ULUSAL DAHİLİ VE CERRAHİ BİLİMLER YOĞUN BAKIM KONGRESİ

16- 20 Kasım 2011 / Swissotel, Ankara

İnteraktif olgu sunumu

Prof Dr Fatih AĞALAR

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KATILIMCI PROFİLİ

1. Genel Cerrahi uzmanı
2. Dahiliye uzmanı
3. Anestezi ve Reanimasyon uzmanı
4. Enfeksiyon hastalıkları uzmanı
5. Araştırma görevlisi
6. Diğer

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Olgu: 52 y, erkek

- Şikayet: Sırta vuran karın ağrısı, genel durum kötülüğü
- Dış merkezde 2 ay önce başarısız gastroskopi, özefagusta darlık
- Pilor stenozu tanısı var
- Halen hastanede Ürolojide yatarken konsülte edildi

FM

- Yüksek ateş
- Taşikardi
- Takipne
- Bilinç bulanıklığı var

- Karın muayenesi: Ayrıntılı bilgi vermiyor

Lab

- BK:11.700 mm^3
- HB: 17.5 gr/dl
- CRP: 80 mg/lt
- KCFT: hafif yüksek
- Üre:35.6
- Kreatinin:1.1 gr/Dl
- LDH:1231 İU/L
- **APACHE II>10**

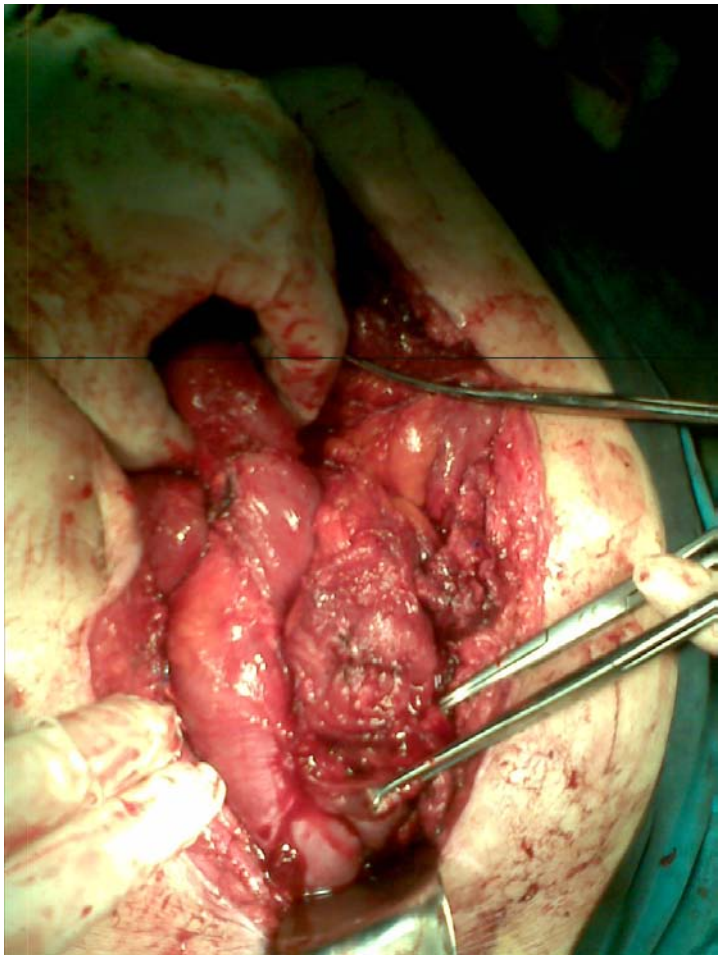
Görüntüleme

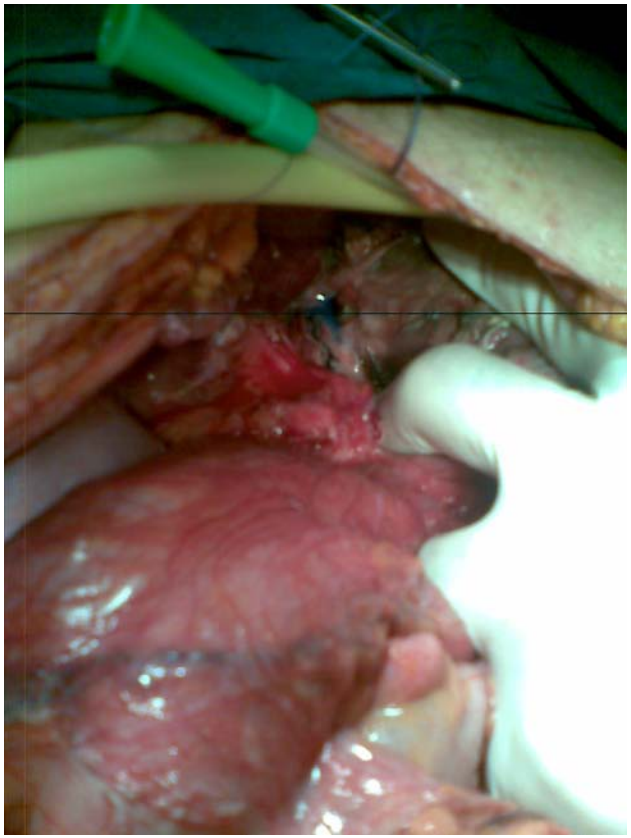
- Direkt grafi: Diayafram altında serbest hava
- USG: Karında serbest sıvı

Ameliyat tanısı

- Pilor stenozu
- Prepilorik antrumda ülser perforasyonu
- Gastroözefageal bileşkenin üst kısmında özefagus delinmesine ilişkin, 10x10 cm çapında abse kolleksiyonu

Peritonit, Üst Gastrointestinal sistemde perforasyon





- Alt özefageal bölgede gastroskopiye ilişkin perforasyon,
- Abse

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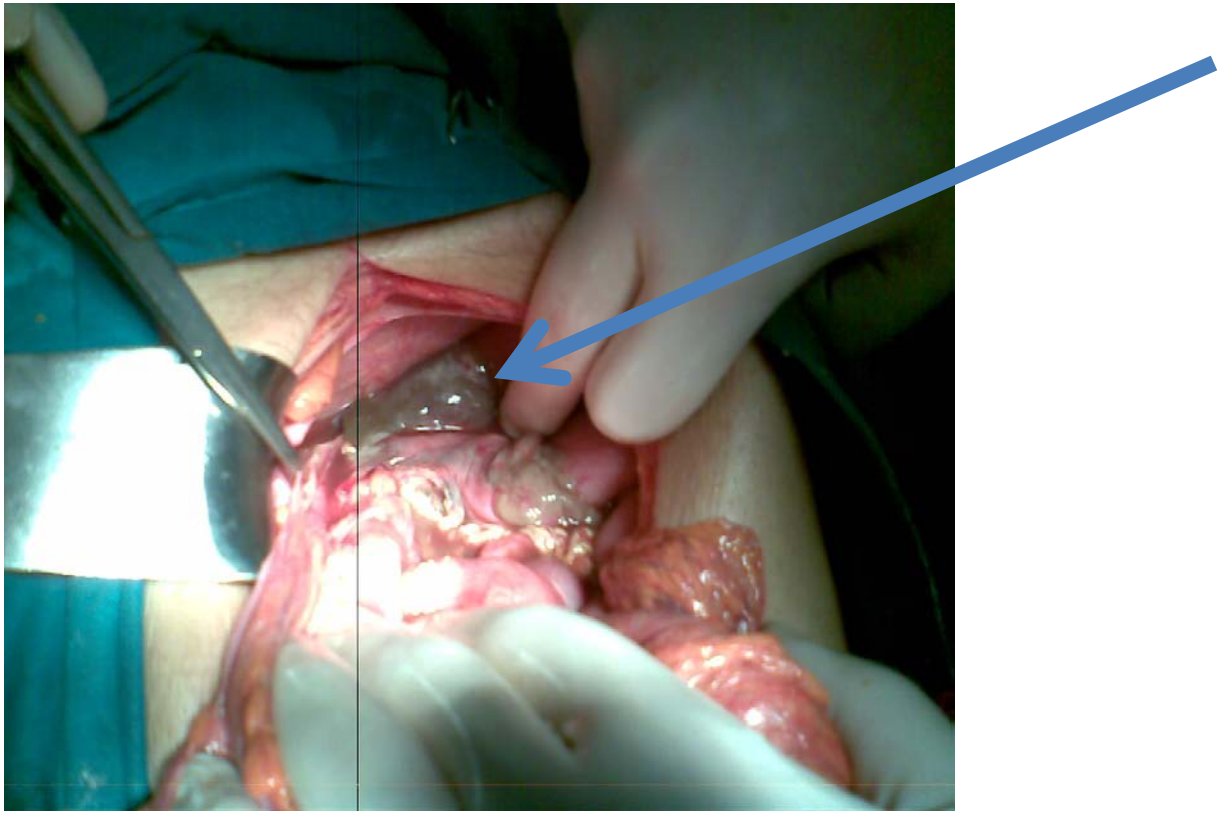
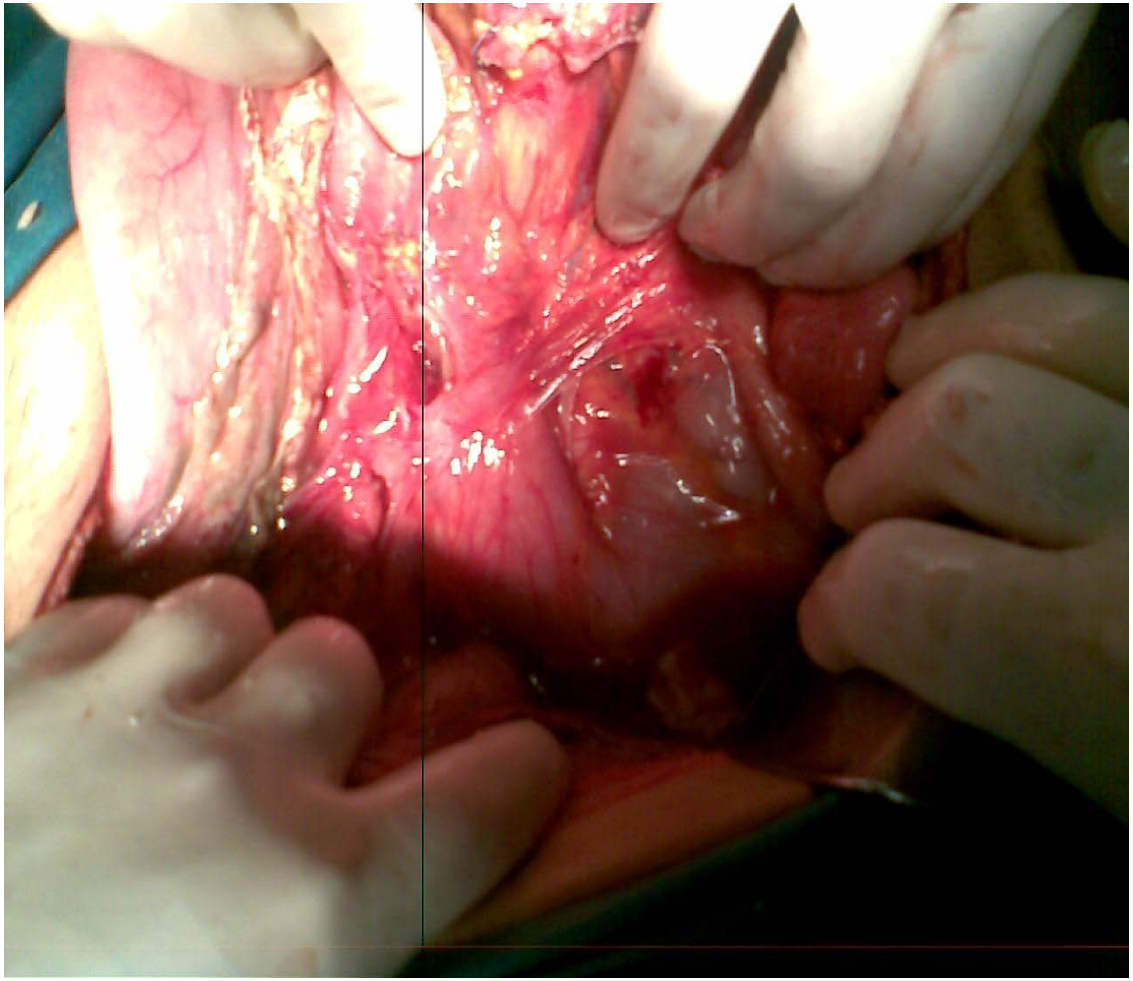
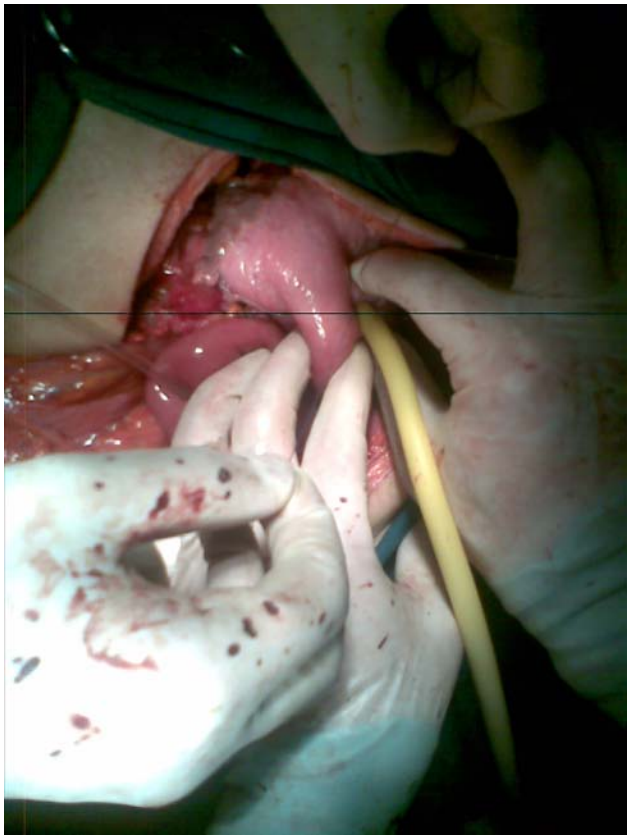
Supra mezokolik alanda peritonit



Ne yazık ki,
aynı zamanda aynı hastada hem pilor stenozu,
hemde prepilorik antrumda perforasyon vardı



Kaynak kontrolü

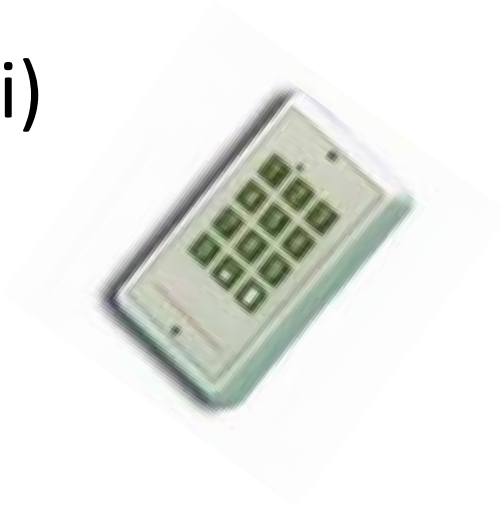


- Hemigastrektomi + abse drenajı + peritoneal tuvalet + STAR ile kaynak kontrolü sağlandı

Hasta entübe tutuluyor

Beslenmesini nasıl sağlayalım ?

1. IV parenteral
2. Nazogastrik yol
3. Enteral (nazo jejunal- jejunostomi)
4. IV+ enteral beslenme



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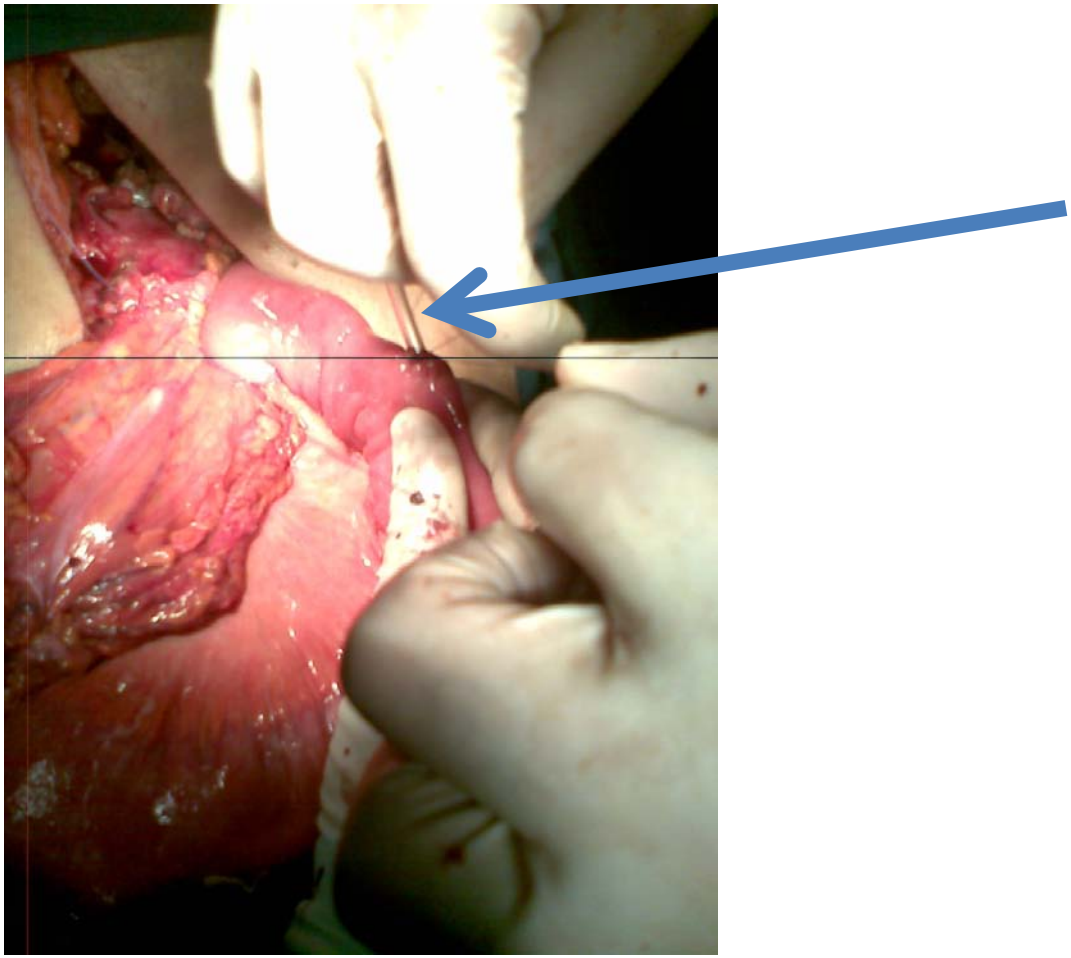
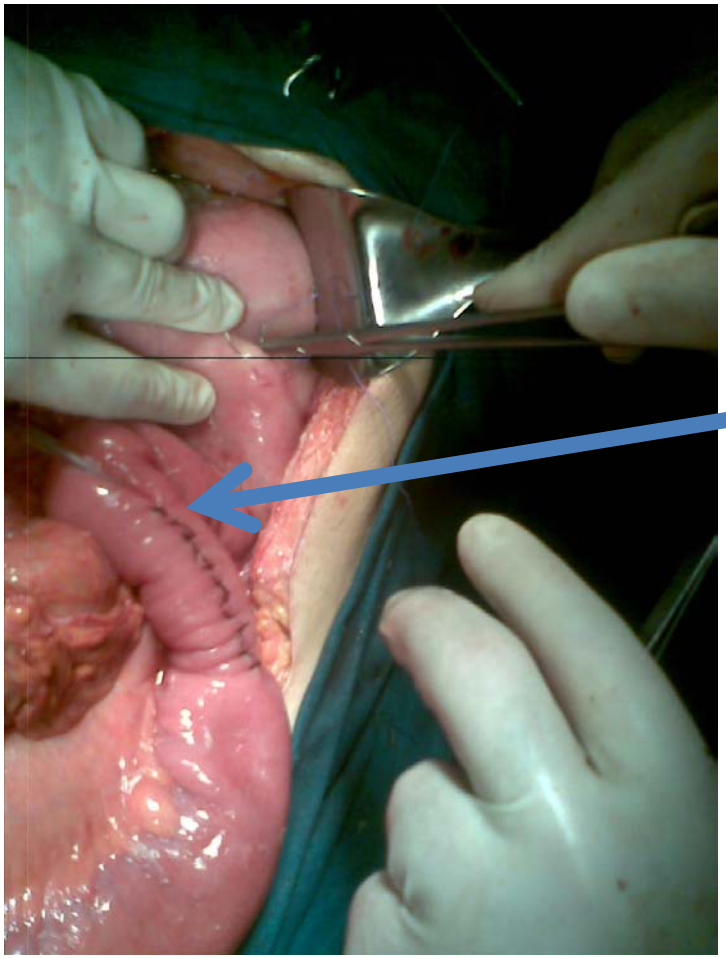
Beslenme jejunostomisi

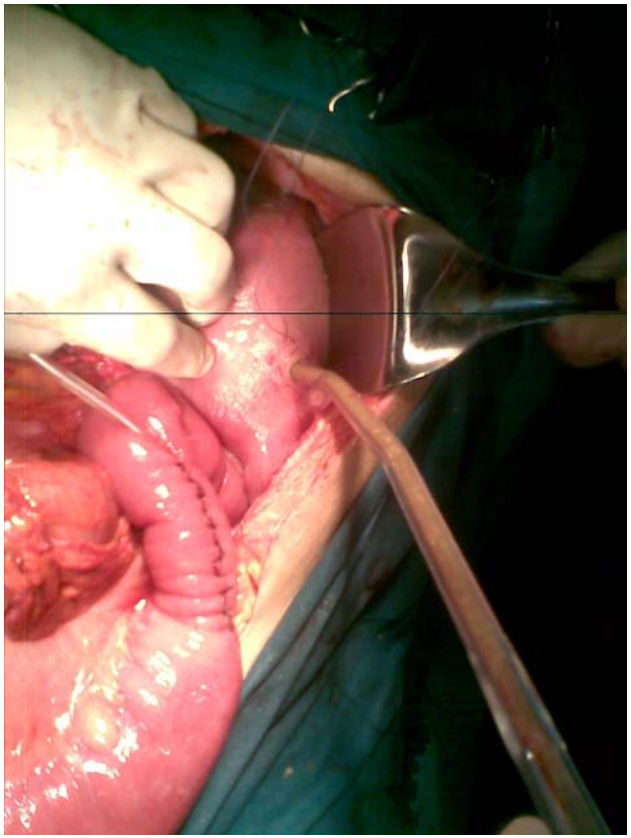


Beslenme jejunostomisi



- Santral kateter yolu ile parenteral nutriyondan mümkün olduğunca kaçınmak için beslenme jejunostomisi kullanırız.

Gastrostomi



- Nazogastrik yolu ile gastro intestinal sistem florasının farinkste kolonize olmasını önlemek için,

  Kritik hastalarda nazogastrik sonda yerine Gastrostomi kullanırız.
- Gastrostomiden besleme YAPMAYIZ ! ! !

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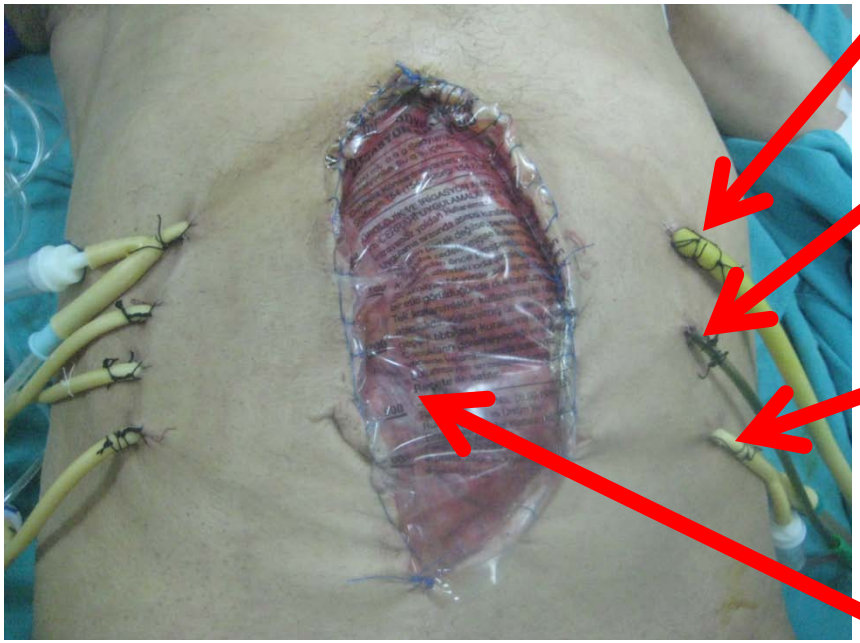

Gastrostomi

Jejunostomi

Karın drenleri

BOGOTA torbası

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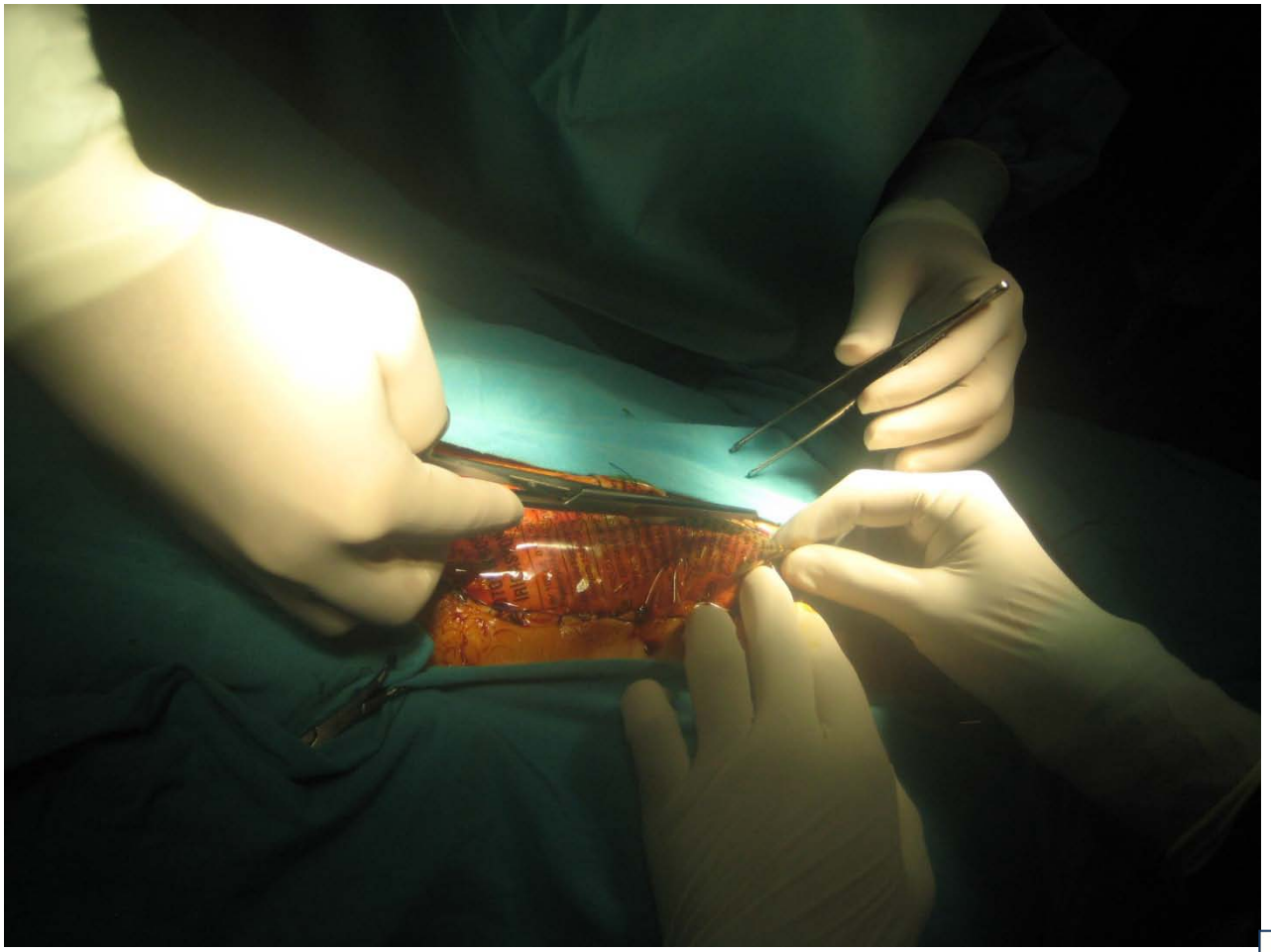
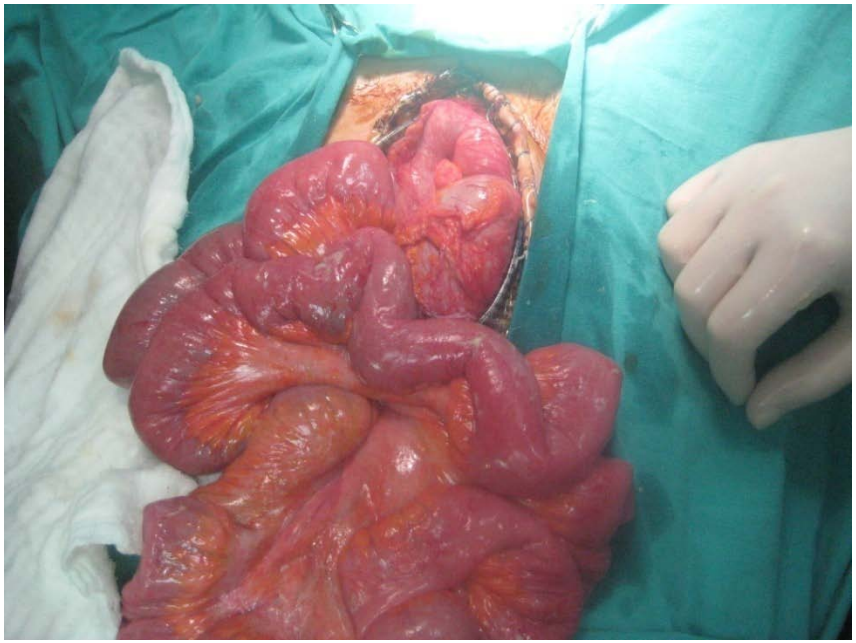
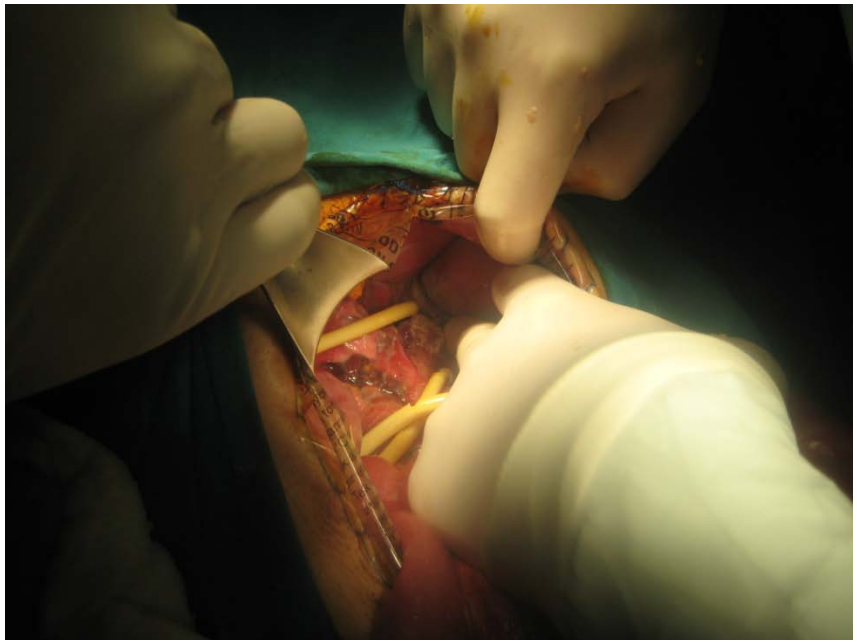
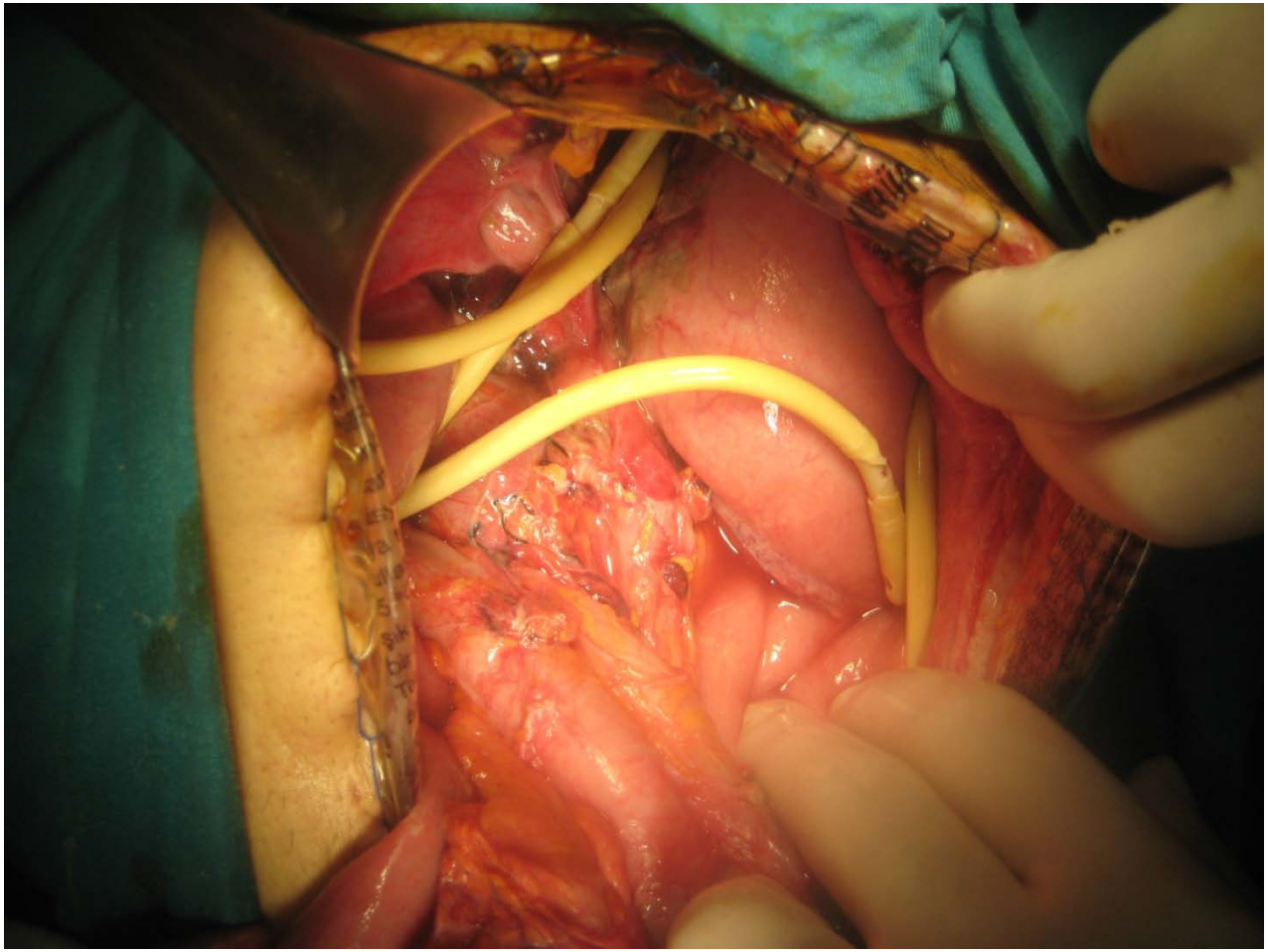
Kaynak kontrolünün devamı için relaparotomi



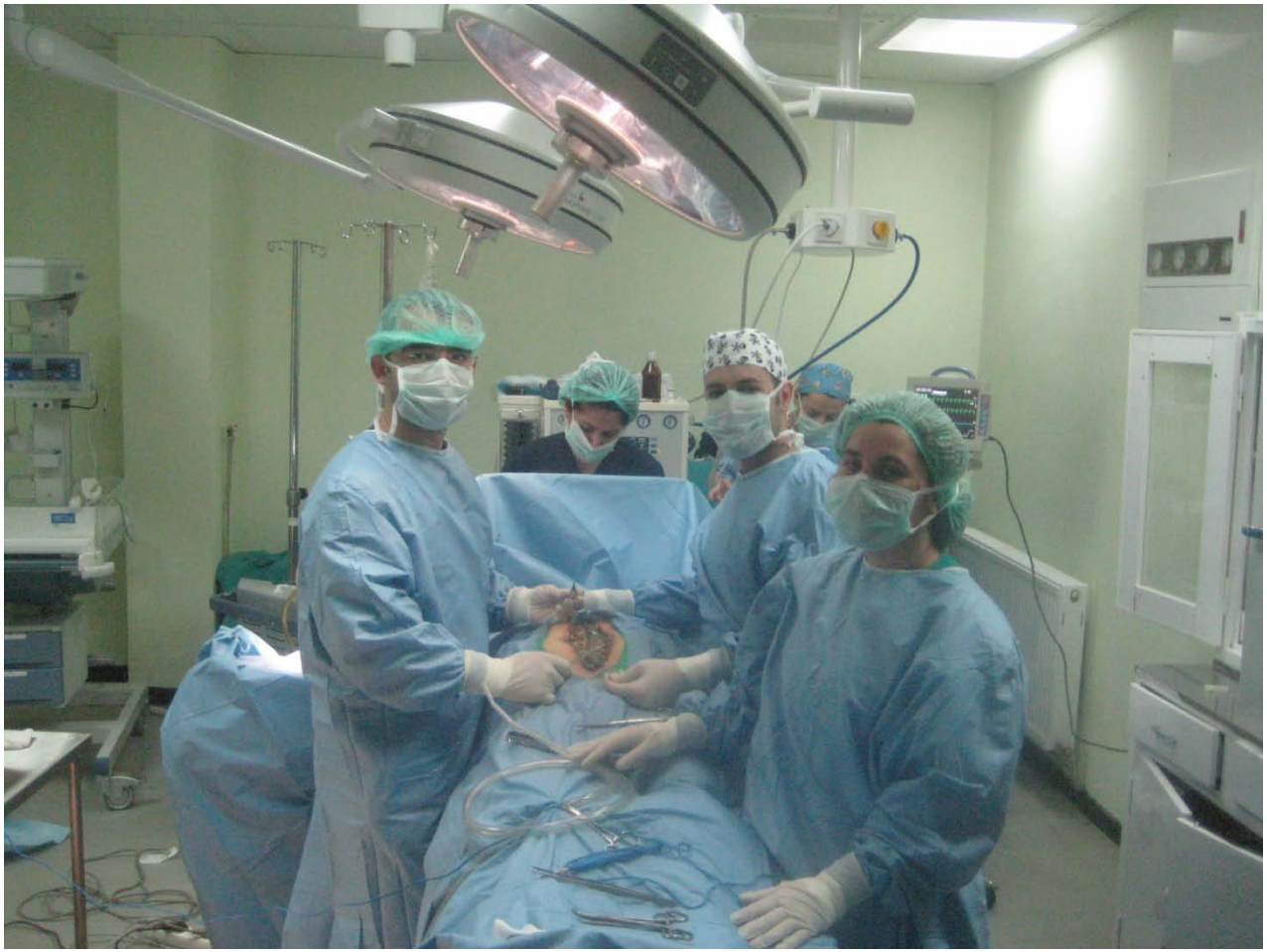
Karın relaparotomilerde temiz görünüyordu







Sterilizasyona ne kadar çok dikkat ederseniz edin



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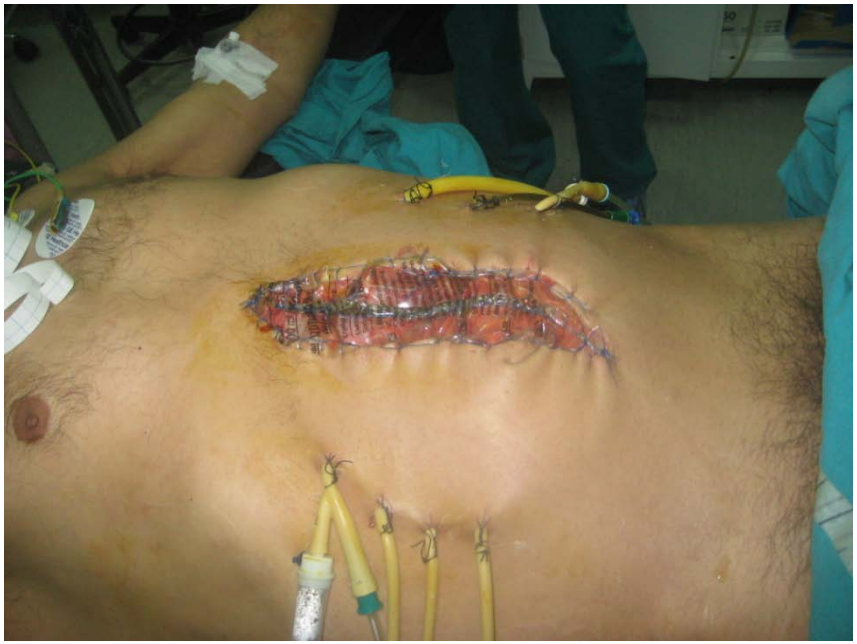
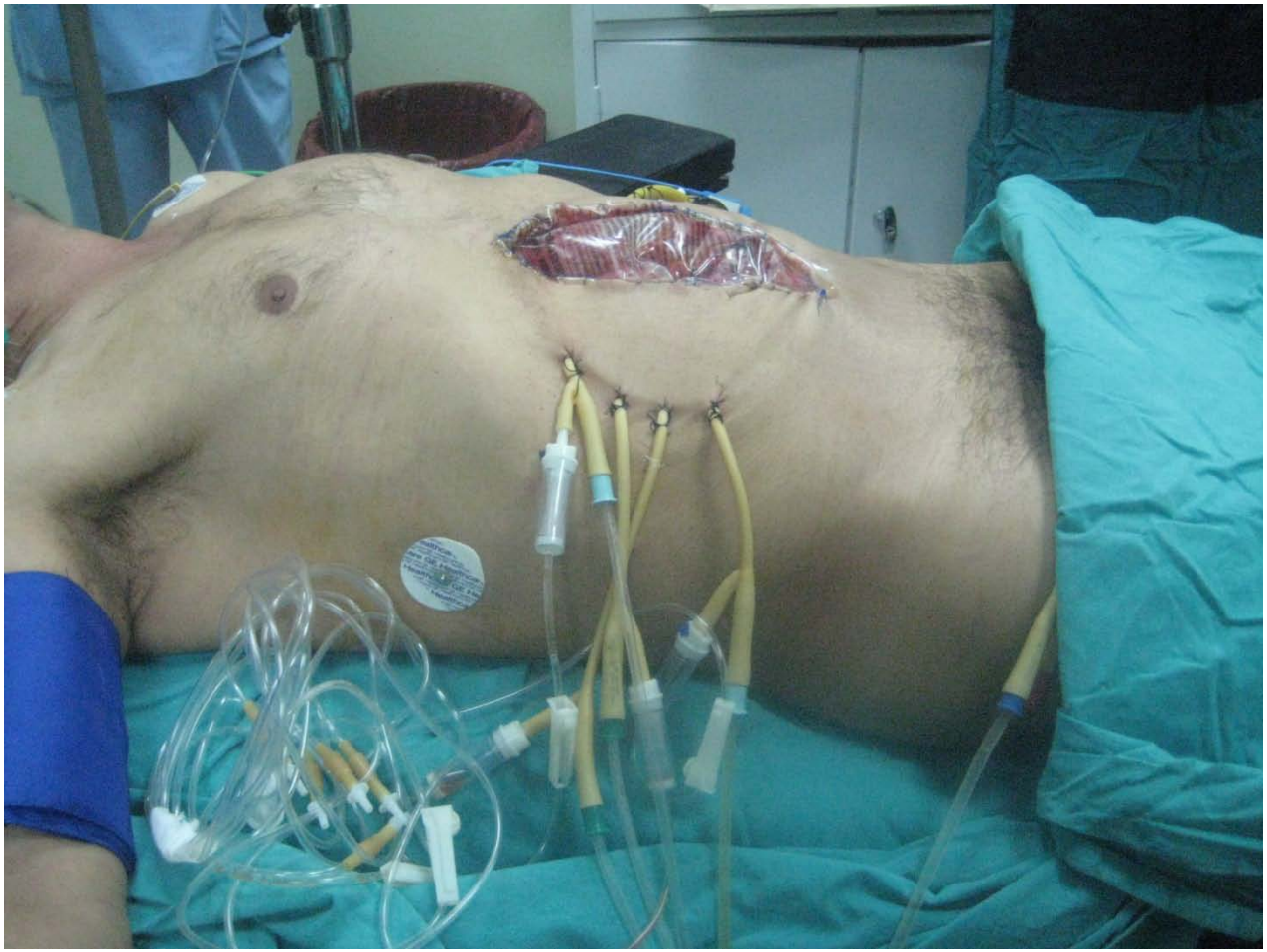


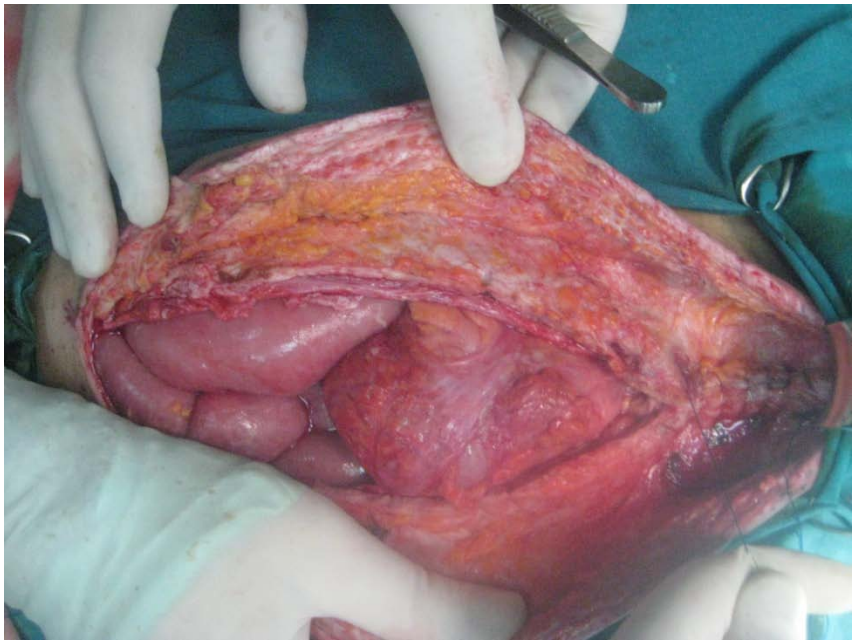
Relaparatomiler sırasında konak savunma mekanizmasının en önemli kaleleri yıkılmıştır.

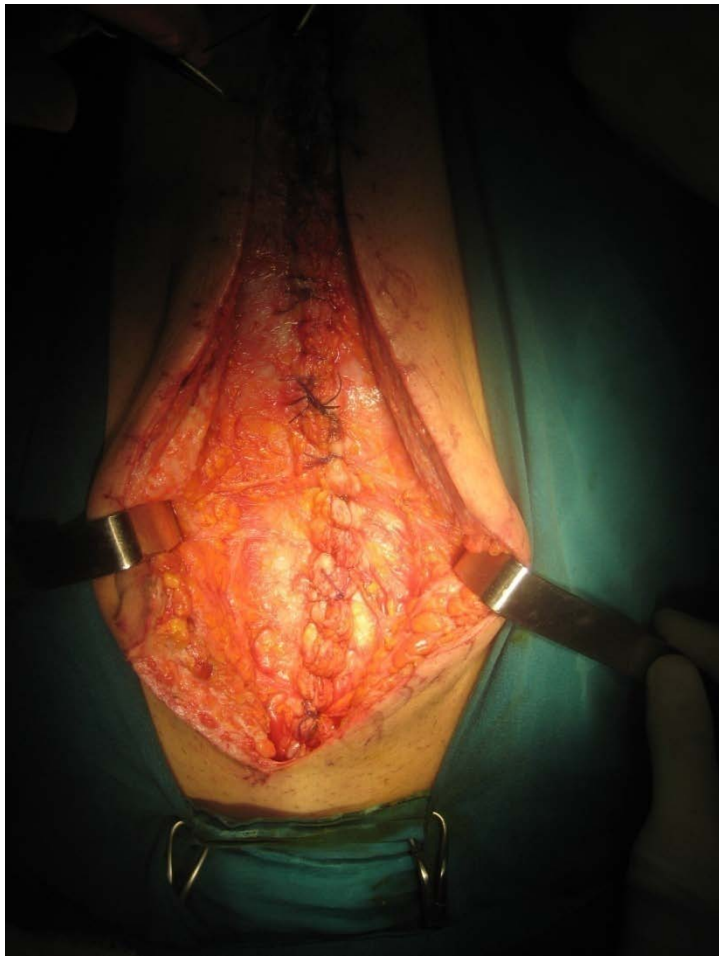
- Bütünlüğü bozulmuş deri
- Karın
  - Peritoneal resident makrofaj
  - IL -8, IL-1, Opsonin, Lizozim yıkama ile dışarı alınır
- Ne kadar seans, o kadar çok entübasyon
  - Glottik savunma yok olur

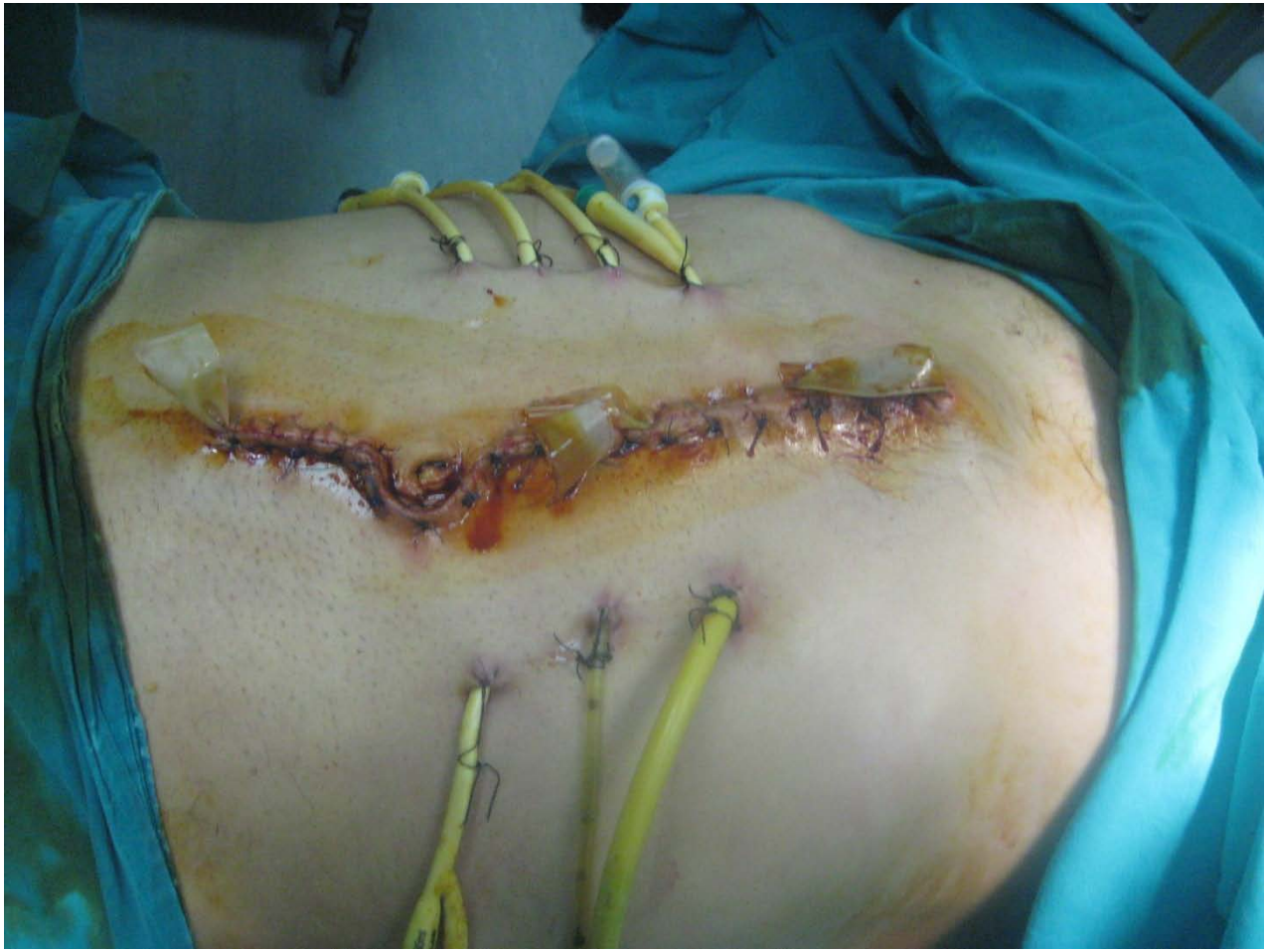
Ortam polimikrobiyal pazar yeri gibi olabilir



8. Gün karın temizdi







Hastaya ne oldu ?

tedavi

Ne verirdiniz ?

Ne verirdiniz ?

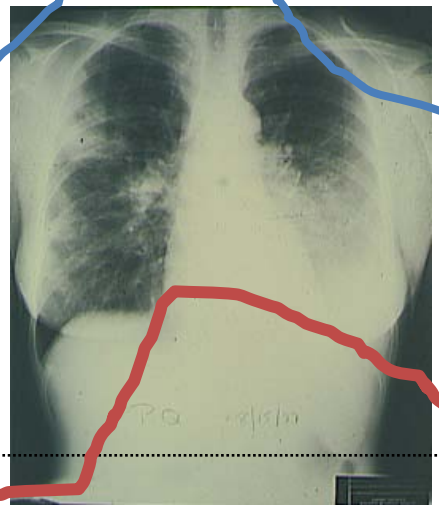
Mediasten dreni ve trakeal aspirattan= **C albicans,** Diğer alanlardan alınan kültürlerde üreme yok

Mediastinit ? Mediastinal abse + Peritonit, antral perforasyon + Özefageal perforasyon

ateş

4
1
40
39
38
37
36

Kaynak kontrol edildi



120
0

Karbapenem+Glikopeptit

90

Nabız sayısı

70

Asinetobakter

Flukanozol IV

0 1 2 7 14 21 28 35 42 49 56 63

Yoğun bakım yatış günü ------->

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Crit Care Med, 2006 , Montravers P et all

Candida as a risk factor for mortality in peritonitis*

Philippe Montravers, MD, PhD; Hervé Dupont, MD, PhD; Remy Gauzit, MD; Benoit Veber, MD; Christian Auboyer, MD; Patrick Blin, MD, MSc; Christophe Hennequin, MD, PhD; Claude Martin, MD

Objective: The clinical significance of *Candida* cultured from peritoneal fluid specimens remains a matter of debate. None of the studies that have addressed this issue have clearly distinguished between community-acquired peritonitis and nosocomial peritonitis. The current study tried to differentiate the pathogenic role of *Candida* in these two clinical settings and assess its importance on outcome.

Design: A multiple-center, retrospective, case-control study was conducted in intensive care unit patients. The interaction between mortality rates and type of patients was assessed. In the case of a significant interaction, a separate analysis of mortality and morbidity was planned.

Setting: Seventeen intensive care units in teaching and nonteaching hospitals.

Patients: Cases were patients operated on for peritonitis with *Candida* cultured from the peritoneal fluid, whereas controls were operated patients free from yeast. Cases and controls were matched for type of infection, Simplified Acute Physiology Score II, age, and time period of hospitalization.

Interventions: None.

Measurements and Main Results: The following characteristics were collected: demographic variables, underlying disease, severity score, site of infection, microbiological features, and antiinfective treatments. Survival was defined as the main outcome criterion and morbidity variables as secondary criteria. Odds ratios of mortality were calculated. Matching was achieved in 91 cases and 168 controls. Matching criteria, clinical characteristics, and mortality rate were not statistically different between cases and controls. A significant interaction was demonstrated between mortality rates and type of infection, leading to separate analysis of patients with community-acquired peritonitis and nosocomial peritonitis. The subgroup analysis demonstrated an increased mortality rate only in nosocomial peritonitis with fungal isolates (49% vs. 28% in controls, $p < .01$). Upper gastrointestinal tract site (odds ratio, 4.9; 95% confidence interval, 1.6–14.8) and isolation of *Candida* species (odds ratio, 3.0; 95% confidence interval, 1.3–6.7, $p < .001$) were found to be independent risk factors of mortality in nosocomial peritonitis patients.

Conclusions: Isolation of *Candida* species appears to be an independent risk factor of mortality in nosocomial peritonitis but not in community-acquired peritonitis. (Crit Care Med 2006; 34:646–652)

KEY WORDS: peritonitis; *Candida*; nosocomial infections; community-acquired infection; outcome; pathogenicity

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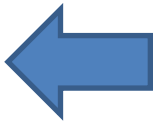
Crit Care Med, 2006 , Montravers P et al

Table 4. Univariate and multivariate analysis with regard to deaths of patients with nosocomial peritonitis (n = 164)

	Univariate Analysis		Multivariate Analysis	
Risk Factors	Odds Ratio (95% CI)	p Value	Adjusted Odds Ratio (95% CI)	p Value
Case group	2.4 (1.2–4.6)	.01	3.0 (1.3–6.7)	.009
Upper gastrointestinal tract site	2.1 (1.1–4.1)	.02	4.9 (1.6–14.8)	.005
Empirical antifungal treatment	1.9 (0.9–3.9)	.07	—	—
Inappropriate empirical antibiotic treatment	2.2 (1.1–4.3)	.02	1.6 (0.6–4.3)	.3

CI, confidence interval.



Kandida nazokomiyal peritonitte mortalite için bağımsız bir risk faktörüdür

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The Epidemiology of *Candida* Colonization and Invasive Candidiasis in a Surgical Intensive Care Unit Where Fluconazole Prophylaxis is Utilized

Follow-Up to a Randomized Clinical Trial

Shelley S. Magill, MD, PhD,† Sandra M. Swoboda, RN, MS,‡ Christine E. Shields, BS,† Elizabeth A. Colantuoni, MS,§ Annette W. Fothergill, MA, MBA,¶ William G. Merz, PhD,† Pamela A. Lipsett, MD,‡ and Craig W. Hendrix, MD**

Objective: To determine whether *Candida glabrata* colonization and invasive candidiasis (IC) increased among critically ill surgical patients 3 years after the introduction of fluconazole prophylaxis to a surgical intensive care unit (SICU).

Summary Background Data: Fluconazole prophylaxis has been shown in randomized clinical trials to reduce the occurrence of candidiasis in some

Conclusions: There was no increase in *C. glabrata* colonization or in the proportion of IC due to *C. glabrata* after a 3-year period of routine fluconazole prophylaxis for selected SICU patients.

(*Ann Surg* 2009;249: 657–665)

Ann Surg, 2009, 249, Magill S et al

Profilaktik Flukanazol C Glabrata kolonizasyon ve IC oranlarında değişikliğe neden olmaz

olonization Among
003 Cohort as Compared
8 Trial

Colonizing			
Any *Candida* spp., n (%)			
At least 2 colonizing fungal spp., n (%)			
C. albicans, n (%)	47 (46)		
C. glabrata, n (%)	30 (29)	34 (28)	
C. tropicalis, n (%)	8 (7.8)	13 (11)	0.45
C. parapsilosis, n (%)	7 (6.8)	8 (6.6)	0.96
C. krusei, n (%)	5 (4.9)	0	0.02†
Other *Candida* spp., n (%)	4 (3.9)	0	0.04†

*χ^2 test unless otherwise indicated.
†Fisher exact test.

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Ann Surg, 2009, 249, Magill S et al

TABLE 4. Odds of *C. glabrata* Colonization Detected by Initial ICU Surveillance Cultures* for Patients in the 2003 Cohort as Compared With Patients in the 1998 Trial, Adjusted for Propensity Score Quintile

Anatomic Site	2003 Cohort, n = 235	1998 Trial, n = 247	OR (95% CI)
Rectal/Fecal, n (%)			
Total cultured	218 (93)	243 (98)	
Colonized with *C. glabrata*	41 (19)	51 (21)	0.87 (0.49–1.51)
Urine, n (%)			
Total cultured	230 (98)	242 (98)	
Colonized with *C. glabrata*	6 (2.6)	13 (5.4)	0.62 (0.20–1.92)
Endotracheal aspirate, n (%)			
Total cultured	106 (45)	196 (79)	
Colonized with *C. glabrata*	19 (18)	26 (13)	1.55 (0.78–3.09)

*Includes all patients who had one or more surveillance cultures performed from a given anatomical site.

The value of CRP, IL-6, leptin, cortisol, and peritoneal caspase-3 monitoring in the operative strategy of secondary peritonitis Agalar F et al. Turkish Journal of Trauma & Emergency Surgery, 2011

N:15 sekonder peritonit, APACHE II>10
STAR
Ampirik karbapenem

- Mortalite= APACHE II çok yüksek
  n:4 (3 olguda KKI=0.4)
- KKI n:3 (KKI=0.4),
  n:3 (KKI<0.4)
- Kullanılan ajan: Flukanazol

- APACHE II çok yüksekse ilacın tipi gidişi değiştirmiyor

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teşekkür ederim

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