-d. The complete closure of DFIs in a 57 years old male patients with Dermobor gel within 50th days. (a) before Dermobor treatment, (b) 16th days of Dermobor treatment, (c) 38th days of Dermobor treatment, (d) 50th days of Dermobor treatment

The treatment of DFIs with local antimicrobial agents depends on several factors. General health of the patient, the process of tissue repair, and description and classification of the wound should be considered when deciding. Generally both local and systemic antimicrobials are using together in the treatment of patients with DFIs. Dermobor gel is licenced as a local treatment agents for DFIs in 2014. It contains 0.2% chlorhexidine digluconate and 3% sodium pentaborate pentahidrate (NaB), Chlorhexidine digluconate 0.2% has strong antibacterial and antiviral effect. This product has not only antimicrobial properties but also has wound closure effect with NaB.

We used Dermobor gel (Genbor Biyosidal Yaşam Ürünleri San. Tic.Ltd.Şti, İstanbul, Turkey) in ten patients with DFIs. Seven of them were male and the mean ages of patients were 64.12 ± 12.16 . The duration of diabetes mellitus was 15 years. In four patients the causative microorganisms were grown (*Staphylococcus aureus* in two patients, *Escherichia coli* and *Klebsiella pneumonia* in one patient respectively) from deep of ulcers taken by sterile biopsy techniques. In six patients the wound area was 10-19 cm² while in four it was more than 20 cm². Most of the patients (n: 6) were moderate and severe diabetic foot infections. Patients with severe diabetic foot infections (n: 2) antibiotics were given parenteral route. Patients with moderate diabetic foot infec-



FIGURE 2. a-d. The formation of granulation tissue $\geq 75\%$ of DFIs in a 72 years old female patient with Dermobor gel within 40th days. (a) before Dermobor treatment, (b) 10th days of Dermobor treatment, (c) 16th days of Dermobor treatment, (d) 40th days of Dermobor treatment

tions and wound culture results positives were used oral antibiotics also. All of the patients received Dermobor gel two times a day. Dermobor gel pomaded around and into the wounds' areas. Granulation tissue formation $> 75\%$, were seen in six patients in 4-5 weeks, wound closure has occurred in two patients in 6-7 weeks. The treatment has been continuing in remaining four patients.

In the first picture complete closure in the DFI was seen at the end of 50th days of Dermobor gel, twice a day, in a 57 years old male patient with DFI. In the second picture 75% granulation tissue formation was occurred at the 16th days of Der-

mobor gel treatment in a 72 years old female patient with DFI (Figure 1 and Figure 2).

CONCLUSION

Diabetic foot ulcers and infections are the one of the most hopeless chronic complications for diabetic patients since the healing of the wounds and infections generally take longtime. On the other hand sometimes the efforts of treatment cannot be resulted in success and can progress to the need of amputation. But currently it is believed that most of the DFUs and DFIs can be managed and the foot amputation can be prevented with careful patient management. According to the

results of the small number of cases with Dermobor in DFIs, it's seen that Dermobor is seen one of the hopeful choice in DFIs. It acts as both antibacterial and formation of granulation tissue. But for the assessment of the effect of Dermobor, multi-centric studies are needed.

Peer-review: Externally peer-reviewed.

Conflict of Interest: No conflict of interest was declared by the author.

Financial Disclosure: The author declared that this study has received no financial support.

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A Case of Left Main Coronary Thrombosis Treated Using Tirofiban

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Cite this article as: Küçükseymen S, Erkal Z, Bayar N, Arslan Ş. A Case of Left Main Coronary Thrombosis Treated Using Tirofiban. *Cyprus J Med Sci* 2017; 2: 35-7

The presence of a thrombus in the left main coronary artery is a rare but serious situation. Although there are no clear guidelines for such cases, a coronary artery bypass grafting (CABG) surgery is generally performed. Also, in the literature, there are limited reports on cases treated successfully through percutaneous procedures or medical therapy. In this article, we report the case of a patient with a thrombus in his left main coronary artery, a completely obstructed right coronary artery, and chronic renal failure. Because an immediate CABG was highly risky, the patient was given a tirofiban infusion for 48 hours. After the tirofiban infusion, the patient's thrombus had shrunk and his clinical situation had stabilized, paving the way for a successful surgical intervention.

Keywords: Coronary, thrombosis, tirofiban

INTRODUCTION

The presence of a thrombus in the left main coronary artery (LMCA) is a rare but serious situation. Although the incidence of a thrombus in LMCA has been reported to be 0.8%-1.7% and probably higher owing to undiagnosed sudden cardiac deaths (1). In this report, we present the case of a 57-year-old patient diagnosed with non-ST segment elevation myocardial infarction, who was found to have a thrombus in his LMCA and was treated with tirofiban infusion.

CASE PRESENTATION

A 57-year-old male patient presented at our Emergency Department with an intermittent constrictive chest pain. On obtaining his history, he was found to have chronic kidney failure and to be on hemodialysis for 7 years. On arrival, the electrocardiogram (ECG) showed extensive ST depressions and elevation on aVR (Figure 1). Troponin I level of 2.7 ng/ml and transferred to the intensive care unit. On transthoracic echocardiography, the lateral and posterior walls were hypokinetic and ejection fraction was 40% and serious mitral insufficiency was observed. Two hours after admission, the patient experienced chest pain again. ECG showed enlarged QRS complexes and increased ST elevation on aVR (Figure 2), then, underwent to the catheterization laboratory and showed the total obstruction of proximal right coronary artery (RCA). Thrombus formation was also observed in LMCA, left anterior descending artery (LAD), and proximal circumflex artery (Figure 3). Stenosis of over 80% in coronary angiography, which is considered to be very serious, was observed. An urgent coronary artery bypass grafting (CABG) was planned, but the Cardiac Surgery Department said that owing to the high risk, surgery could not be performed and suggested that it be postponed until the clinical condition improved. Upon this, tirofiban infusion was started for 48 hours accompanied by continuous hemodialysis so that it could pass through the dialysis. At the second hour during infusion, ECG showed some regressions; the QRS complexes enlargement had diminished (Figure 4). The patient was hemodynamically stable, and the chest pain had ameliorated. On the third day, control CAG was performed, and the thrombus in LMCA had regressed. Only in the ostial segment of the circumflex artery, the thrombus could still be observed, with a TIMI 3 flow in LAD (Figure 5). The patient subsequently underwent surgery with triple CABG. The patient was discharged on the 15th day after admission with no further symptoms or complications. The patient's consent was assessed.

DISCUSSION

The presence of a thrombus in LMCA is a serious life-threatening situation. The clinical presentation and prognosis depend on the presence of collateral flow from RCA and on the amount of distal flow confinement that the thrombus creates. Thrombus development can be secondary to the rupture of an atherosclerosis plaque, but it may also occur in a normal coronary artery. In our case, we believe that the thrombus development was secondary to an atherosclerosis plaque rupture or to a protein C deficiency owing to chronic kidney disease.

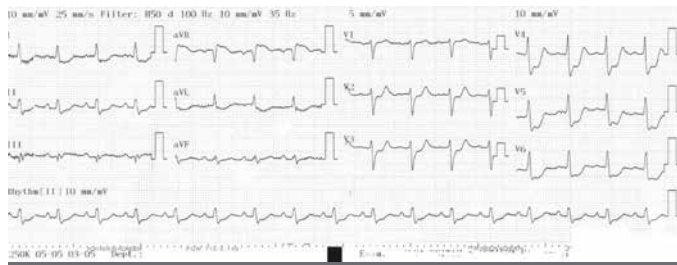


FIGURE 1. The patient’s ECG showing extensive ST segment depression and ST segment elevation on aVR on admission



FIGURE 2. Two hours after the hospitalization, ECG showing enlarged QRS complexes and increased ST segment elevation on aVR



FIGURE 3. Left coronary angiogram demonstrating coronary thrombus in LMCA, proximal LAD, and proximal circumflex artery

There is no consensus as to how patients with a thrombus in LMCA should be treated. Most of the cases reported in the literature involved the use of CABG (2, 3). Nevertheless, there have been successful reports of treatment via thrombus aspiration (4). Few studies in the literature report patients with a thrombus in LMCA to have been treated medically. Gürkan et al. (5) first

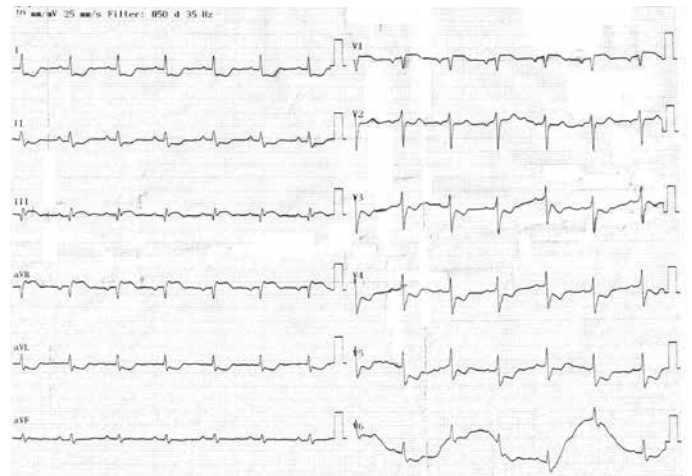


FIGURE 4. At the second hour of the tirofiban infusion therapy, the patient’s ECG findings showing some regressions; the QRS complex enlargement is diminished

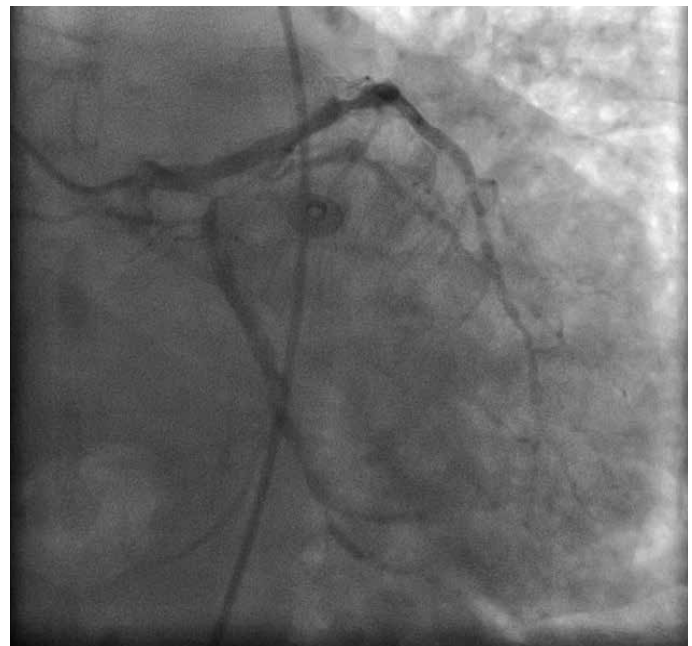


FIGURE 5. Control coronary angiography, demonstrating coronary thrombus only in the ostial segment of the circumflex artery

reported successful results in treating a patient with a thrombus in LMCA with a tissue plasminogen activator infusion. Ayari et al. have reported thrombus regression in treating a thrombus in LMCA in a patient with angio-Behçet disease via infusion of tirofiban for 48 hours (6). Sayin et al. have also reported regression of a thrombus in LMCA in patients with protein C and protein S deficiency using tirofiban (7). In another case of a thrombus in LMCA, the blood flow in the coronary artery was achieved through a “kissing balloon” technique, followed by the initiation of abciximab infusion, yielding successful results (8). In our case, the patient did not have ST elevation; therefore, thrombolytic therapy was not planned. Although our patient was hemodynamically stable, totally obstructed RCA and renal condition would have let to a high risk of operative mortality. Therefore, tirofiban infusion was initiated, which successfully shrank the thrombus and made an elective, but successful, operation possible.

CONCLUSION

In patients with a thrombus in LMCA, who despite being clinically stable, for some reason, cannot undergo urgent surgery, treatment with tirofiban can shrink the thrombus and help decrease the mortality risk in the surgery to follow.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Concept - S.K., Z.E.; Design - N.B., Ş.A.; Supervision - N.B., Ş.A.; Resource - N.B.; Materials - N.B.; Data Collection and/or Processing - S.K., Z.E.; Analysis and/or Interpretation - S.K., Z.E.; Literature Search - S.K., Z.E.; Writing - S.K., Z.E. N.B.; Critical Reviews - N.B., Ş.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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A Case of Thrombus Formation in a Patient with Preserved Left Ventricle Ejection Fraction and Development of Peripheral Embolization

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Cite this article as: Erkal Z, Bayar N, Arslan Ş. A case of thrombus formation in a patient with preserved left ventricle ejection fraction and development of peripheral embolization. *Cyprus J Med Sci* 2017; 2: 38-9

Echocardiography is a substantial imaging modality for evaluating intracardiac masses. Thrombi and primary or metastatic cardiac tumors should be considered in the differential diagnosis of left ventricle (LV) masses. Thrombus formation in the LV generally occurs because of diseases that cause LV systolic dysfunction. Some cases have been reported to be related to thrombus formation in patients with a normal LV ejection fraction. Physicians should consider that a thrombus might develop in patients with a normal ejection fraction.

Keywords: Thrombus, embolization, echocardiography

INTRODUCTION

Thrombus formation in the left ventricle (LV) generally occurs because of diseases that cause LV systolic dysfunction such as dilated cardiomyopathy, myocardial infarction, and aneurysm. Here we present the case of a patient with an LV thrombus and peripheral embolization and who had a normal LV systolic function.

CASE PRESENTATION

A 63-year-old male patient with severe pain in the left leg was admitted to the emergency department. He underwent embolectomy of the right femoral artery 2 weeks ago at another hospital. The patient's physical examination was quite normal, except for the absence of left popliteal and distal pulses. His blood pressure was 113/67 mmHg, with a pulse rate of 67 beats/min. Electrocardiogram revealed a normal sinus rhythm. The ejection fraction of LV was 65%, and a mobile, pedunculated heterogeneous mass originating from the interventricular septum and measuring 1.3x1.1 cm in diameter was observed on echocardiography (Figure 1). It was primarily considered to be a myxoma rather than a thrombus. Peripheral angiography was performed, and acute 100% obstruction was detected at the left femoral artery on an angiogram. Therefore, emergency left femoral embolectomy was performed by cardiovascular surgeons. Histopathology of the embolectomy specimen was relevant with a thrombus. Thus, echocardiography was repeated, and a 4-mm-sized residual mass was detected 3 days after the surgery (Figure 2). Factor V Leiden and prothrombin 20210 mutation tests were negative, and protein C and S and antithrombin 3 levels and eosinophil counts were all normal. Moreover, tests for Antinuclear anticore level and lupus anticoagulants were negative. The patient was anticoagulated with warfarin, with a target INR of 2-3. The patient's consent was obtained.

DISCUSSION

Echocardiography is a substantial imaging modality for evaluating intracardiac masses. Thrombi and primary or metastatic cardiac tumors should be considered in the differential diagnosis of LV masses (1-6). Myxoma was primarily considered to be the diagnosis because of a mobile, pedunculated mass in a normal functioning LV, although, with a low probability, our differential diagnosis included a thrombus (7).

Protein C and S deficiency, antiphospholipid syndrome, myeloproliferative disorders, idiopathic hypereosinophilic syndrome, pheochromocytoma, and Takatsubo cardiomyopathy were reported to be related with thrombus formation in patients with a normal LV ejection fraction (8-10).

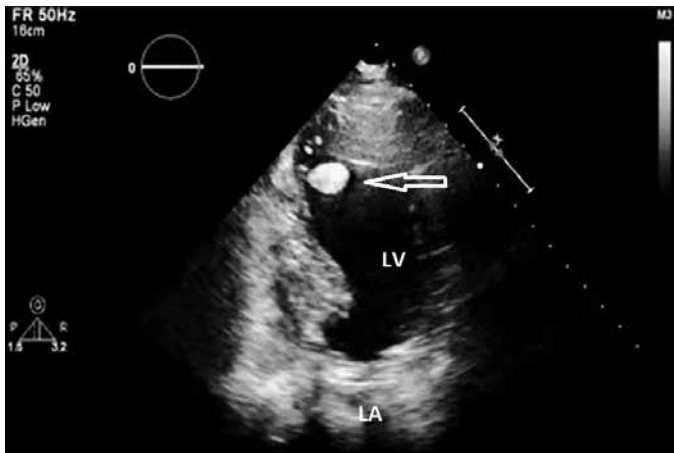


FIGURE 1. Thrombus image clinging to the left ventricle

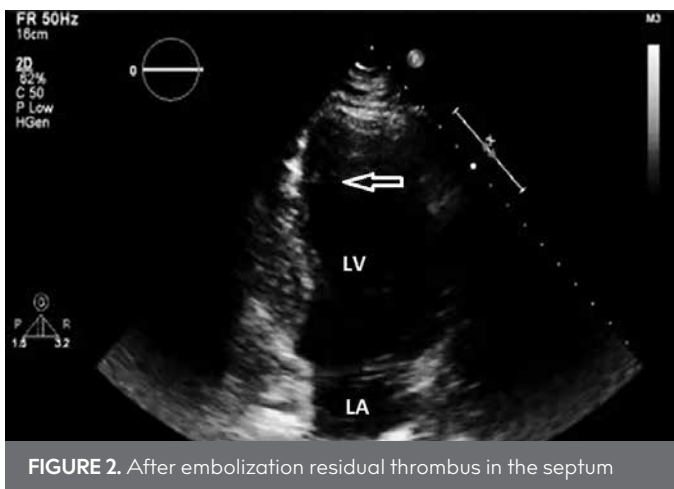


FIGURE 2. After embolization residual thrombus in the septum

Clinical and laboratory findings of our case were all normal. LV thrombus formation and peripheral embolization are extremely rare in patients with a normal ejection fraction and without any etiologic cause, as in our case (7).

Physicians should be careful regarding LV thrombus or mass formation in patients with a history of systemic embolus. Furthermore, it should be considered that a thrombus might develop in patients with a normal ejection fraction.

Informed Consent: Informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Concept - Z.E.; Design - Z.E., N.B.; Supervision - N.B., Ş.A.; Resource - N.B.; Materials - Z.E.; Data Collection and/or Processing - Z.E., Ş.A.; Analysis and/or Interpretation - Ş.A.; Literature Search - Z.E., N.B.; Writing - Z.E., N.B.; Critical Reviews - Ş.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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A Rare Co-Existence of Os Accessorium Supracalcaneum and Os Trigonum

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Cite this article as: Apaydin M, Sezgin G, Birlik B. A Rare Co-Existence of Os Accessorium Supracalcaneum and Os Trigonum. *Cyprus J Med Sci* 2017; 2: 40-1

Dear Editor,

A 28-year-old man presented to the orthopedics clinic with the complaints of a severe pain and swelling over his left ankle. He stated that the swelling had appeared a long time ago. The swelling was noted to be located on both sides of the Achilles tendon of the left foot and was very firm on palpation. He had difficulty in inversion and plantar flexion of left foot. X-ray and CT of the left foot were obtained (Figure 1, shows lateral X-ray of the left ankle, two bony masses separately indentified and located to the posterior of the left talus above the calcaneus. The details were seen more

clearly in reconstructed CT image; Figure 2, a: sagittal and b: coronal views).



FIGURE 1. X-ray of the left ankle showing two distinct bony masses, separately identified and located posterior to the talus

Accessory ossicles in the foot are in variable appearance and prevalence. Vesal called the first accessory ossicle as "os peroneum" in 1555. Since then 23 true accessory bones were reported. The symptoms of accessory ossicles depend on their location and associated pathologies such as degeneration, trauma and others. Clinical findings usually cause minor symptoms but basic knowledge of incidental variants is necessary to avoid misinterpretations such as fractures. The most commonly seen accessory bones in the foot are os trigonum, os peroneum and os naviculare. Os vesalinum, os intermetatarsale, os supratarsale, os supranaviculare, os calcaneus secundarium and os talotibiale are rarely seen forms of accessory bones (1). Os accessorium supracalcaneum (OAS) is the rarest form of accessory bone in the foot. At the beginning, there was a conflict about the existence of os supracalcaneum apart from os trigonum. But it was accepted as an accessory ossicle radiologically and surgically. The OAS is seen behind the os trigonum. This bony structure has clear margins with adjacent to the tendon of Achilles. The shape and location was different from the os trigonum (2). Also, we report that the coexistence of two bones. Os trigonum which persists separately in 7-14% of the population, has cartilaginous synchondrosis between the ossicle. The differential diagnosis includes lateral tubercle's (Shepherd's fracture) non-union fracture. Cartilage synchondrosis disorder makes os trigonum syndrome with recurrent microtrauma in between talar tubercle and os trigonum (3). OAS may cause the same symptoms because of its location. In our case, we detected cartilaginous synchondrosis between OAS and calcaneus as like in be-



FIGURE 2. On sagittal (a) and coronal (b) reformatted CT scan images showing two bony masses separately from the posterior portion of the talus, and appeared to articulate with the posterosuperior segment of the calcaneus. Bony bridges between these bony masses and the calcaneus are identified. The masses were separated from the tendon of Achilles

tween talus and os trigonum. In addition, os trigonum and OAS has cartilaginous synchondrosis (Figure 2).

The patient had pain with palpation in the talus area which was also investigated visual scale. He underwent physical therapy including ankle motion range and strengthening exercises as well as electrical nerve stimulation and cold therapy. The pa-

tient's pain was alleviated. Operation was planned in case of persistence of the pain and symptoms.

Ethics Committee Approval: N/A

Informed Consent: N/A

Peer-review: Externally peer-reviewed.

Author contributions: Concept - M.A., G.S.; Design - M.A., B.B.; Supervision - M.A.; Resource - M.A.; Materials - M.A., G.S.; Data Collection and/or Processing - M.A.; Analysis and/or Interpretation - M.A.; Literature Search - B.B., G.S.; Writing - M.A.; Critical Reviews - M.A., G.S., B.B. A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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Converting an Urgent Case into an Elective Procedure: Volvulus of Sigmoid Colon

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Cite this article as: Karaisli S, Bağ H, Cengiz F, Kar H, Genç H. Converting an Urgent Case into an Elective Procedure: Volvulus of Sigmoid Colon. *Cyprus J Med Sci* 2017; 2: 42-3

Dear Editor,

A 73-year-old male patient was admitted to the emergency room with abdominal distention and obstipation which had lasted for two days. On performing a physical examination, significant asymmetric abdominal distention was detected. There were no signs of peritonitis, and his digital rectal examination revealed an empty rectum. In his abdominal X-ray, a dilated colonic segment was observed, with a coffee bean sign (Figure 1). Sigmoid colon and mesentery torsion were detected on performing contrast-enhanced computed tomography (CT) (Figure 2). The patient underwent urgent colonoscopy. The sigmoid colon was decompressed after detorsion (Figure 3). On the 7th day after urgent admission, the patient underwent elective sigmoid colon resection and side-by-side anastomosis. The patient was discharged on the postoperative day 6. A pathologic examination revealed a benign disease. The patient had completed a 4-month follow-up period.

Sigmoid volvulus (SV) may cause ischemia, perforation, sepsis, and finally, mortality (1). SV is the third most common cause of colonic obstruction in adults (2). Middle-aged patients are more frequently affected in countries with a high incidence of SV, whereas the incidence increases at around 70 years of age in developed countries (3, 4). The main predisposing factors are a long sigmoid colon and prolonged mesocolon (3). In addition to advanced age, male sex, postoperative adhesions, pregnancy, psychiatric illness, and some medications can be cited as other risk factors (1-5).

Abdominal X-ray and CT are diagnostic imaging methods (3, 4). The first step is to attempt colonoscopic reduction. Urgent surgery is required in patients with unsuccessful colonoscopic reduction or

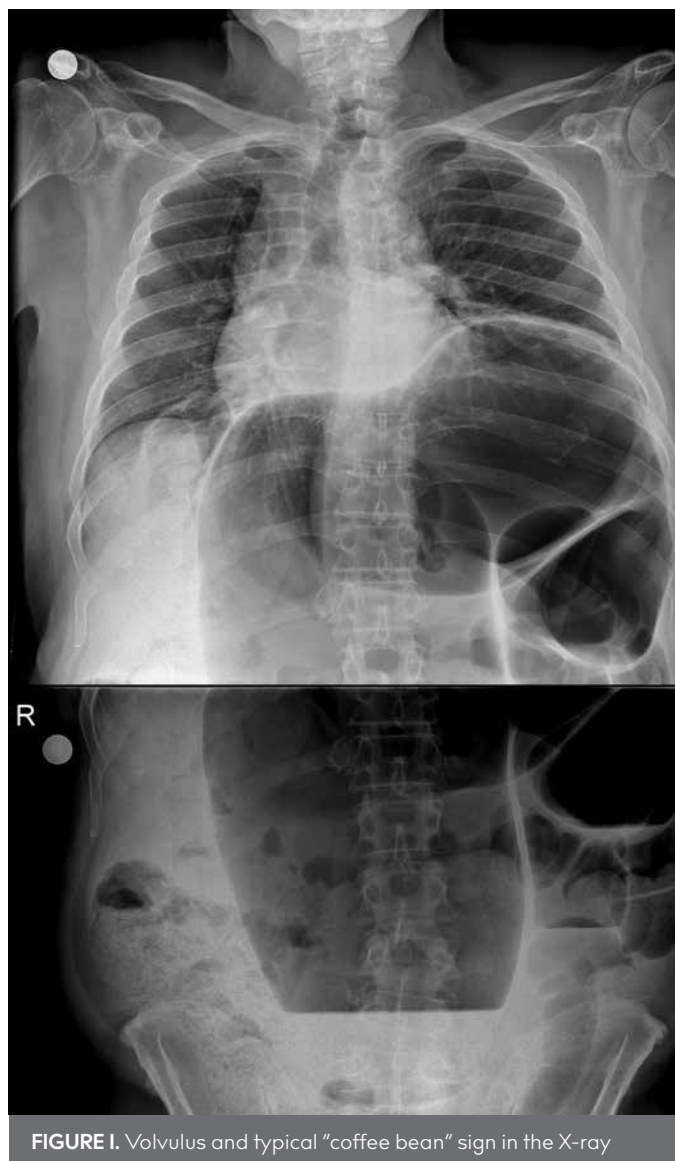


FIGURE 1. Volvulus and typical "coffee bean" sign in the X-ray

This study was presented as a poster at the 16th Turkish Colon and Rectal Surgery Congress, 16-20 May 2017, Antalya, Turkey

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Received: 22.05.2017
Accepted: 12.07.2017

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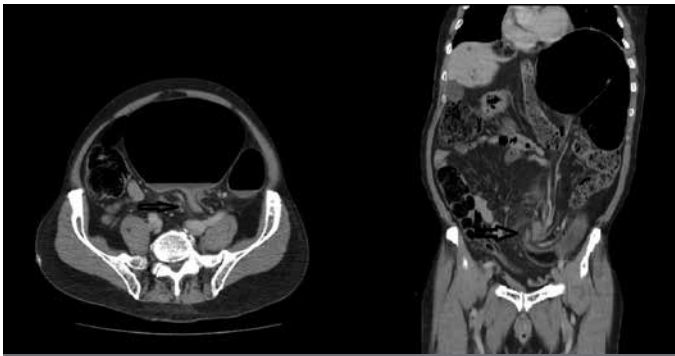


FIGURE 2. Twisted mesentery of the sigmoid colon (arrow)



FIGURE 3. Fecal movement to the distal segment after colonoscopic detorsion

peritonitis or perforation associated with colonoscopy (1, 3-5). The mortality rate in emergency operations is high due to multiple comorbidities of affected patients (1-5). The recommended method for patients with acute SV with peritonitis is Hartman colectomy following sigmoid resection (1, 3-5). However, in cases with successful colonoscopic reduction, even if in the first volvulus episode, elective colectomy should be planned after reduction in patients with hemodynamic stability and low risk of surgical mortality and morbidity. Grossman et al. (2) reported a 6% mortality rate with an elective operation after colonoscopic

decompression. They reported that the SV recurrence rate and mortality rate associated with recurrence were 23% and 20%, respectively, in the colonoscopic decompression group.

Colonoscopic reduction should be planned as soon as possible after a diagnosis is made. If reduction is achieved and the patient is hemodynamically stable, we believe that performing elective surgery will help eliminate the possibility of relapses of SV, reduce the mortality and morbidity rates of the urgent operation, and not required additional surgical intervention to close the colostomy that might open during an emergency operation.

Ethics Committee Approval: N/A.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Concept - S.K., F.C.; Design - S.K., H.B.; Supervision - H.B., H.G.; Resource - H.K., H.G.; Materials - S.K., F.C.; Data Collection and/or Processing - S.K., H.B.; Analysis and/or Interpretation - H.B., H.K., H.G.; Literature Search - S.K., F.C.; Writing - H.B., F.C.; Critical Reviews - H.K., H.G.

Acknowledgements: We thank all general surgery department staff for their cooperation and Dr. Fatma Tatar for his help on preparing this manuscript.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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Should Every Coronary Chronic Total Occlusion be Treated Invasively?

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Cite this article as: Karabulut A. Should every coronary chronic total occlusion be treated invasively? *Cyprus J Med Sci* 2017; 2: 44-5

Dear Editor,

A 48 year-old female patient presented with recurrent Canadian class 2 angina-equivalent chest pain. She had inferior myocardial infarction two years ago. In addition, she was under medication for hypertension. In the first presentation, patient was taking aspirin 100 mg and ramipril/hydrochlorothiazide 5/12.5 mg once a day as medical treatment. Electrocardiography showed pathological Q waves on inferior derivations, whereas, echocardiogram revealed a normokinetic left ventricular wall. Further coronary ischemia indicating tests including treadmill and scintigraphy were not performed. Coronary angiography showed total occlusion of proximal right coronary artery (RCA) once after sinus branch. However, there was thrombolysis in myocardial infarction (TIMI) 3 flow through the RCA, which was provided by proximally originating multiple collateral branches (Figure 1, 2). The occluded segment was short, and inattentive assessment could interpret RCA to have a normal flow without significant stenosis, hence, collaterals was coursing over the occluded segment in unison. Medical following was proposed for the patient. Patient was discharged with a medical recipe including aspirin 100 mg, ramipril/hydrochlorothiazide 5/12.5 mg, atorvastatin 20 mg, bisoprolol 5 mg and benipin 4 mg. Sixth month follow was uneventfull.

Coronary chronic total occlusion (CTO) lesions are defined as the coronary lesions with TIMI-0 flow, within the occluded segment along with angiographic or clinical evidence of occlusion duration >3months. They account for about one-third of the coronary lesions. The technical success for PCI of CTO was below the 50 % in the early period of percutaneous cor-

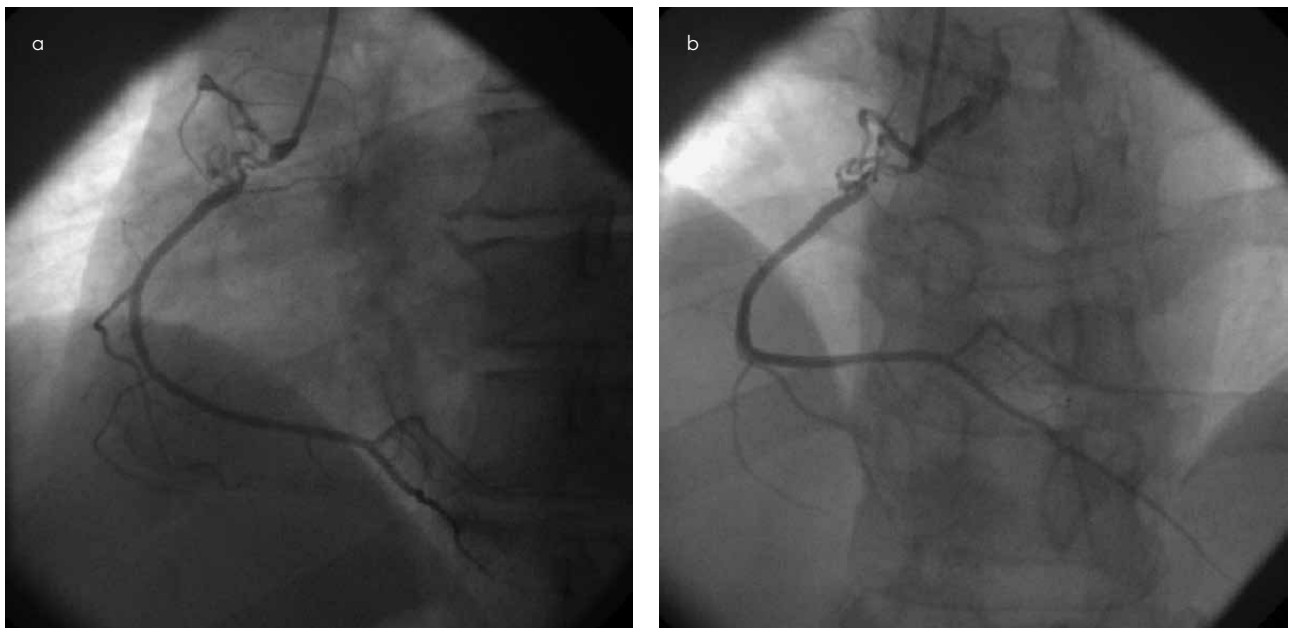


FIGURE 1. a, b. Left anterior oblique (a) and anterior cranial projection (b) showing the right coronary artery

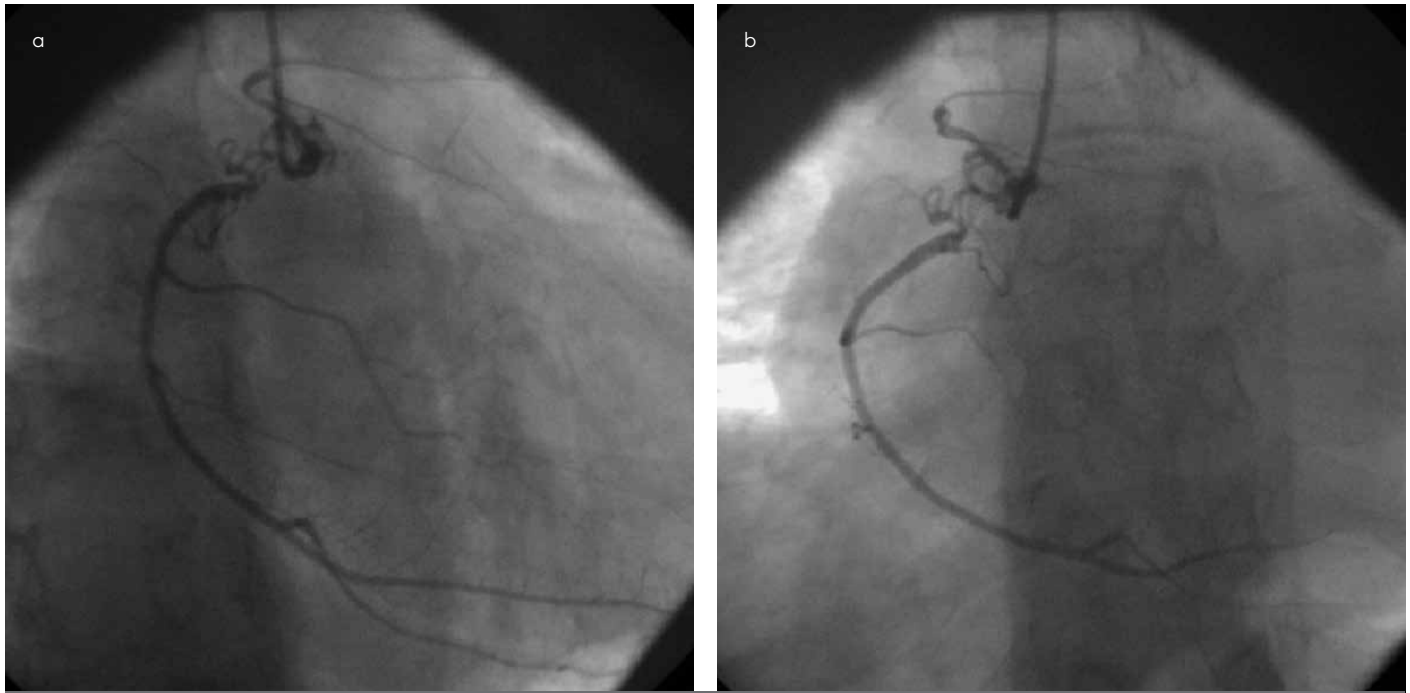


FIGURE 2. a, b. Right anterior oblique caudal (a) and anterior caudal (b) projection showing the right coronary artery

onary interventions' era and majority of the cases was treated either medically or surgically (3). However, due to advancement of guidewires, devices, and techniques in recent years, successful recanalization may now be achieved in as many as 80% of the CTO lesions. Parallel to technical improvements, the number of CTO procedures was increased markedly and many operators, who defend the open-artery hypothesis, encouraged to perform such procedures more commonly. On contrary, all the successful intervention does not lead to improvement in the clinical status of the patients. Thus, all the CTO cases should be evaluated in detail before such complex procedures.

Coronary territories of the occluded coronary vessels are usually supplied by collateral circulation. The protective effect of coronary collateral circulation was well established. It is known that well-developed collaterals diminish coronary ischemia and can limit the infarct size. Collateral circulation is also associated with decreased mortality and predicts better prognosis (4). Moreover, a well-developed Rentrop-3 collateral circulation has a similar prognosis as a revascularized patient. In our case, multiple bridge collaterals supplied the coronary artery with antegrade TIMI 3 flow without evidence of coronary ischemia. Although the left ventricular function was normal, such a well-developed collateral circulation would eliminate the necessity of a percutaneous intervention.

In conclusion, every coronary CTO should not be invasively treated, even in the presence of a normal ventricular function. A

well-developed coronary collateral circulation should consider the evaluation of coronary CTO therapy.

Ethics Committee Approval: Ethics committee approval was received for this study from the local ethics committee of the Acibadem Atakent Hospital.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Conflict of Interest: No conflict of interest was declared by the author.

Financial Disclosure: The author declared that this study has received no financial support.

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Turtle Headache: A Case Report and Approach To Hypnic Headaches

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Cite this article as: Gelener P. Turtle Headache: A Case Report and Approach To Hypnic Headaches. *Cyprus J Med Sci* 2017; 2: 46

Dear Editor,

The first turtle headache was reported in 1972 in a patient who had bilateral headache after awakening in the early morning. The headache developed while trying to go back to sleep and feeling discomfort in the daylight. The headache went worse if he pulls his head under his bed cover (1).

In the latest beta version of international headache classification hypnic headache (HH) syndrome, 'alarm clock headache' is regarded as a rare primary headache disorder (2). The attacks mostly occur at the same time at night. The etiopathogenesis of HH is poorly understood, yet the gray matter volume changes in the posterior hypothalamus, which is the biological clock, was detected in a study including HH patients (3).

Herein we report a case of 51 year-old man, who regularly awakened from sleep at the same time of the early morning by a diffuse headache which persisted for 15 to 40 minutes since one and a half year. He noticed that the headache continued only if he returns to sleep after he wakes up but it resolves if he gets up. In the last two months he had this headache everyday. He never reported any nausea, autonomic symptoms or restlessness. Cranial magnetic resonance imaging was normal. The patient was started on dual therapy including melatonin and caffeine. In the follow up visit after a month, he reported that the frequency and duration of the attacks decreased more than 50%.

Insistent circadian rhythm of these headache attacks points hypothalamic involvement and another clinical feature of HH is its therapeutic response to caffeine and melatonin (4, 5).

Distinction from other primary headaches especially from cluster headache (CH) is essential, which is an autonomic cephalgia occurring as paroxysmal excruciatingly severe unilateral pain grouped in cluster periods. The pain intensity in CH is estimated to be 100 to 1000 times worse. Another important point for HH is the exclusion of other causes of headache causing waking from sleep including sleep apnea, nocturnal hypertension, hypoglycaemia, medication overuse and intracranial disorders. Though the diagnosis of sleep apnoea does not exclude accompanying HH syndrome (1).

Informed Consent: Verbal informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

Conflict of Interest: No conflict of interest was declared by the author.

Financial Disclosure: The author declared that this study has received no financial support.

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