DİYABETİK AYAK İNFEKSİYONU OLGU SUNUMU

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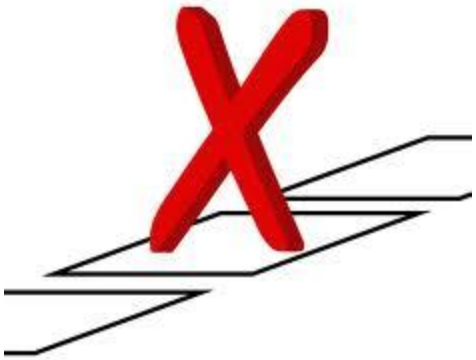
Dr.Lütfi Kırdar Kartal Eğitim ve Araştırma Hastanesi
İnfeksiyon Hastalıkları ve Klinik Mikrobiyoloji Kliniği

II. ULUSAL DİYABETİK AYAK İNFEKSİYONLARI SİMPOZYUMU
24-26 Mayıs 2012


YANIK VE YARA TEDAVİ MERKEZİ



DİYABETİK AYAK KONSEYİ

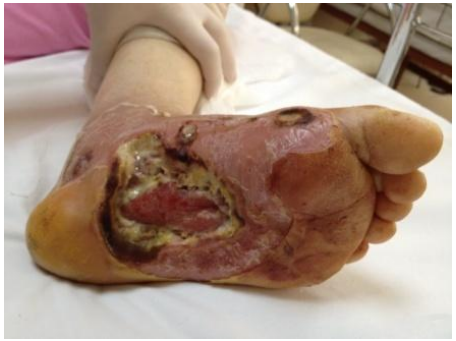
OLGU- 1

- NB, 53 y, kadın, 160 cm, 73 kg, ev hanımı
- Yatış tarihi: 27.3.2012
- **Yakınma**: Sol ayak tabanında açık yara ve sağ kalçada ağrı, akıntı

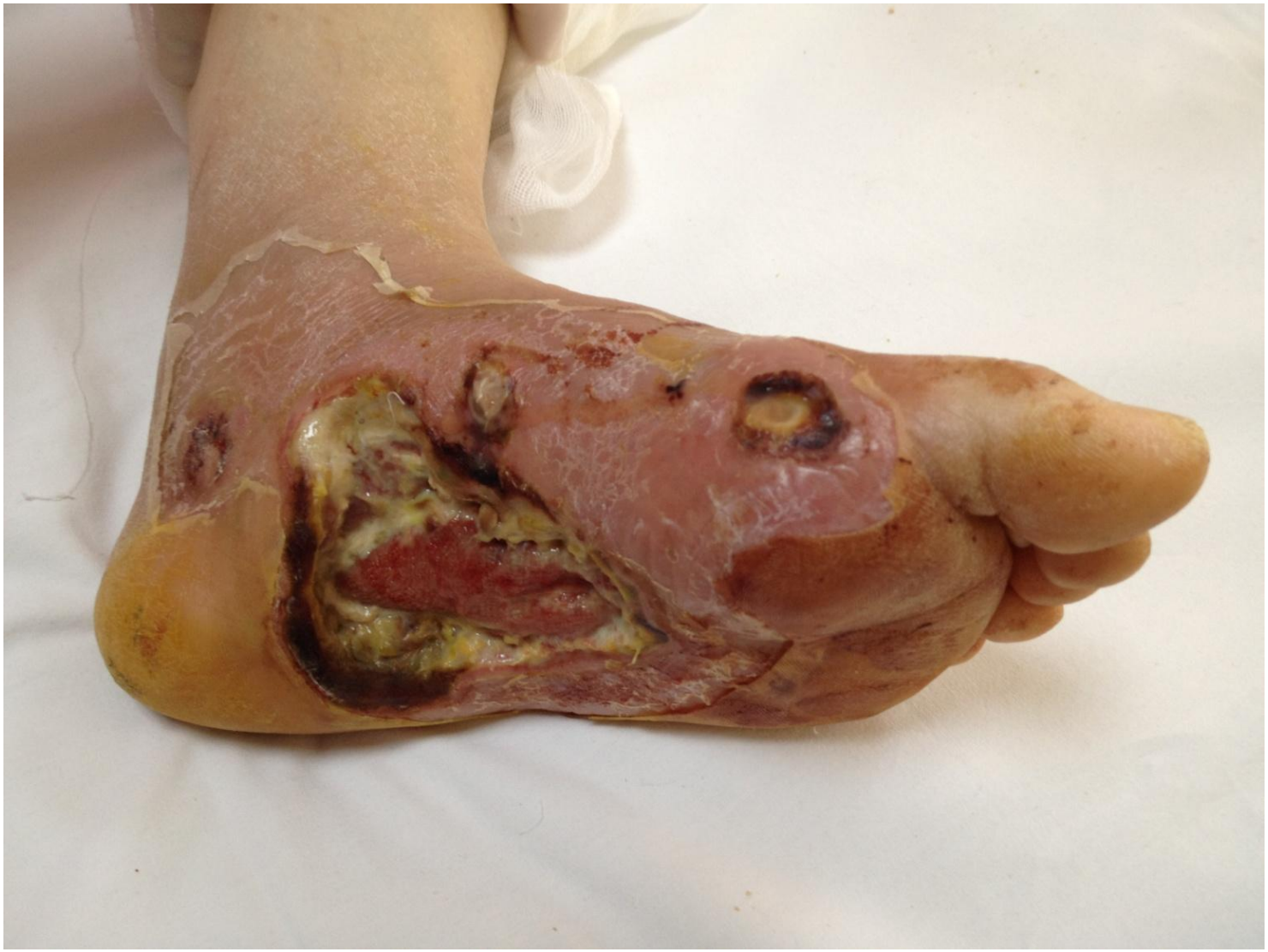
- **Öykü**:
- Sol ayak tabanında **45 gün önce ülser** nedeniyle yara-yanık merkezinde ayaktan takipli
- Bir ay öncesinde üriner enfeksiyon (dış merkezde) 12 günlük seftriakson (2x1 g) tedavisi.
- O dönemde IM enjeksiyon sonrası sağ tarafta gluteal abse → drenaj ve kültür → *E.coli* üremiş
- Diyabetik ayak infeksiyonu ve sağ gluteal abse
- **Özgeçmiş:** Tip 2 DM (9 yıldır)
  TAH-BSO (7 yıl önce)
- **Kullandığı İlaçlar**: İnsülin : 3x13 ü Regüler,
  1x 20 ü NPH
  Siprofloksasin (2x750 mg po) 15 gün

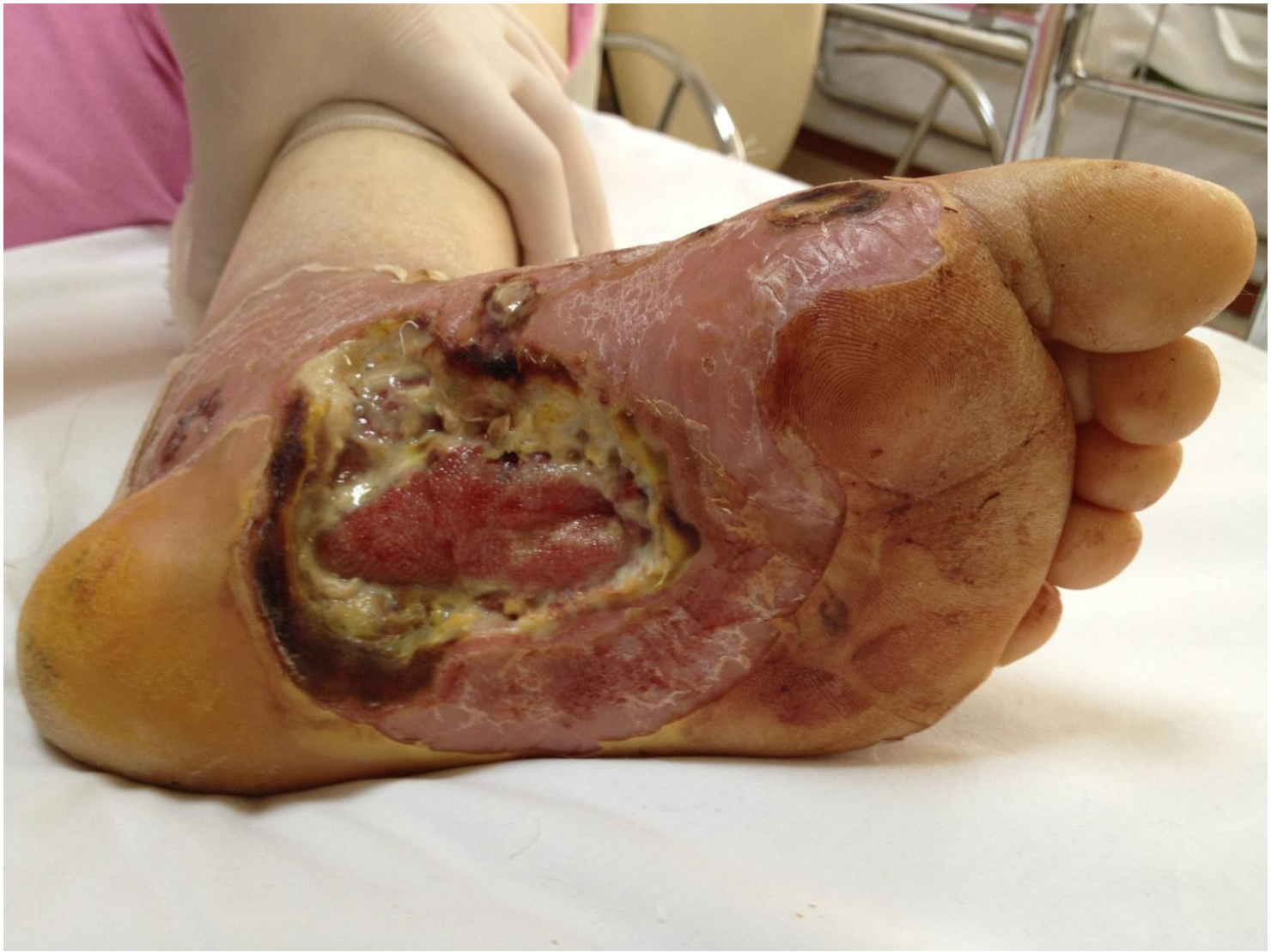
FİZİK İNCELEME

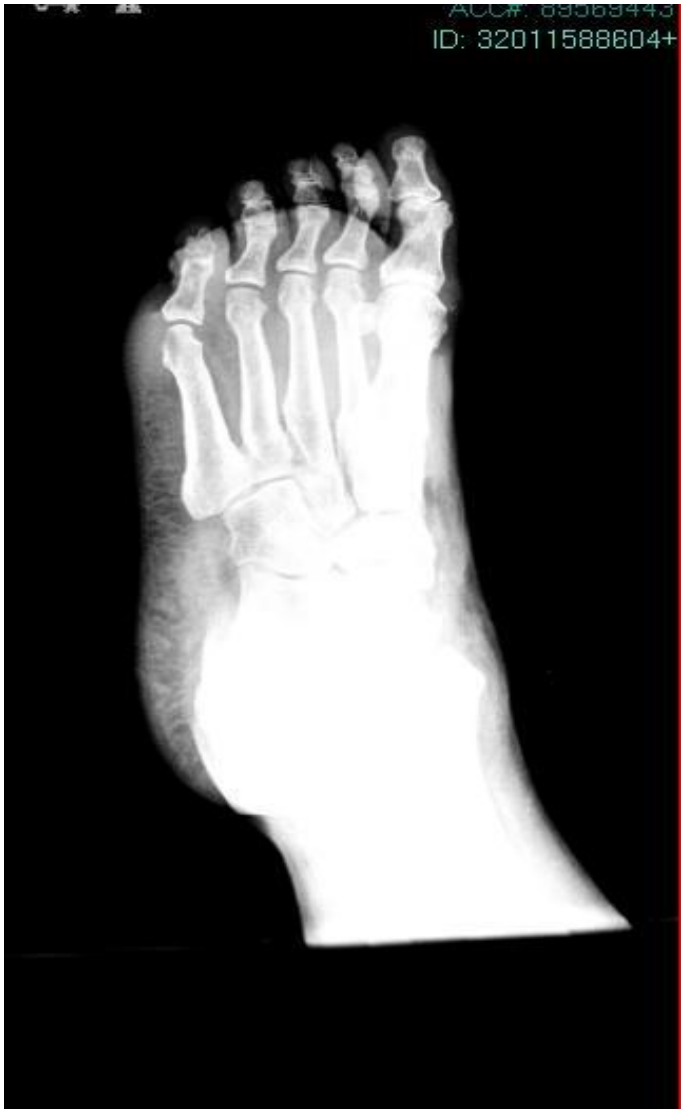
- A: 36.3°C Nb: 90/dk TA:100/60 mmHg SS: 16/dk
- Sol ayak tabanında pürülan akıntılı yaklaşık **8x8 cm** boyutlarında, **3 cm** derinliğinde **kemiğin ekspoze olduğu** ülsere nekrotik açık yara
- Periferik nabızlar palpabl.
- Ayakta duyu kaybı (+)
- Sağ gluteal bölgede drene olan abse.
- Sistem muayeneleri doğal.
- PTB: (+)
-



Sol ayak – Debridman sonrası



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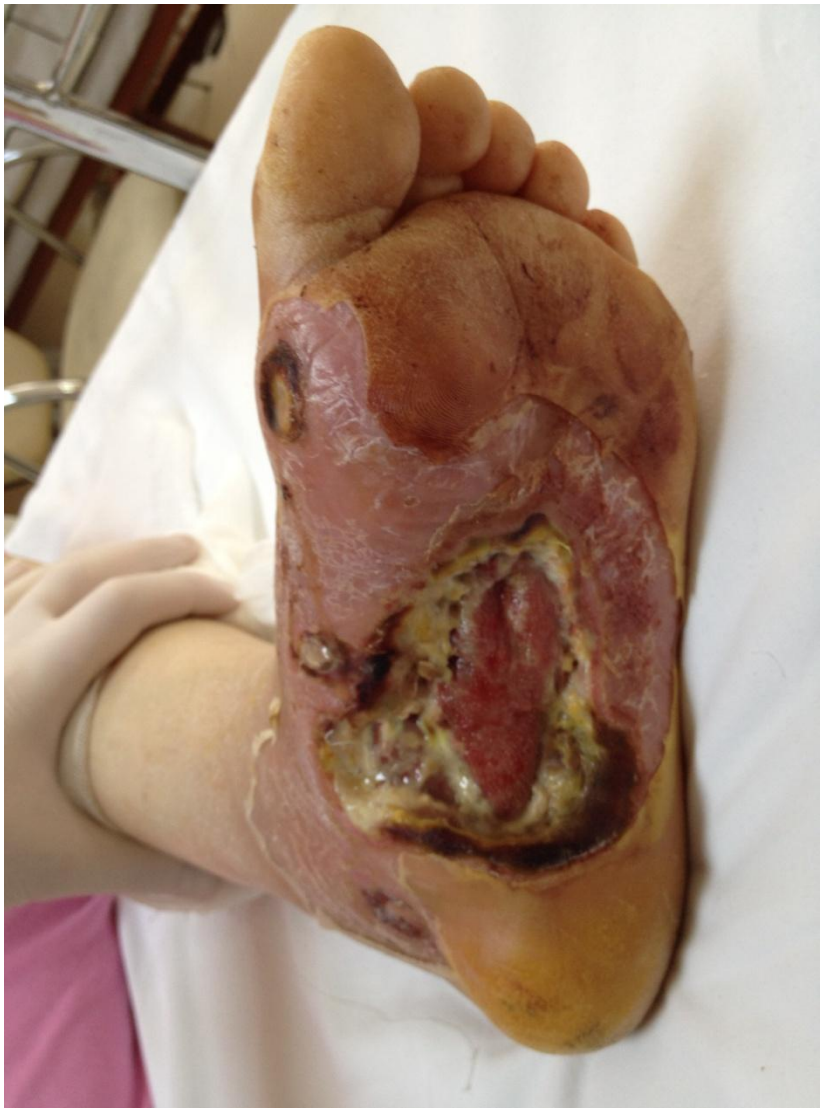


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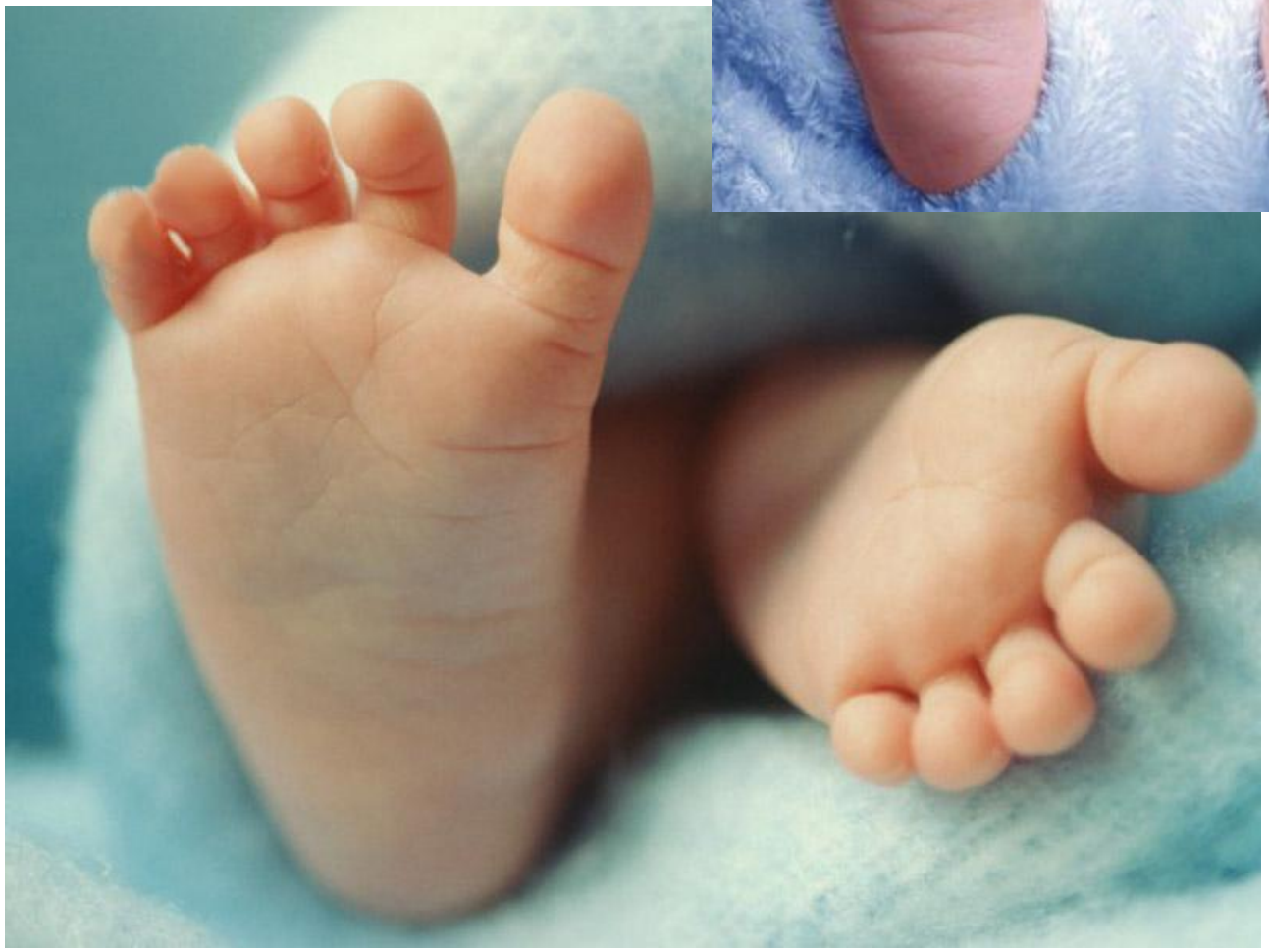

LABORATUVAR BULGULARI

- WBC: 19 700 /mm3
- PNL: 17 500 /mm3
- **Hb: 8.8** mg/dl
- **Htc: % 26**
- Plt: 243 000 /mm3
- ESH: 132 mm/sa
- CRP: 156 mg/L (N: 0-3)
- Prokalsitonin: 9.9
- HbA1c: % 7.9
- Glukoz: 285 mg/dl
- Üre: 54 mg/dl
- Kreatinin: 0.9 mg/dl

TİT: Normal

- AST: 37 U/L
- ALT: 27 U/L
- T. Protein: 6.9 mg/dl
- **Alb.: 2.1** mg/dl
- Glb: 3 mg/dl
- Na: 125 mEq/L
- K: 4.4 mEq/L
- PT: 16 sn
- PTT: 62 sn
- INR: 1.4
- T.kolesterol: 149 mg/ dl
- Trigliserid: 88 mg/ dl
- HDL: 13 mg/ dl
- LDL: 45 mg/ dl

Table 2. Infectious Diseases Society of America and International Working Group on the Diabetic Foot Classifications of Diabetic Foot Infection

Clinical Manifestation of Infection	PEDIS Grade	IDSA Infection Severity
No symptoms or signs of infection	1	Uninfected
Infection present, as defined by the presence of at least 2 of the following items: • Local swelling or induration • Erythema • Local tenderness or pain • Local warmth • Purulent discharge (thick, opaque to white or sanguineous secretion)		
Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). If erythema, must be >0.5 cm to ≤2 cm around the ulcer. Exclude other causes of an inflammatory response of the skin (eg, trauma, gout, acute Charcot neuro-osteoarthropathy, fracture, thrombosis, venous stasis).	2	Mild
Local infection (as described above) with erythema > 2 cm, or involving structures deeper than skin and subcutaneous tissues (eg, abscess, osteomyelitis, septic arthritis, fasciitis), **and** No systemic inflammatory response signs (as described below)	3	Moderate
Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following: • Temperature >38°C or <36°C • Heart rate >90 beats/min • Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg • White blood cell count >12 000 or <4000 cells/μL or ≥10% immature (band) forms	4	Severe[a]

WAGNER : **Evre 3:** Apse ve/veya osteomiyeliti içeren derin ülser

Olası Etkenler ?

Table 5. Pathogens Associated with Specific Diabetic Foot Infection Syndromes (Modified from Reference 2)

Diabetic Foot Infection Syndrome	Pathogen
Cellulitis without ulceration	β-hemolytic streptococci (especially group B) and *Staphylococcus aureus*
Ulcer or wound, recently developed and no prior antibiotic treatment	*S. aureus* and β-hemolytic streptococci
Ulcer or wound, chronic or recent antibiotic treatment	Usually polymicrobial – *S. aureus* and β-hemolytic streptococci plus *Enterobacteriaceae*. Enterococci if previous cephalosporin therapy.
Ulcer or wound, prior hydrotherapy or green-blue colored drainage	*Pseudomonas aeruginosa* (often in combination with other organisms)
Extensive necrosis or gangrene, ischemic limb, feculent odor ("fetid foot")	Polymicrobial – mixed aerobic Gram-positive cocci (including enterococci), *Enterobacteriaceae*, nonfermentative Gram-negative rods, and obligate anaerobes
Healthcare-associated	MRSA; ESBL-producing Gram-negative rods

MRSA: Methicillin-resistant *Staphylococcus aureus*

ESBL: Extended spectrum β-lactamase

Lipsky BA, et al. Guidelines for Diabetic Foot Infections . CID 2004:39; 885-910

- **Sol ayak 1. parmak medialindeki abse** drenajı + debridman
- Diyabetik ayak yarasından ve abseden derin doku örneği
- Gluteal abse bölgesinden aspirasyon ile örnek
- 2 adet kan kültürü
- **Piperasilin-tazobaktam** (3x4.5 g IV)
- Clexane 0.4 cc (1x1 sc)

IDSA GUIDELINES

23 Mayıs 2012

2012 Infectious Diseases Society of America Clinical Practice Guideline for the Diagnosis and Treatment of Diabetic Foot Infections[a]

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		against streptococci	
Moderate (may be treated with oral or initial parenteral agent[s]) or severe (usually treated with parenteral agent[s])	MSSA; *Streptococcus* spp; Enterobacteriaceae; obligate anaerobes	Levofloxacin[b]	Once-daily dosing; suboptimal against *S. aureus*
		Cefoxitin[b]	Second-generation cephalosporin with anaerobic coverage
		Ceftriaxone	Once-daily dosing, third-generation cephalosporin
		Ampicillin-sulbactam[b]	Adequate if low suspicion of *P. aeruginosa*
		Moxifloxacin[b]	Once-daily oral dosing. Relatively broad-spectrum, including most obligate anaerobic organisms
		Ertapenem[b]	Once-daily dosing. Relatively broad-spectrum including anaerobes, but not active against *P. aeruginosa*
		Tigecycline[b]	Active against MRSA. Spectrum may be excessively broad. High rates of nausea and vomiting and increased mortality warning. Nonequivalent to ertapenem + vancomycin in 1 randomized clinical trial
		Levofloxacin[b] or ciprofloxacin[b] with clindamycin[b]	Limited evidence supporting clindamycin for severe *S. aureus* infections; PO & IV formulations for both drugs
		Imipenem-cilastatin[b]	Very broad-spectrum (but not against MRSA); use only when this is required. Consider when ESBL-producing pathogens suspected
	MRSA	*Linezolid*[b]	Expensive; increased risk of toxicities when used >2 wk
		Daptomycin[b]	Once-daily dosing. Requires serial monitoring of CPK
		Vancomycin[b]	Vancomycin MICs for MRSA are gradually increasing
	Pseudomonas aeruginosa	***Piperacillin-tazobactam***[b]	TID/QID dosing. Useful for broad-spectrum coverage. *P. aeruginosa* is an uncommon pathogen in diabetic foot infections except in special circumstances (2)

Table 5. Recommendations for Collection of Specimens for Culture From Diabetic Foot Wounds

Do
• Obtain an appropriate specimen for culture from almost all infected wounds • Cleanse and debride the wound before obtaining specimen(s) for culture • Obtain a tissue specimen for culture by scraping with a sterile scalpel or dermal curette (curettage) or biopsy from the base of a debrided ulcer • Aspirate any purulent secretions using a sterile needle and syringe • Promptly send specimens, in a sterile container or appropriate transport media, for aerobic and anaerobic culture (and Gram stain, if possible)
Do not
• Culture a clinically uninfected lesion, unless for specific epidemiological purposes • Obtain a specimen for culture without first cleansing or debriding the wound • Obtain a specimen for culture by swabbing the wound or wound drainage

Mikroskobik Inceleme:

Direkt Gram Boyama :Lökositler görüldü.Bakteri görülmedi.

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ABSE KÜLTÜRÜ	Abse	()	

Sağ Gluteal Abse

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Abse	()	

KÜLTÜR:

1.Sonuç:Escherichia coli üredi.

Uygulanan Antibiyotikler:

Antibiyotik	1
Amoksisilin/Klavulonik Asit	Dirençli
Ampisilin/Sulbaktam	Dirençli
Sefazolin	Dirençli
Seftriakson	Dirençli
Sefotaksim	Dirençli
Gentamisin	Dirençli
Amikasin	Az Hassas
Kotrimoksazol	Hassas
Siprofloksasin	Dirençli
İmipenem	Hassas
Sefuroksim	Dirençli
Sefoperazon/Sulbaktam	Hassas
Meropenem	Hassas
Sefepim	Dirençli
Piperasilin/Tazobaktam	Hassas
Tigesiklin	Hassas

Mikroskobik Inceleme:

Direkt Gram Boyama :Lökosit ve gram (-) basil görüldü.

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
YARA KÜLTÜRÜ	Yara	()	

Ayak Derin Doku

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Yara	()	

KÜLTÜR:

1.Sonuç:Proteus spp. üredi.

Uygulanan Antibiyotikler:

Antibiyotik	1
Amoksisilin/Klavulonik Asit	Hassas
Ampisilin/Sulbaktam	Hassas
Sefazolin	Hassas
Seftriakson	*****
Sefotaksim	*****
Gentamisin	Dirençli
Amikasin	Hassas
Kotrimoksazol	Dirençli
Siprofloksasin	Hassas
İmipenem	*****
Sefuroksim	Hassas
Sefoperazon/Sulbaktam	*****
Meropenem	*****
Sefepim	*****
Piperasilin/Tazobaktam	Hassas
Tigesiklin	Hassas

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
BOYALI MİKRO	Yara	()	

Ayak I. parmaktaki abse

Mikroskobik Ince

Direkt Gram Boyama :Lökosit ve gram (-) basil görüldü.

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Yara	()	

KÜLTÜR:

1.Sonuç:Pseudomonas spp. üredi.
2.Sonuç:Proteus vulgaris üredi.

Uygulanan Antibiyotikler:

Antibiyotik	1	2
Amikasin	Hassas	Hassas
Siprofloksasin	Dirençli	Hassas
İmipenem	Hassas	Hassas
Seftazidim	Dirençli	Hassas
Sefoperazon/Sulbaktam	Dirençli	Hassas
Meropenem	Dirençli	Hassas
Sefepim	Az Hassas	Hassas
Piperasilin/Tazobaktam	Dirençli	Hassas
Doripenem	Dirençli	
Amoksisilin/Klavulonik Asit		Hassas
Ampisilin/Sulbaktam		Hassas
Sefazolin		Hassas
Seftriakson		Hassas
Sefotaksim		Hassas
Kotrimoksazol		Dirençli
Sefuroksim		Hassas

3. günde imipenem (4x500 mg ıv)

İZLEM

Alt ekstremite Arteriyel Doppler USG:

- İntimal kalınlaşma ve yer yer cidar kalsifikasyonları

Alt ekstremite Venöz Doppler USG: Normal

- **Plastik cerrahi** : debridman
- **Dahiliye ve diyetisyen** : KŞ regülasyonu
- **Genel cerrahi ve ortopedi konsültasyonları**
- **Göz konsültasyonu** : diyabetik retinopati bulgusu yok
- **Nefroloji:** diyabetik nefropati dışlandı.
- Yara-yanık merkezinde günlük pansumanı ve HBO tedavisi

7. gün:

- WBC: 13400 /mm^3
- PNL: 9200 /mm^3
- CRP:149 mg/L

İZLEM

9. gün:

- Analjezik enjeksiyonu uygulanan **sol gluteal** bölgesinde şişlik, kızarıklık, ısı artışı ve fluktuasyon
- **Yüzeyel USG:** Yumuşak dokuda 70x27 mm **abse**
- Drenaj + kültür için örnek

- Tekrarlanan kan kültürlerinde üreme olmadı.

SOL GLUTEAL ABSE

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Yara	()	

KÜLTÜR:

1.Sonuç:Enterobacter spp. üredi.

Uygulanan Antibiyotikler:

Antibiyotik	1
Amoksisilin/Klavulonik Asit	Dirençli
Ampisilin/Sulbaktam	Dirençli
Sefazolin	Dirençli
Seftriakson	Dirençli
Sefotaksim	Dirençli
Gentamisin	Dirençli
Amikasin	Hassas
Kotrimoksazol	Hassas
Siprofloksasin	Hassas
İmipenem	Hassas
Sefuroksim	Dirençli
Sefoperazon/Sulbaktam	Hassas
Meropenem	Hassas
Sefepim	Hassas
Piperasilin/Tazobaktam	Az Hassas
Tigesiklin	Hassas

- **WBC: 18700 /mm^3,**
- **CRP: 104 mg/L,**
- **ESH: 115 mm/sa**

Osteomiyelit ?

- Kemik dokuyla ilişkili geniş (>2x2 cm) ve derin (>3 cm) ayak lezyonu
- 6 haftadır (>2 hafta)

- **Diyabetik ayak osteomyeliti**ni destekleyen klinik bulgular:

1. Kronik, derin yumuşak doku ülseri
2. ESH , CRP yüksekliği
3. Pozitif "probe to bone" testi

Lipsky BA. Bone of contention: diagnosing diabetic foot osteomyelitis. *Clin Infect Dis. 2008; 47(4): 528-30.*

- Pozisyon verme ve kooperasyonda problemi
- **MR Görüntüleme**: 2 kez denendi, yapılamadı.
- Sedasyon uygulanıp MR çekilmesi planlandı fakat anestezi tarafından sedasyon uygulanamadı.

KEMİK SİNTİGRAFİSİ

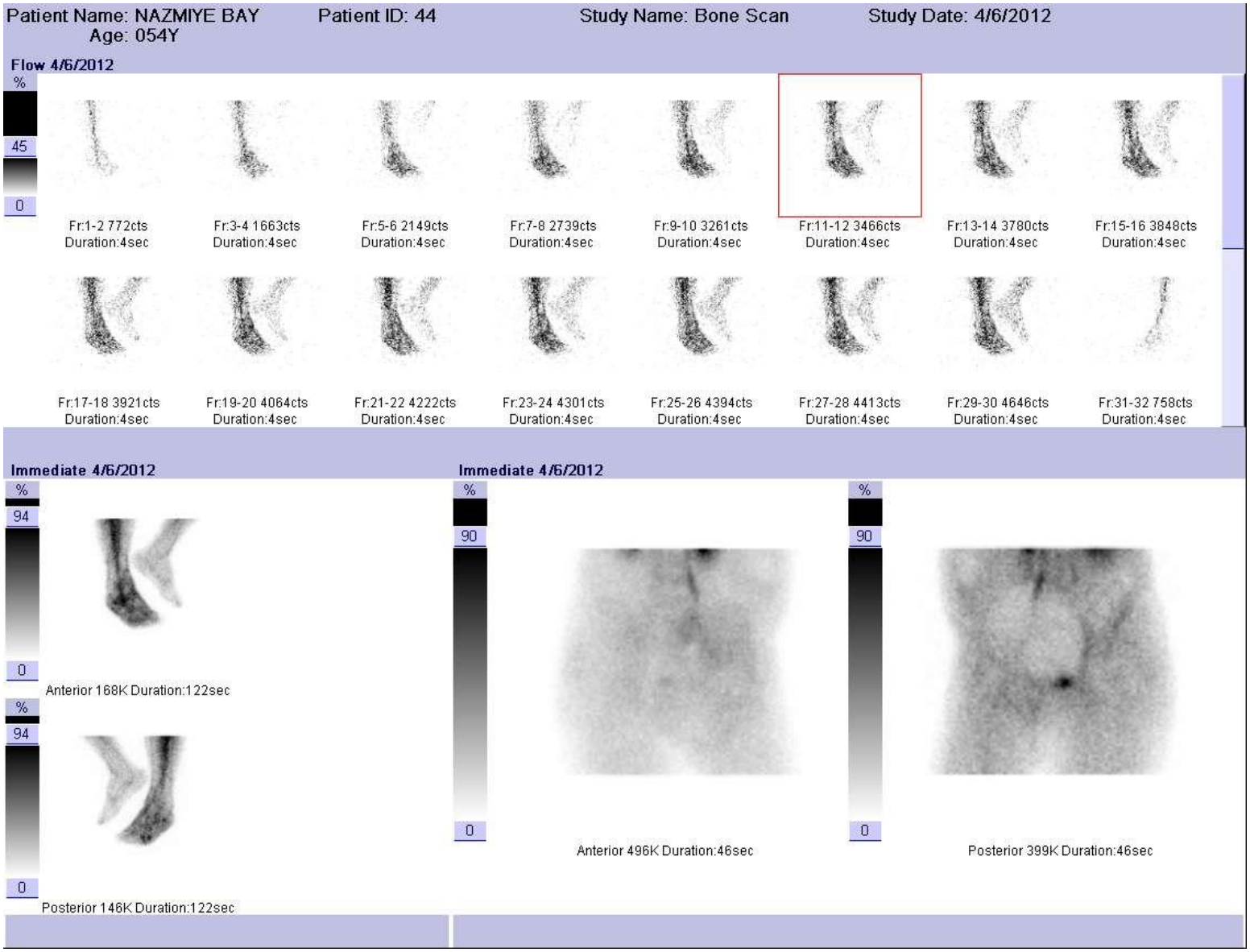
Schwegler B, Stumpe KD, Weishaupt D, *et al. Unsuspected* osteomyelitis is frequent in persistent diabetic foot ulcer and better diagnosed by MRI than by 18F-FDG PET or 99mTc-MOAB. *J Intern Med 2008; 263(1): 99-106.*

Tüm vücut kemik sintigrafisi (Tc-99m)

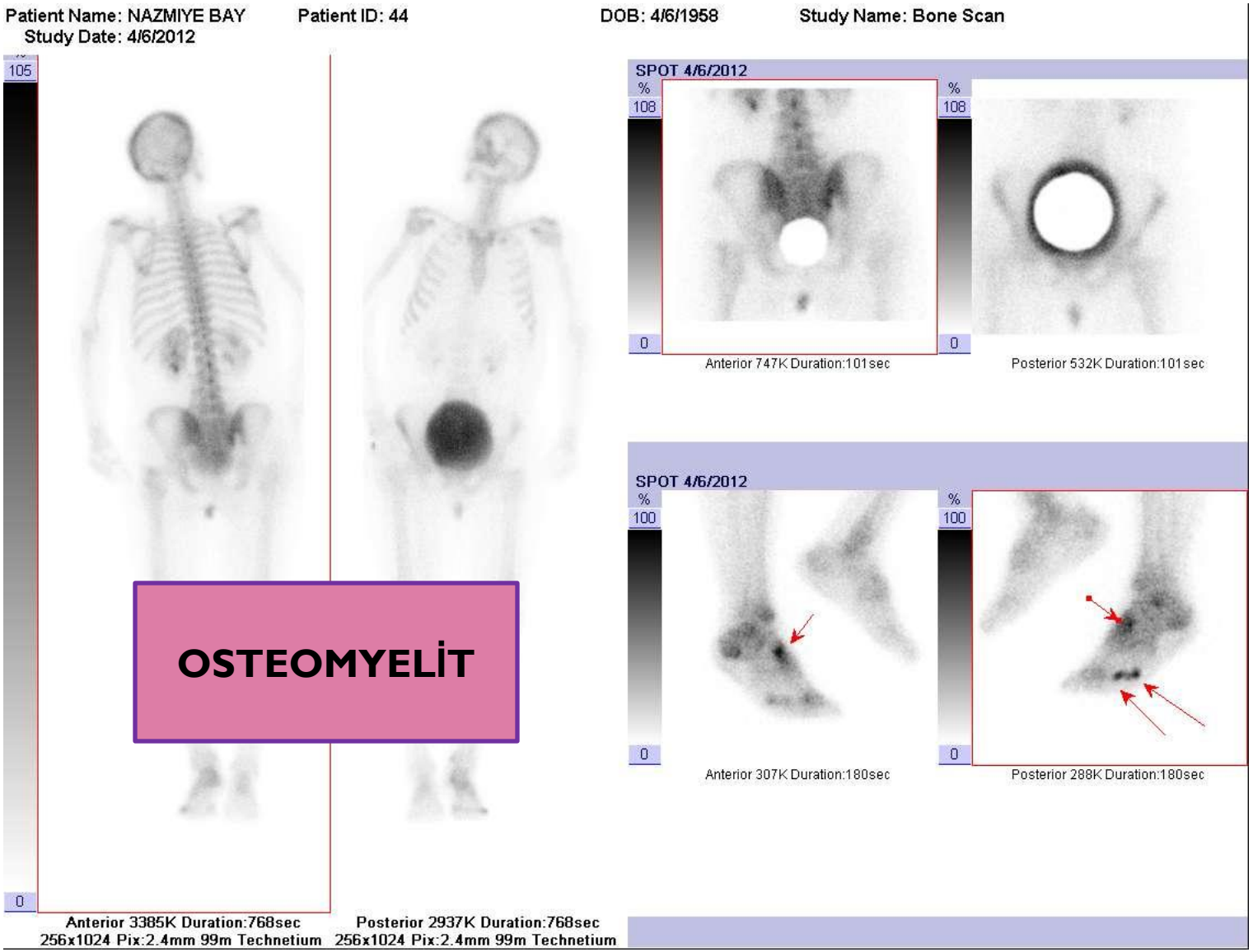
- **Sol kruris ve sol ayakta belirgin artmış akümülasyon,**
- **Sol tibiotalar**
- **Talar,**
- **4. ve 5. metatarsofalengeal eklemlerde daha belirgin**

heterojen tarzda **şiddetli artmış aktivite tutulumları**

Kemik sintigrafisi (Tc-99m)



Kemik sintigrafisi (Tc-99m)



LABORATUVAR BULGULARI

	1. Gün	14. Gün	18. Gün	28. Gün
WBC (/mm3)	19 700	12 700	9700	11500
PNL (/mm3)	17 500	9 400	7100	9 000
ESH (mm/sa)	**132**	**117**	**115**	
CRP (mg/L)	**156**	**76**	**87**	**103**
Hb / Htc (mg/dl)(%)	8.8 / 26	9.2 / 27	8.3 /27	8.5 / 29
PLT (/mm3)	243000	318000	352000	328000
AKŞ (mg/dl)	236	212	156	121
Üre (mg/dl)	54	30	43	38
Kreatinin(mg/dl)	0.9	0.6	0.5	0.6
AST (U/L)	37	21	14	12
ALT (U/L)	27	12	8	8

Osteomyelit Tanı Kriterleri

*1. **pozitif kültür** veya

2. **Karakteristik histopatoloji** (inflamatuvar hücreler, nekroz)

3. MR görüntüleme**

**Birçok çalışmada DİYABETİK AYAK OSTEOMYELİTİ tanısı için en iyi görüntüleme yöntemi

*Senneville E. [Diabetic foot osteomyelitis]. *Rev Prat. 2007; 57: 991-4.*

**Russell JM, Peterson JJ, Bancroft LW. MR Imaging of the Diabetic Foot. *Magn Reson Imaging Clin N Am. 2008; 16(1): 59-70.*

Osteomyelit

İZLEM

- **21. gün:** yarada akıntının kesildiği, nekrotik dokuların azaldığı, infeksiyon tablosunun gerilediği ve yaranın iyiye gittiği gözlendi.

- **Yara bakım merkezi hekimi:** yumuşak doku defekti çok büyük olduğundan VAC (negatif basınçlı pansuman) tedavisi veya plastik cerrahi tarafından rekonstrüksiyon amaçlı flap operasyonu önerdi.

- Ortopedi tarafından **BT anjiyografi** planlandı. Hasta tedaviyi red formu imzalayarak yakınlarının isteği ile taburcu edildi.

- Toplamda 26 gün antimikrobiyal tedavi, 21 seans hiperbarik oksijen tedavisi

- **Hastanın son durumu:**

Table 11. Suggested Route, Setting, and Duration of Antibiotic Therapy, by Clinical Syndrome

Site of Infection, by Severity or Extent	Route of Administration	Setting	Duration of Therapy
Soft-tissue only			
Mild	Topical or oral	Outpatient	1–2 wk; may extend up to 4 wk if slow to resolve
Moderate	Oral (or initial parenteral)	Outpatient/ inpatient	1–3 wk
Severe	Initial parenteral, switch to oral when possible	Inpatient, then outpatient	2–4 wk
Bone or joint			
No residual infected tissue (eg, postamputation)	Parenteral or oral	...	2–5 d
Residual infected soft tissue (but not bone)	Parenteral or oral	...	1–3 wk
Residual infected (but viable) bone	Initial parenteral, then consider oral switch	...	4–6 wk
No surgery, or residual dead bone postoperatively	Initial parenteral, then consider oral switch	...	≥3 mo

OLGU - 2

- O.Ç., 54 y, erkek, 63 kg, 169 cm Yatış tarihi: 25.4.012
- **Yakınma:** Sağ ayakta akıntılı yara, ateş
- **Öykü**:
- 4 ay önce sağ ayak 4. ve 5. parmaklarda siyah- mor renkli yara gelişmiş. Parmaklar kendiliğinden koparak düşmüş (oto-ampütasyon).
- 2 aydır siprofloksasin 2x500 mg tb ve HBO tedavisine rağmen yarası geçmemiş.
- 40 °C'ye varan ateş
- **Özgeçmiş:**

  25 yıldır Tip 2 DM (İnsülin kullanıyor)

  2006' da ayak 2. parmak DAİ nedeniyle ampüte.

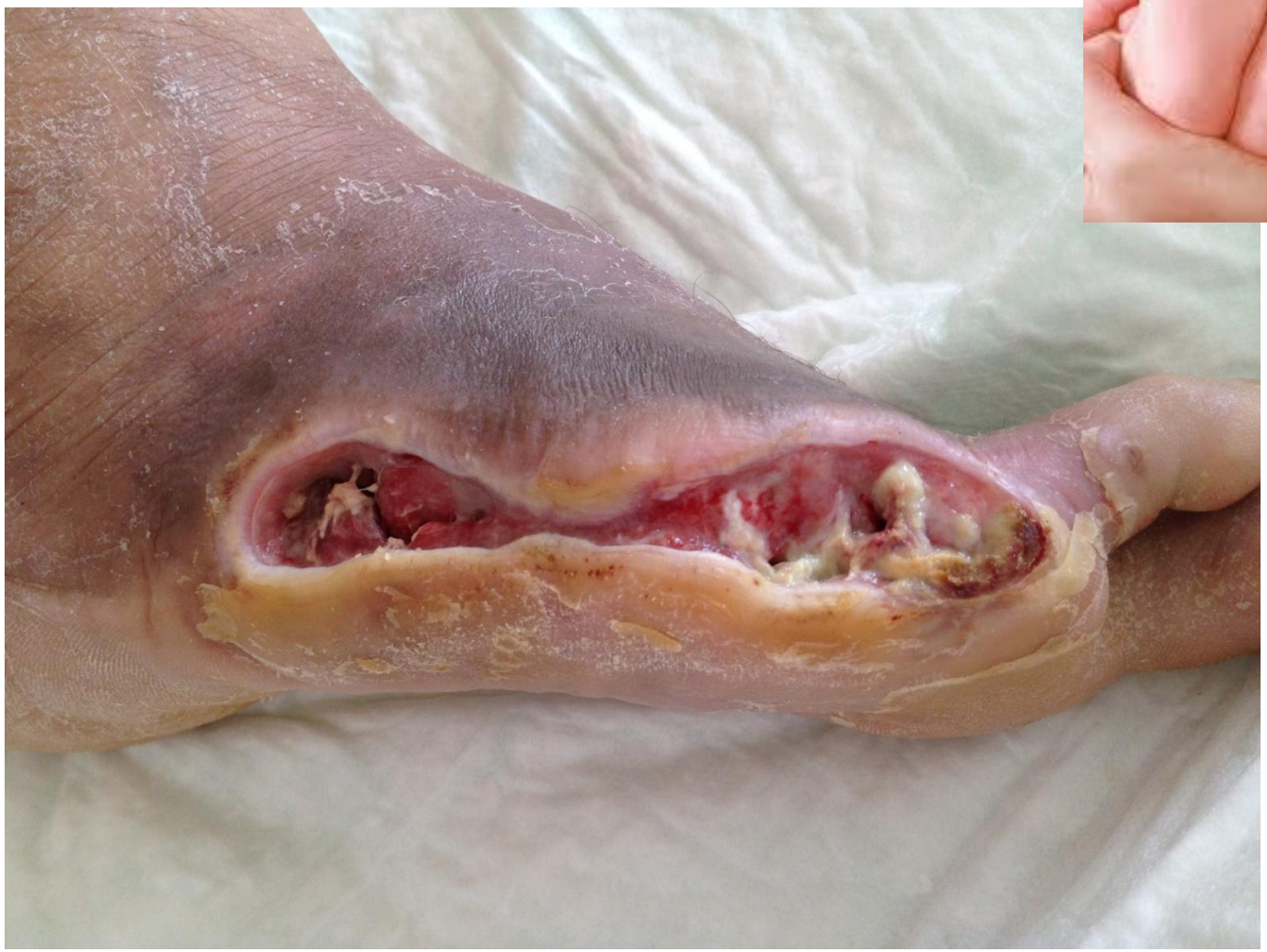
  6 yıldır KBY (hemodiyalize giriyor)

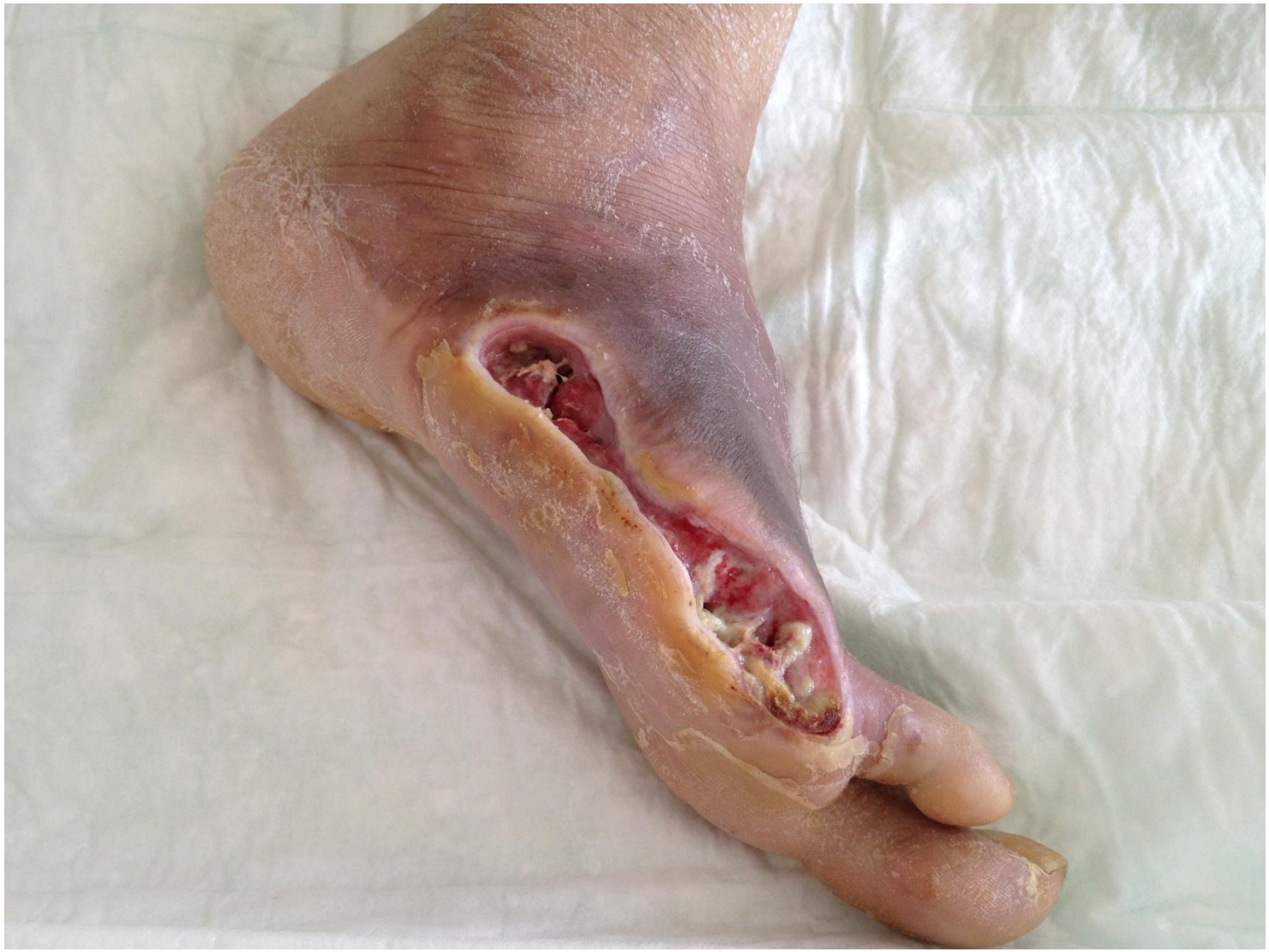
  HT (Nifedipine 1x60 mg tb)

  Parkinson Hst.

FİZİK İNCELEME

- A: **38.3**°C Nb: **98** /dk TA:130/70 mmHg SS: 14/dk
- GD: Orta-iyi, bilinç açık, koopere, oriyente
- Sağ ayak lateralinde pürülan akıntılı yaklaşık **10x3 cm** boyutlarında, **2 cm** derinliğinde **kemiğin ekspoze olduğu** ülsere nekrotik açık yara
- Hemodiyaliz fistül yerinde inflamasyon yok
- Periferik nabızlar alınamıyor.
- Dizaltında bilateral duyu kaybı (+)
- Sistem muayeneleri doğal.





Direkt grafi

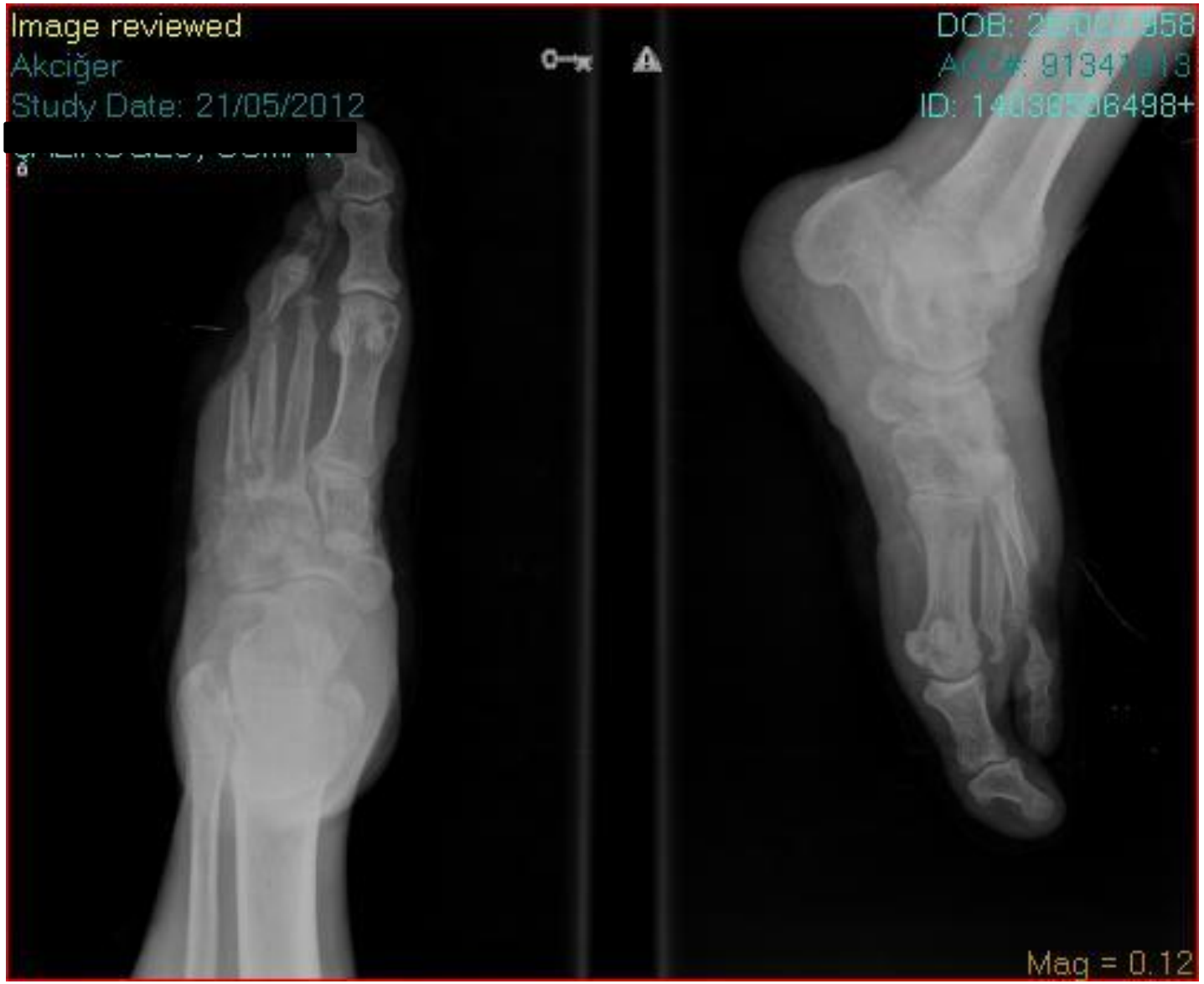


Table 2. Infectious Diseases Society of America and International Working Group on the Diabetic Foot Classifications of Diabetic Foot Infection

Clinical Manifestation of Infection	PEDIS Grade	IDSA Infection Severity
No symptoms or signs of infection	1	Uninfected
Infection present, as defined by the presence of at least 2 of the following items: • Local swelling or induration • Erythema • Local tenderness or pain • Local warmth • Purulent discharge (thick, opaque to white or sanguineous secretion)		
Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). If erythema, must be >0.5 cm to ≤2 cm around the ulcer. Exclude other causes of an inflammatory response of the skin (eg, trauma, gout, acute Charcot neuro-osteoarthropathy, fracture, thrombosis, venous stasis).	2	Mild
Local infection (as described above) with erythema > 2 cm, or involving structures deeper than skin and subcutaneous tissues (eg, abscess, osteomyelitis, septic arthritis, fasciitis), **and** No systemic inflammatory response signs (as described below)	3	Moderate
Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following: • Temperature >38°C or <36°C • Heart rate >90 beats/min • Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg • White blood cell count >12 000 or <4000 cells/μL or ≥10% immature (band) forms	4	Severe[a]

Risk faktörleri

- ✓ İleri yaş
- ✓ Uzun süreli diyabet varlığı
- ✓ Hiperglisemi
- ✓ Hipertansiyon
- ✓ Periferik nöropati
- ✓ Periferik vasküler hastalık
- ✓ Diyabetin komplikasyonları (retinopati-nefropati)

Lipsky BA. A report from the international consensus on diagnosing and treating the infected diabetic foot. ***Diabetes Metab Res Rev. 2004; 20 (Suppl 1): S68-77.***

Lipsky BA, Berendt AR, Deery HG, 2nd , ***et al. IDSA Guidelines:*** **Diagnosis and treatment of diabetic foot infections.** ***Clin Infect Dis. 2004; 39: 885-910.***

LABORATUVAR BULGULARI

- WBC: 15 300 /mm3
- PNL: 14 600 /mm3
- Hb: 8.5 mg/dl
- Htc: % 26
- Plt: 205 000 /mm3
- ESH: 98 mm/sa
- CRP: › 214 mg/L (N: 0-3)
- Prokalsitonin: 3.9
- HbA1c: 6.6
- Glukoz: 358 mg/dl
- Üre: 59 mg/dl
- Kreatinin: 4.5 mg/dl

TİT: Normal

- AST: 28 U/L
- ALT: 32 U/L
- T. Protein: 5.9 mg/dl
- Alb.: 2.4 mg/dl
- Glb: 3.5 mg/dl
- Na: 130 mEq/L
- K: 4.7 mEq/L
- PT: 12 sn
- PTT: 36 sn
- INR: 1.1
- T. kolesterol: 156 mg/ dl
- Trigliserid: 302 mg/ dl
- HDL: 13 mg/ dl
- LDL: 81 mg/ dl

- Diyabetik ayak yarasından **derin doku örneği** ve 2 adet **kan kültürü**
- Empirik İmipenem (2x 250 mg IV) ve Linezolid 2x600 mg IV
- Clexane 0.4 cc (1x1 sc)
- HBO tedavisine devam edildi.

BOYALI MİKROSKOBİK İNCELEME	Yara	()

Mikroskobik Inceleme:

Direkt Gram Boyama :Lökosit ve gram (-) basil görüldü.

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
YARA KÜLTÜRÜ	Yara	()	

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Yara	()	

KÜLTÜR:

1.Sonuç:Proteus spp. üredi.

Uygulanan Antibiyotikler:

Antibiyotik	1
Amoksisilin/Klavulonik Asit	Hassas
Ampisilin/Sulbaktam	Hassas
Sefazolin	Dirençli
Seftriakson	Dirençli
Sefotaksim	Dirençli
Gentamisin	Dirençli
Amikasin	Az Hassas
Kotrimoksazol	Dirençli
Siprofloksasin	Dirençli
İmipenem	*****
Sefuroksim	Dirençli
Sefoperazon/Sulbaktam	*****
Meropenem	*****
Sefepim	*****
Piperasilin/Tazobaktam	Hassas

İZLEM

- Plastik cerrahi : debridman,
- Dahiliye ve diyetisyen: KŞ regülasyonu
- Genel cerrahi ve ortopedi konsültasyonları
- **Göz konsültasyonu:**

Sağ makülada sert eksüda plağı, **ağır NPDR bulguları**, yoğun nükleer katarakt

sol fundus seçilemiyor, matür katarakt

- **Diyabetik nefropati** (+)
- Yara-yanık merkezinde günlük pansumanı ve hiperbarik oksijen tedavisi

- **Arteriyel Renkli Doppler USG:**

Yoğun cidar kalsifikasyonları ve aterom plakları

Her iki tibialis posteriorda **oklüzyon**

Her iki tibialis anteriorda **stenoz** bulguları

- **Venöz Renkli Doppler USG :**

Trombüs saptanmadı.

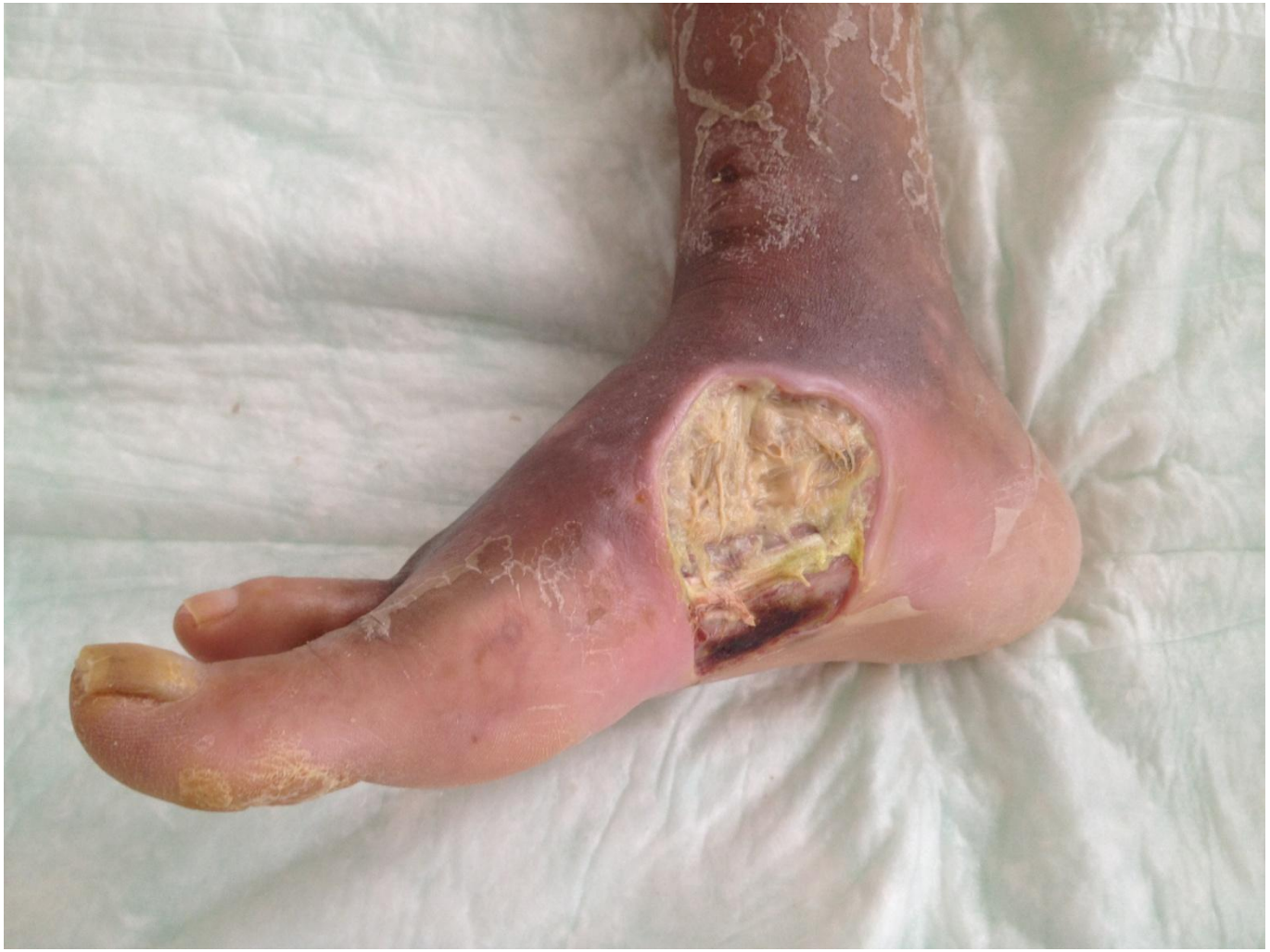
3. gün: Ateş düştü.

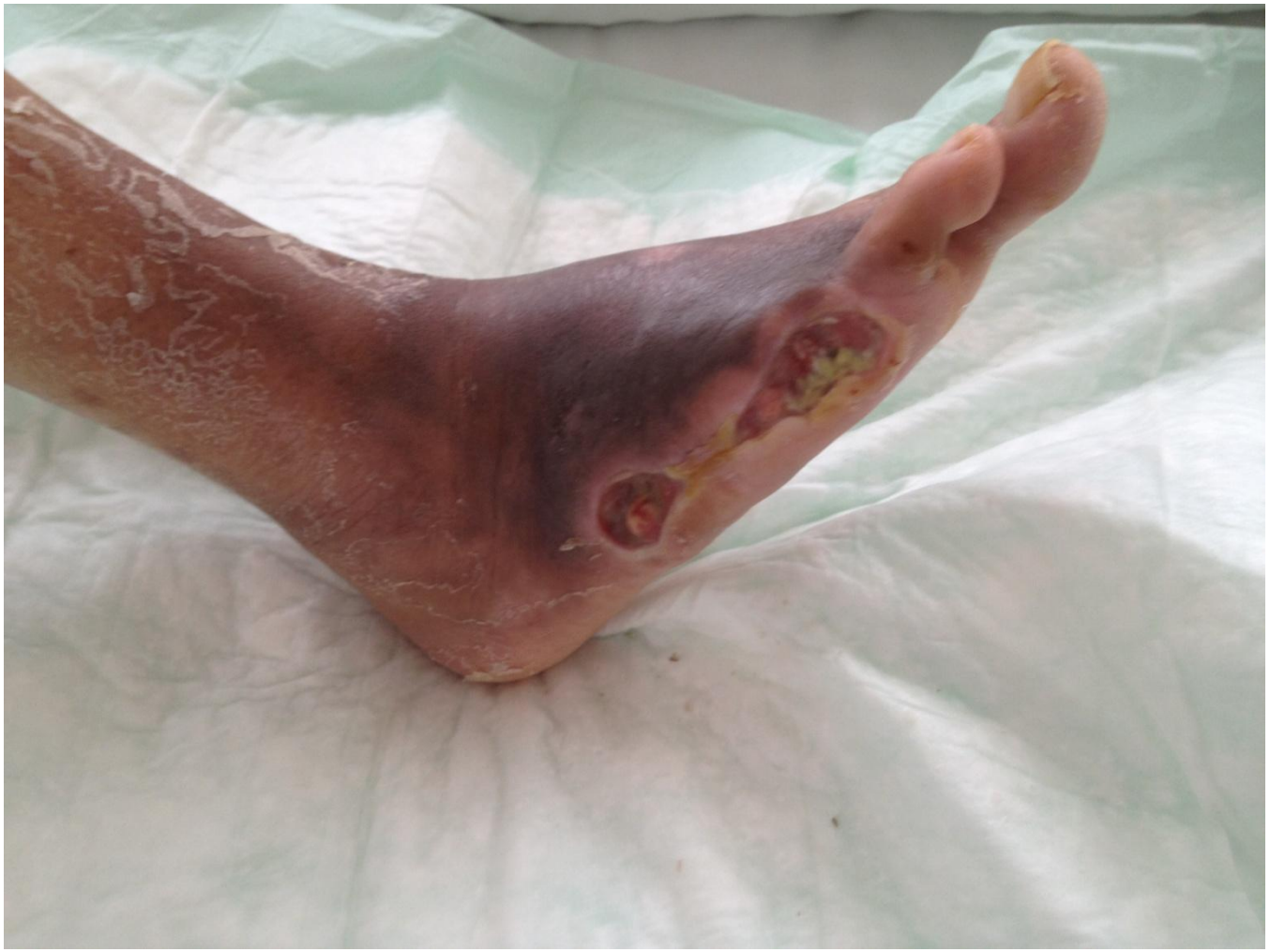
6. gün:

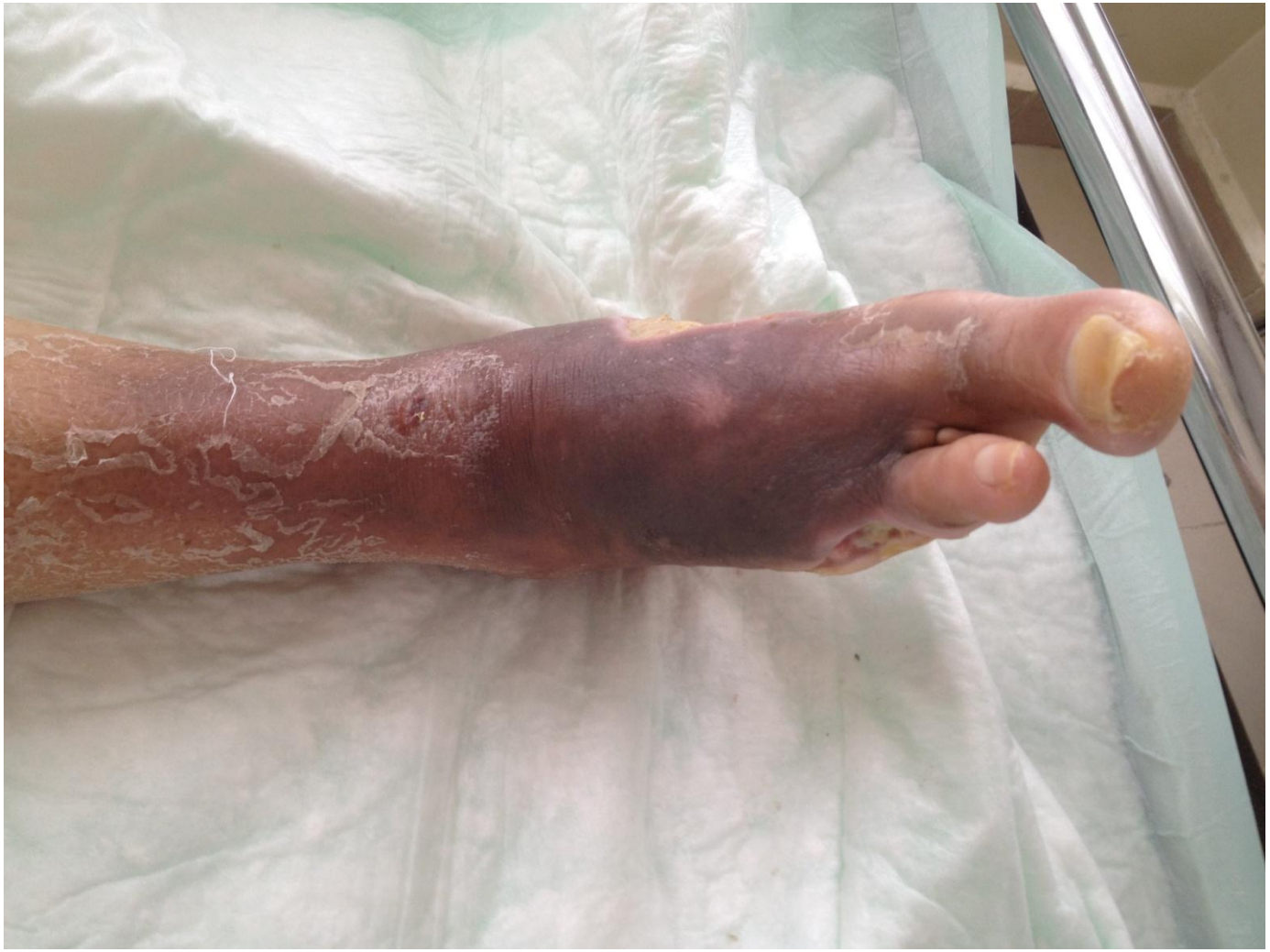
- Sağ ayak medialinde orta 1/3 te 5x5 cm yeni bir yara
- Nekrotik doku debride edildi.
- Akıntı minimal.
- Granülasyon ve epitelizasyon hiç yok.
- Yara kültüründe *E.coli* ve *Proteus spp* üredi (2.5.2012).
- Kan kültürlerinde üreme olmadı.

10. gün:

- Lateraldeki yara iyileşmekte







İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
YARA KÜLTÜRÜ	Yara	()	

İstek Adı	Örnek Türü	Alındığı Yer	Açıklama
ANTİBİYOGRAM DUYARLILIK TESTİ	Yara	()	

KÜLTÜR:

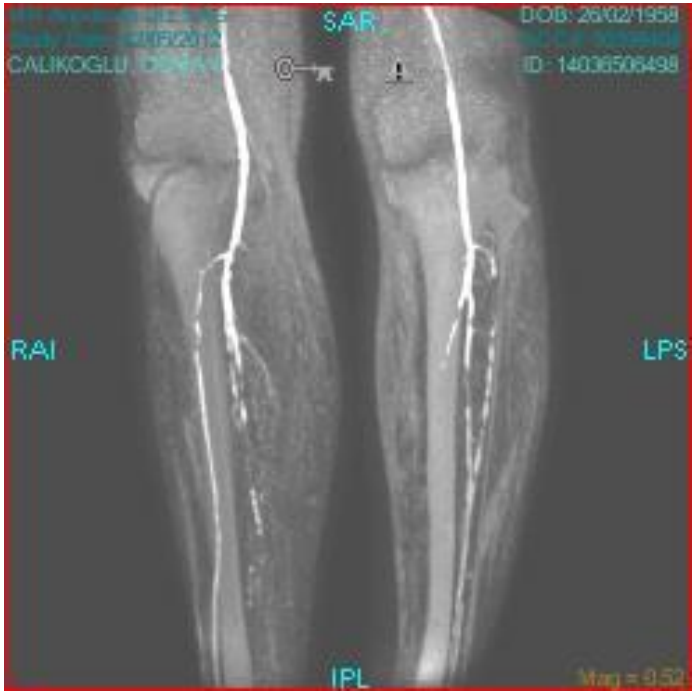
1.Sonuç:Escherichia coli üredi.
2.Sonuç:Proteus spp. üredi.

Uygulanan Antibiyotikler:

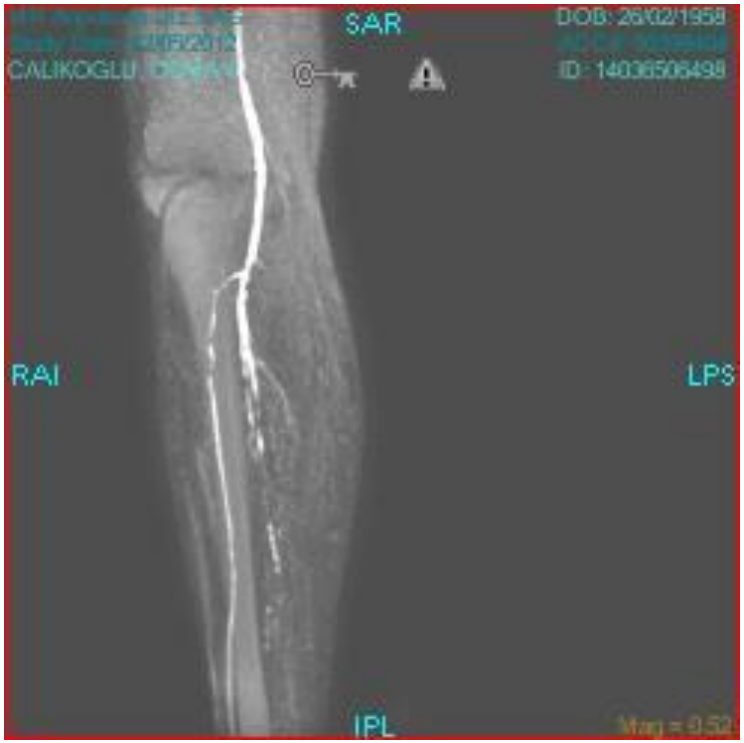
Antibiyotik	1	2
Amoksisilin/Klavulonik Asit	Dirençli	Hassas
Ampisilin/Sulbaktam	Dirençli	Hassas
Sefazolin	Dirençli	Dirençli
Seftriakson	Dirençli	Dirençli
Sefotaksim	Dirençli	Dirençli
Gentamisin	Hassas	Dirençli
Amikasin	Hassas	Az Hassas
Kotrimoksazol	Hassas	Dirençli
Siprofloksasin	Dirençli	Dirençli
İmipenem	Hassas	Hassas
Sefuroksim	Dirençli	Dirençli
Sefoperazon/Sulbaktam	Az Hassas	Hassas
Meropenem	Hassas	Hassas
Sefepim	Dirençli	Hassas
Piperasilin/Tazobaktam	Az Hassas	Hassas
Tigesiklin	Hassas	Dirençli

Sağ diz ve kruris MR- Anjio

- Sağ popliteal arterde küçük, sağ anterior tibial arterde yaygın aterosklerotik kontür düzensizlikleri
- Sağ anterior ve posterior tibial arterlerde prox. 1/3 e kadar **preoklüsif stenotik segmentler**, **distale akım seçilememekte**dir.



MR Anjiyografi



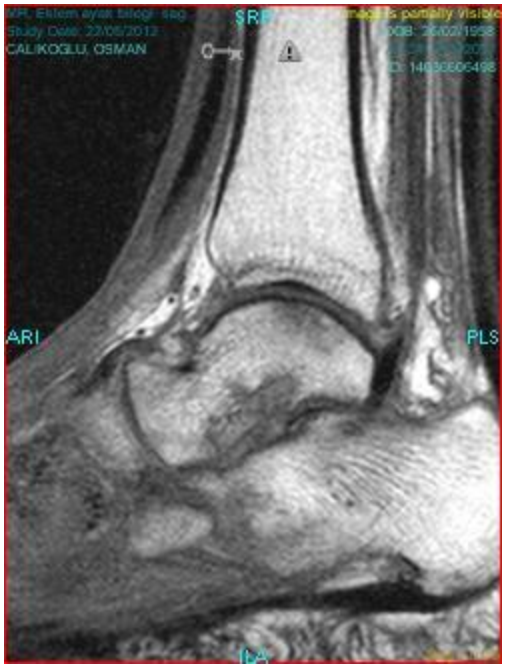
"The only risk factors for limb **amputation** found to be statistically significant by stepwise logistic regression analysis were **peripheral vascular disease and infection**."

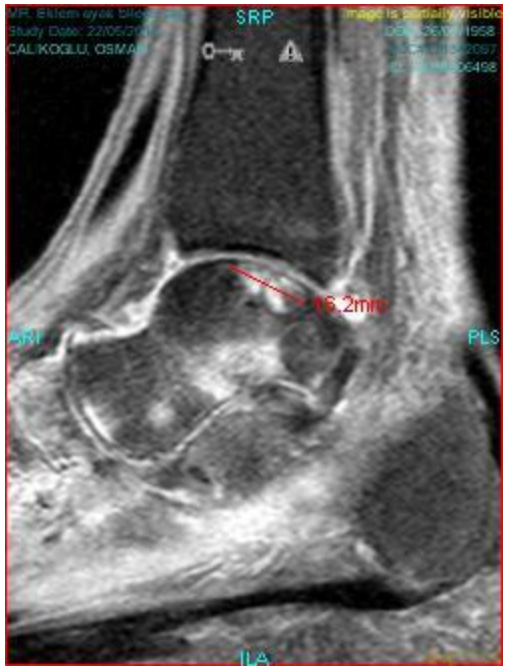
Nather A, Bee CS, Huak CY, *et al. Epidemiology of diabetic* foot problems and predictive factors for limb loss. *J Diabetes Complications. 2008; 22(2): 77-82.*

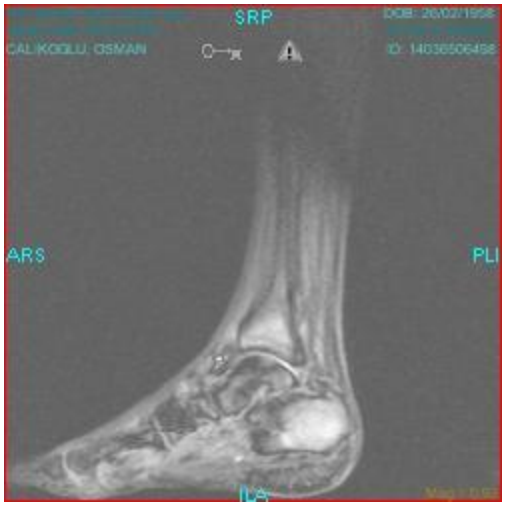
Sağ ayak- ayak bileği MR

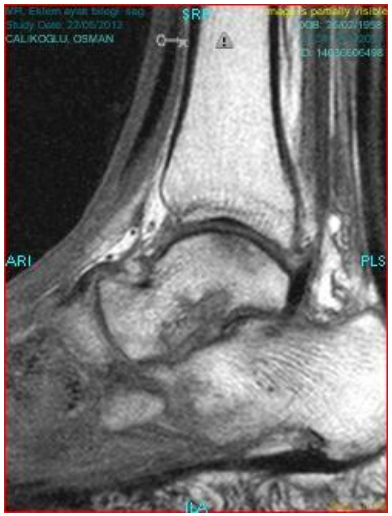
- Tibiotalar, fibulotalar, talokalkaneal, diğer intertarsal eklem mesafelerinde genel daralmalar
- Eklem yüz kartilaj kalınlıklarında azalmalar
- Eklem köşelerinde osteofitik sivrileşmeler
- Talus superomedial bölümünde 16 mm çapında fokal osteokondral defekt
- Talus ve kalkaneusta silik sınırlı, anterior tarsal kemiklerde belirgin T1W sekansta hipointens, T2W sekansta hiperintens karakterde sinyal intensite değişiklikleri:
- **Osteomyelit - eşlik eden yumuşak doku enfeksiyonu**

Ayak MR Görüntüleme









LABORATUVAR BULGULARI

	1. Gün	14. Gün	21. Gün	28. Gün
WBC (/mm3)	15 300	11500	7900	9500
PNL (/mm3)	14 600	9500	5400	6300
ESH (mm/sa)	**98**	**115**	**105**	
CRP (mg/L)	› 214	**210**	**118**	**140**
Prokalsitonin	**3.9**			**1.69**
AKŞ (mg/dl)	358	234	187	192
Hb / Htc	8.5 / 26	9.9 / 31	8.9 / 28	8.7 / 28
PLT	330 000	167 000	161 000	175 000
Üre (mg/dl)	59	93	59	61
Kreatinin(mg/dl)	4.5	6.7	4.3	5
AST (U/L)	39	23	12	11
ALT (U/L)	52	22	6	8

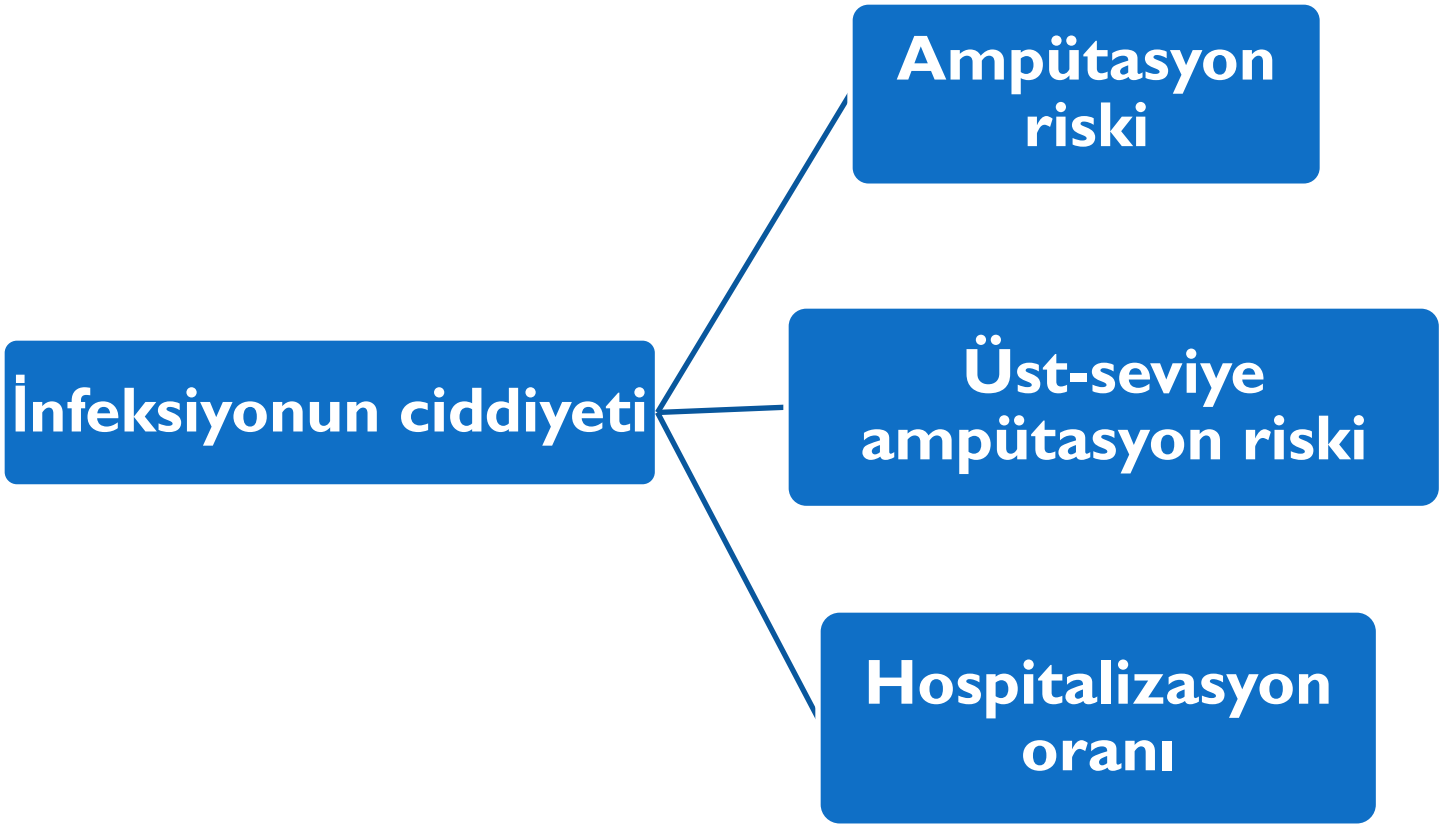
- **13. gün**:
- Ortopedi: **dizaltı ampütasyon kararı** aldı.
- Hasta ve yakınları kabul etmedi.
- 21.5.2012: İmipenem tedavisi 28. günde.
- Halen kliniğimizde izlenmekte…

Ağır DAİ Bulguları ve Hospitalizasyon Endikasyonları

Table 3. Characteristics suggesting a more serious diabetic foot infection and potential indications for hospitalization

(A) Findings suggesting a more serious diabetic foot infection	
Wound specific	
Wound	Penetrates into subcutaneous tissues, i.e., fascia, tendon, muscle, joint, bone
Cellulitis	Extensive (>2 cm), distant from ulceration or rapidly progressive
Local signs	Severe inflammation, crepitus, bullae, marked induration, discoloration, necrosis/gangrene, ecchymoses or petechiae
General	
Presentation	Acute onset or rapidly progressive
Systemic signs	Fever, chills, hypotension, confusion, volume depletion
Laboratory tests	Leukocytosis, severe or worsening hyperglycaemia, acidosis, azotaemia, electrolyte abnormalities
Complicating features	Presence of a foreign body (accidental or surgically implanted), puncture wound, abscess, arterial or venous insufficiency, lymphoedema
Current treatment	Progression while on apparently appropriate antibiotic therapy
(B) Factors suggesting hospitalization may be necessary	
• Severe infection (Table 3A)	
• Metabolic instability	
• Intravenous therapy needed (and not available/appropriate as outpatient)	
• Diagnostic tests needed that are not available as outpatient	
• Critical foot ischemia present	
• Surgical procedures (more than minor) required	
• Failure of outpatient management	
• Patient's inability or unwillingness to comply with outpatient-based treatment	
• Need for more complex dressing changes than patient/caregivers can provide	

Expert opinion on the management of infections in the diabetic foot. DIABETES/METABOLISM RESEARCH AND REVIEWS .Diabetes Metab Res Rev 2012; 28(Suppl 1): 163–178.



Lavery LA, et al. *Clin Infect Dis.* 2007; 44(4): 562-565.

Ciddi DAİ – Tedavi başarısızlığı

- Lökositoz
- CRP yüksekliği
- ESH yüksekliği
- Yüksek yara ağırlık skoru
- Hospitalizasyon
- Düşük serum albümini
- Erkek cinsiyet

Osteomyelit varlığı;

- Ayak infeksiyonunun başarılı eradikasyon oranını düşürür.
- Bacak ampütasyon riskini arttırır.

Lipsky BA, Sheehan P, Armstrong DG, et al. Clinical predictors of treatment failure for diabetic foot infections: data from a prospective trial. ***Int Wound J. 2007; 4(1): 30-8.***
Lipsky BA. Bone of contention: diagnosing diabetic foot osteomyelitis. ***Clin Infect Dis. 2008; 47(4): 528-30.***

Ciddi DAİ – Tedavi başarısı

- Rehberler eşliğinde hasta takip ve tedavisinin yönetimi
- Multlidisipliner takımların oluşturulmasıyla ampütasyon oranları azaltılabilmiş.

1. Dargis V, et al. Benefits of a multidisciplinary approach in the management of recurrent diabetic foot ulceration in Lithuania: a prospective study. Diabetes Care 1999; 22:1428–31.

2. Fitzgerald RH, et al. The diabetic rapid response acute foot team: 7 essential skills for targeted limb salvage. Eplasty 2009; 9:e15.

2012 Infectious Diseases Society of America Clinical Practice Guideline for the Diagnosis and Treatment of Diabetic Foot Infections[a]

Benjamin A. Lipsky,[1] Anthony R. Berendt,[2] Paul B. Cornia,[3] James C. Pile,[4] Edgar J. G. Peters,[5] David G. Armstrong,[6] H. Gunner Deery,[7] John M. Embil,[8] Warren S. Joseph,[9] Adolf W. Karchmer,[10] Michael S. Pinzur,[11] and Eric Senneville[12]

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Foot infections are a common and serious problem in persons with diabetes. Diabetic foot infections (DFIs) typically begin in a wound, most often a neuropathic ulceration. While all wounds are colonized with microorganisms, the presence of infection is defined by ≥2 classic findings of inflammation or purulence. Infections are then classified into mild (superficial and limited in size and depth), moderate (deeper or more extensive), or severe (accompanied by systemic signs or metabolic perturbations). This classification system, along with a vascular assessment, helps determine which patients should be hospitalized, which may require special imaging procedures or surgical interventions, and which will require amputation. Most DFIs are polymicrobial, with aerobic gram-positive cocci (GPC), and especially staphylococci, the most common causative organisms. Aerobic gram-negative bacilli are frequently copathogens in infections that are chronic or follow antibiotic treatment, and obligate anaerobes may be copathogens in ischemic or necrotic wounds.

Wounds without evidence of soft tissue or bone infection do not require antibiotic therapy. For infected wounds, obtain a post-debridement specimen (preferably of tissue) for aerobic and anaerobic culture. Empiric antibiotic therapy can be narrowly targeted at GPC in many acutely infected patients, but those at risk for infection with antibiotic-resistant organisms or with chronic, previously treated, or severe infections usually require broader spectrum regimens. Imaging is helpful in most DFIs; plain radiographs may be sufficient, but magnetic resonance imaging is far more sensitive and specific. Osteomyelitis occurs in many diabetic patients with a foot wound and can be difficult to diagnose (optimally defined by bone culture and histology) and treat (often requiring surgical debridement or resection, and/or prolonged antibiotic therapy). Most DFIs require some surgical intervention, ranging from minor (debridement) to major (resection, amputation). Wounds must also be properly dressed and off-loaded of pressure, and patients need regular follow-up. An ischemic foot may require revascularization, and some nonresponding patients may benefit from selected adjunctive measures. Employing multidisciplinary foot teams improves outcomes. Clinicians and healthcare organizations should attempt to monitor, and thereby improve, their outcomes and processes in caring for DFIs.

Received 21 March 2012; accepted 22 March 2012.

[a]It is important to realize that guidelines cannot always account for individual variation among patients. They are not intended to supplant physician judgment with respect to particular patients or special clinical situations. IDSA considers adherence to these guidelines to be voluntary, with the ultimate determination regarding their application to be made by the physician in the light of each patient's individual circumstances.

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REVIEW ARTICLE

Expert opinion on the management of infections in the diabetic foot

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Accepted: 12 October 2011

Summary

This update of the International Working Group on the Diabetic Foot incorporates some information from a related review of diabetic foot osteomyelitis (DFO) and a systematic review of the management of infection of the diabetic foot. The pathophysiology of these infections is now well understood, and there is a validated system for classifying the severity of infections based on their clinical findings. Diagnosing osteomyelitis remains difficult, but several recent publications have clarified the role of clinical, laboratory and imaging tests. Magnetic resonance imaging has emerged as the most accurate means of diagnosing bone infection, but bone biopsy for culture and histopathology remains the criterion standard. Determining the organisms responsible for a diabetic foot infection via culture of appropriately collected tissue specimens enables clinicians to make optimal antibiotic choices based on culture and sensitivity results. In addition to culture-directed antibiotic therapy, most infections require some surgical intervention, ranging from minor debridement to major resection, amputation or revascularization. Clinicians must also provide proper wound care to ensure healing of the wound. Various adjunctive therapies may benefit some patients, but the data supporting them are weak. If properly treated, most diabetic foot infections can be cured. Providers practising in developing countries, and their patients, face especially challenging situations. Copyright © 2012 John Wiley & Sons, Ltd.

Keywords diabetes mellitus; diabetic foot; infection; osteomyelitis; antibiotics; surgery; systematic review

Introduction

This report from the expert panel on infectious diseases of the International Working Group on the Diabetic Foot (IWGDF) is an update of the one published in 2004 [1], incorporating some information from a related IWGDF 2008 publication on osteomyelitis [2] and from the concurrently published 'Systematic Review of the Effectiveness of Interventions in the Management of Infection in the Diabetic Foot' [3]. Our intention is to present a brief overview to assist clinicians worldwide in diagnosing and treating foot infections in persons with diabetes. Separately, we have proposed 'Specific Guidelines on the Management of Diabetic Foot Infections', also published concurrently in this journal.

The development of a foot infection is associated with substantial morbidity, including discomfort, healthcare provider visits, antibiotic therapy, wound care and often surgical procedures. Furthermore, foot infection is now the most frequent diabetic complication requiring hospitalization and the most common precipitating event leading to lower extremity amputation [4–6]. Managing infection requires careful attention to properly diagnosing the condition,

HBO

- *4 randomize kontrollü çalışmanın analizi
  - Toplam 147 hastada; tedavide yaralı
  - Majör ampütasyon oranlarını azalttığı

*Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. *Cochrane Database Syst Rev. 2004: (2): CD004123.*

**Bir çalışmada etkisiz

**Berendt AR. Counterpoint: hyperbaric oxygen for diabetic foot wounds is not effective. *Clin Infect Dis. 15 2006; 43(2): 193-8.*

DAİ - PROKALSİTONİN

- 2 prospektif çalışma
- DAİ dahil bakteriyel infeksiyonların tanısında yararlı
- Cut- off değerleri: 17 mg/L , 0.08 ng/ ml
- ESH ve CRP 'ye kıyasla infeksiyonun klinik kanıtı ile daha doğru korelasyon

1.Jeandrot A, Richard JL, Combescure C, et al. Serum procalcitonin and C-reactive protein concentrations to distinguish mildly infected from non-infected diabetic foot ulcers: a pilot study. Diabetologia 2008; 51:347–52.

2. Uzun G, Solmazgul E, Curuksulu H, et al. Procalcitonin as a diagnostic aid in diabetic foot infections. Tohoku J Exp Med 2007; 213:305–12.

Osteomyelit Tedavisi

600 hasta, tek başına en az 3 aylık antibiyotik tedavisi (genellikle florokinolon)

Hastaların %60'ında osteomyelit remisyonu sağlanmış. *

**113diyabetik ayak osteomyelitli hastayı içeren prospektif bir çalışmada;

Hastaların % 82'sinde cerrahi uygulanmadan antibiyotik tedavisi ile remisyom sağlanabilmiş.

Yine de çoğu otorite nekrotik kemiği uzaklaştırmanın en iyi yöntem olduğuna inanmakta.

Hastanın cerrahi kabul etmediği durumlarda antibiyotik tedavisi denenebilir.

***Kronik osteomyelitte seçilecek antibiyotik tedavisi ile ilgili kanıta dayalı bilgilerimiz oldukça sınırlı. (86).

Tedavi Süresi?

İnfekte kemik cerrahi olarak çıkarıldı ise kısa süreli tedavi (1 hafta ?)

İnfekte kemik yerinde duruyorsa uzun süreli tedavi (4-6 hafta).

* **Jeffcoate WJ, Lipsky BA. Controversies in diagnosing and managing osteomyelitis of the foot in diabetes. *Clin Infect Dis.* 2004; 39 (Suppl 2): S115-22.**
** **Game FL, Jeffcoate WJ. Primarily non-surgical management of osteomyelitis of the foot in diabetes. *Diabetologia. 2008; 51(6): 962-7.***
*** **Lazzarini L, Lipsky BA, Mader JT. Antibiotic treatment of osteomyelitis: what have we learned from 30 years of clinical trials? *Int J Infect Dis. 2005; 9(3): 127-38.***

- **TEŞEKKÜR EDERİM.**