Hızlı Ölümcül Enfeksiyonlar

Uzm.Dr. Müge Günalp
AÜTF Acil Tıp AD

Vaka

- ✓ 40 yaşında erkek
- ✓ Ateş:39˚C,TA:60/nabız,Kalp hızı:140/dak
- ✓ Multilober pnömoni

- ✓ Tedavi?
- ✓ İmmün yetmezlik (+)

- ✓ Hızlı ölümcül enfeksiyon??????

Vaka

- ✓ 72 yaşında erkek hasta
- ✓ Ateş:39˚C,TA:90/60 mmHg,Kalp hızı:104/dak
- ✓ DM (+),demans (+)
- ✓ Sol bacak ve ayak dorsolateralde eritem ve palpasyonda krepitasyon

- ✓ Tedavi???

- ✓ Hızlı ölümcül enfeksiyon??????

Vaka

- ✓ 63 yaşında erkek hasta
- ✓ Sol yüz yarısında uyuşma ve ağrı
- ✓ Sol gözde şişlik,görme kaybı,sol göz kapağında düşme
- ✓ İTP,Steroid induced DM
- ✓ Ateş: 36,6 °C,Nabız: 92/dak.,TA:120/80 mmHg

- ✓ Tedavi???

- ✓ Hızlı ölümcül enfeksiyon??????

Sepsis

- ✓ SCCM (Society of Critical Care Medicine)
- ✓ ESICM (European Society of Intensive Care Medicine)
- ✓ ACCP (American College of Chest Physicians)

2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference

Mitchell M. Levy, MD, FCCP; Mitchell P. Fink, MD, FCCP; John C. Marshall, MD; Edward Abraham, MD; Derek Angus, MD, MPH, FCCP; Deborah Cook, MD, FCCP; Jonathan Cohen, MD; Steven M. Opal, MD; Jean-Louis Vincent, MD, FCCP, PhD; Graham Ramsay, MD; For the International Sepsis Definitions Conference

Objective: In 1991, the American College of Chest Physicians (ACCP) and the Society of Critical Care Medicine (SCCM) convened a "Consensus Conference," the goals of which were "to provide a conceptual and a practical framework to define the systemic inflammatory response to infection, which is a progressive injurious process that falls under the generalized term 'sepsis' and includes sepsis-associated organ dysfunction as well." The general definitions introduced as a result of that conference have been widely used in practice and have served as the foundation for inclusion criteria for numerous clinical trials of therapeutic interventions. Nevertheless, there has been an impetus from experts in the field to modify these definitions to reflect our current understanding of the pathophysiology of these syndromes.

Design: Several North American and European intensive care societies agreed to revisit the definitions for sepsis and related conditions. This conference was sponsored by the SCCM, The European Society of Intensive Care Medicine (ESICM), The American College of Chest Physicians (ACCP), the American Thoracic Society (ATS), and the Surgical Infection Society (SIS).

Methods: The conference was attended by 29 participants from Europe and North America. In advance of the conference, five subgroups were formed to evaluate the following areas: signs and symptoms of sepsis, cell markers, cytokines, microbiologic data, and coagulation parameters. The subgroups corresponded electronically before the conference and met in person during the conference. A spokesperson for each group presented the deliberation of each group to all conference participants during a plenary session. A writing committee was formed at the conference and developed the current article based on executive summary documents generated by each group and the plenary group presentations. The present article serves as the final report of the 2001 International Sepsis Definitions Conference.

Conclusion: This document reflects a process whereby a group of experts and opinion leaders revisited the 1992 sepsis guidelines and found that apart from expanding the list of signs and symptoms of sepsis to reflect clinical bedside experience, no evidence exists to support a change to the definitions. This lack of evidence serves to underscore the challenge still present in diagnosing sepsis in 2003 for clinicians and researchers and also provides the basis for introducing PIRO as a hypothesis-generating model for future research. (Crit Care Med 2003; 31:1250–1256)

KEY WORDS: sepsis; severe sepsis; septic shock; systemic inflammatory response syndrome; PIRO

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SIRS

Organizmanın hasar veren olaylara karşı gösterdiği sistemik inflamatuar yanıt:

-Vücut ısısı > 38ºC veya < 35ºC

-Kalp hızı >90 dak

-Solunum hızı >20 dak

-paCO2 < 32 mmHg veya mekanik ventilatör ihtiyacı

-BK > 12000/mm³ veya <4000/mm³ veya > % 10 immatur form

SEPSİS

SIRS ve dokümante edilmiş enfeksiyon bulguları

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CİDDİ SEPSİS

Sepsis + ≥1 organ sistemi disfonksiyonu:

- -Kardiyovasküler (hipotansiyon:SKB <90 mmHg, >40 mmHg düşme)
- -Renal (oligüri, ATN)
- -Akciğer (ARDS)
- -Hepatik
- -Koagülasyon sistemi (DİK, trombositopeni)
- -SSS (ensefalopati)
- -Açıklanamayan metabolik asidoz (Laktat > 2 mmol/L)
- -GİS (ileus)

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SEPTİK ŞOK

Ciddi sepsis bulguları;

-Ortalama arteryel kan basıncı 40-60 ml/kg SF sonrası <60 mmHg

-Ortalama arteryel kan basıncını 60 mmHg↑de tutmak için 5µg/kg/dak dopamin ihtiyacı veya 0.25 µg/kg/dak adrenalin ihtiyacı

Sepsis

- ✓ İnsidans 100.000 vakada 50-95
- ✓ Her yıl % 9 oranında↑
- ✓ En sık 6. dekad
- ✓ Mortalite %15-40

Sepsis

✓ Etyoloji

- Gram (+) sepsis %35-50
  - Metisilin duyarlı Staf aureus
  - MRSA
  - Strep. Pneumonia
- Gram (-) sepsis %25-30
  - E. Coli
  - Pseudomonas
  - Klebsiella

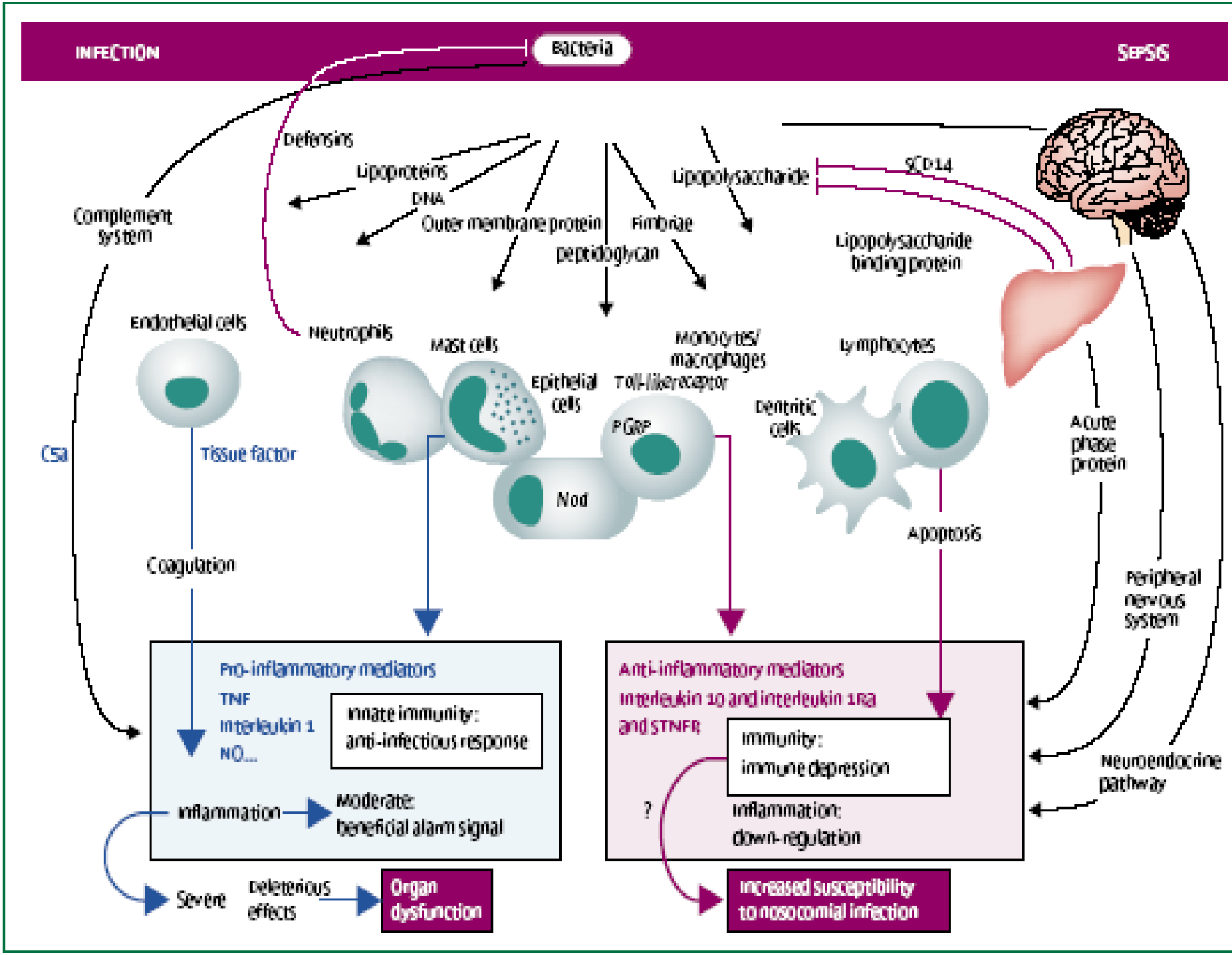
Sepsis Patofizyolojisi

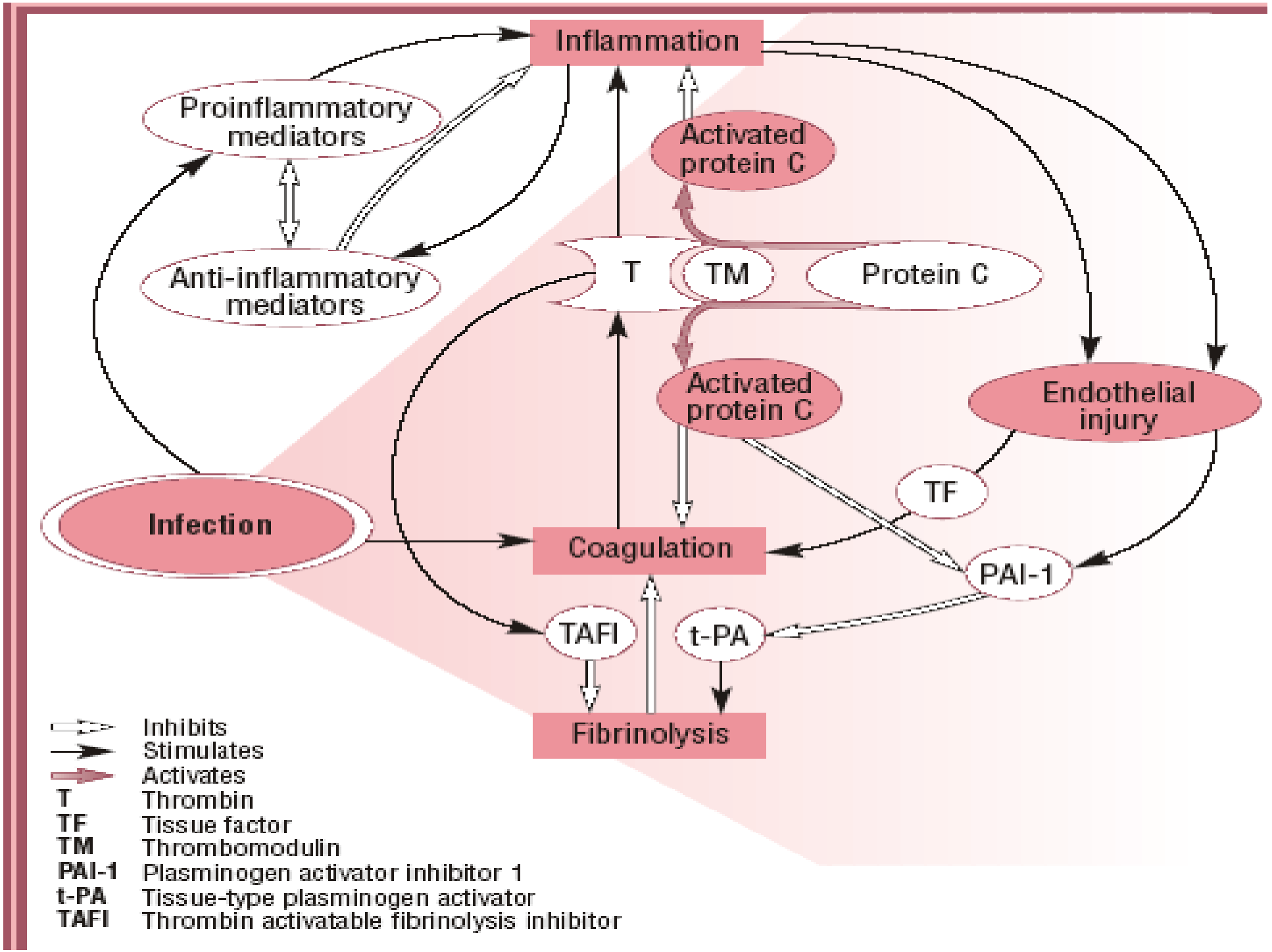
Endotel Disfonksiyonu & Mikrovasküler Tromboz

Hipoperfüzyon & İskemi

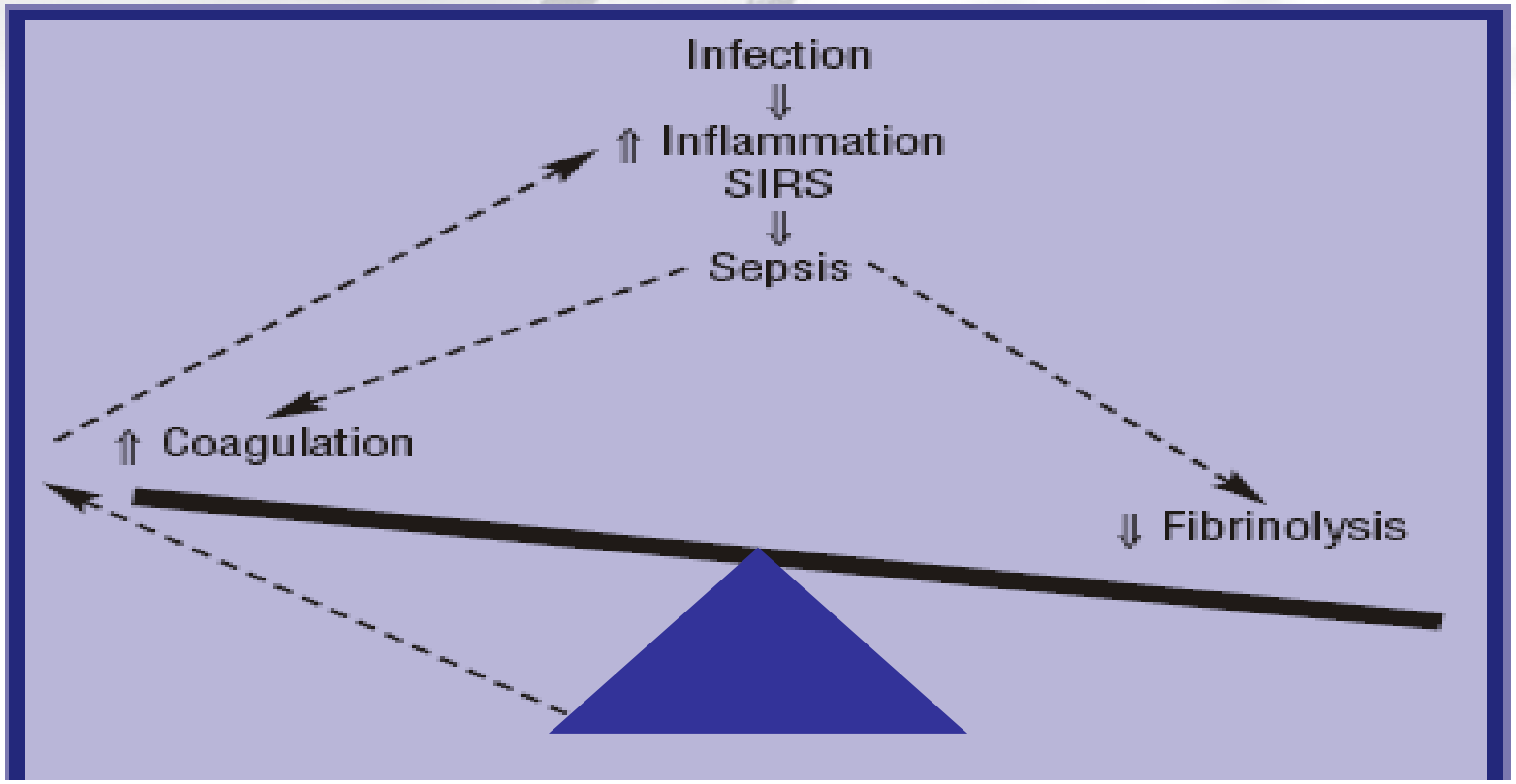
Akut Organ Disfonksiyonu (Ağır Sepsis)

Ölüm


INFECTION
Bacteria
SEPSIS
Defensins
Lipoproteins
DNA
Outer membrane protein
Fimbriae
peptidoglycan
Lipopolysaccharide
sCD14
Lipopolysaccharide binding protein
Complement system
Endothelial cells
Neutrophils
Mast cells
Epithelial cells
Monocytes/ macrophages
Toll-like receptor
PGRP
Nod
Lymphocytes
Dentritic cells
Apoptosis
Acute phase protein
CSa
Tissue factor
Coagulation
Peripheral nervous system
Neuroendocrine pathway
Pro-inflammatory mediators
TNF
Interleukin 1
NO...
Innate immunity: anti-infectious response
Inflammation
Moderate: beneficial alarm signal
Severe
Deleterious effects
Organ dysfunction
Anti-inflammatory mediators
Interleukin 10 and interleukin 1RA and STNFR
Immunity: immune depression
?
Inflammation: down-regulation
Increased susceptibility to nosocomial infection


Inflammation
Proinflammatory mediators
Anti-inflammatory mediators
Activated protein C
T
TM
Protein C
Activated protein C
Endothelial injury
TF
Infection
Coagulation
PAI-1
TAFI
t-PA
Fibrinolysis
Inhibits
Stimulates
Activates
T Thrombin
TF Tissue factor
TM Thrombomodulin
PAI-1 Plasminogen activator inhibitor 1
t-PA Tissue-type plasminogen activator
TAFI Thrombin activatable fibrinolysis inhibitor

Sepsis Patofizyolojisi



Sepsis Patofizyolojisi

Sistemik Koagülasyon

Damariçi fibrin oluşumu

↓

Küçük ve orta çaplı damarlarda tromboz

↓

MOF

Trombosit ve koagülasyon faktörlerinin tüketimi
"TÜKETİM KOAGÜLOPATİSİ"

↓

Kanama

Tanı

- ✓ SIRS belirti ve bulguları olan her hasta aday!!!
- ✓ Sepsisin klasik belirti ve bulguları
- ✓ İnfeksiyona bağlı inflamasyon belirleyicileri
  - CRP *(CRP>50 mg/L %98.5 duyarlı, %75 özgün.Povoa et al. Inten Care Med 1998;24:1052).*
  - Prokalsitonin *(PCT >1.5 ng/ml %100 duyarlı, %72 özgün.deWerra et al. Crit Care Med 1997;25:607)*

Monitörizasyon

- ✓ Vital bulgular
  - -İnvaziv arteryel kan basıncı izlemi
  - -EKG monitörizasyonu
  - -Vücut ısısı izlemi (rektal)
  - -Solunum sayısı izlemi
  - -SpO_2 izlemi (pulse oksimete)
- ✓ Hemodinamik parametreler
  - -Santral venöz kateterizasyon
    - CVP
    - $ScvO_2$

Monitörizasyon

- ✓ Laboratuar testleri
  - Beyaz küre
  - Trombosit
  - Hematokrit
  - CRP
  - Prokalsitonin
  - Laktat
  - Saatlik glukoz
  - BFT
  - KCFT
  - DİK testleri
  - AKG
  - USG *(Crit Care Med 2007;35(5):235-249)*

Laktat

- ✓ O_2 ihtiyacının dolaylı göstergesi
- ✓ Başvuruda laktat>4mmol/L yüksek mortalite ile korele
- ✓ İlk 6 saatte laktat düzeyinin↑kalması mortalite ve morbidite ↑

Rivers EP CMAJ 2005;173(9):10541065

Baz defisit

- ✓ Genel doku perfüzyonu hakkında bilgi
- ✓ Hemorajik şok derecesi,transfüzyon ihtiyacı ve tedaviye cevap
- ✓ Laktat>baz defisit

Husain FA Am J Surg.2003;185(5)485-491

Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

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- ✓ SV basınç 8-12 mmHg
- ✓ Ort. arter basıncı >65 mmHg
- ✓ İdrar akımı ≥ 0.5 ml/kg/saat
- ✓ SV O2 satürasyonu >70 mmHg

BAŞLANGIÇ RESÜSİTASYONU

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- ✓ SV basınç 8-12 mmHg oluncaya kadar sıvı (kristaloid veya kolloid)
- ✓ Hipovolemi şüphesi varlığında 1000cc/30 dak
- ✓ Hemodinami iyilik sağlanıncaya kadar…
- ✓ SVP hedefe ulaştı ama hemodinami bozuk??
- ✓ Mikrosirkülasyon……

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Intensive Insulin Therapy and Pentastarch Resuscitation in Severe Sepsis

Frank M. Brunkhorst, M.D., Christoph Engel, M.D., Frank Bloos, M.D., Ph.D., Andreas Meier-Hellmann, M.D., Max Ragaller, M.D., Norbert Weiler, M.D., Onnen Moerer, M.D., Matthias Gruendling, M.D., Michael Oppert, M.D., Stefan Grond, M.D., Derk Olthoff, M.D., Ulrich Jaschinski, M.D., Stefan John, M.D., Rolf Rossaint, M.D., Tobias Welte, M.D., Martin Schaefer, M.D., Peter Kern, M.D., Evelyn Kuhnt, M.Sc., Michael Kiehntopf, M.D., Christiane Hartog, M.D., Charles Natanson, M.D., Markus Loeffler, M.D., Ph.D., and Konrad Reinhart, M.D., for the German Competence Network Sepsis (SepNet)

N ENGL J MED 358;2 WWW.NEJM.ORG JANUARY 10, 2008

CONCLUSIONS

The use of intensive insulin therapy placed critically ill patients with sepsis at increased risk for serious adverse events related to hypoglycemia. As used in this study, HES was harmful, and its toxicity increased with accumulating doses. (ClinicalTrials.gov number, NCT00135473.)

Dikkat!

- ✓ Hydroxyethyl starch ➡ sepsiste ABY riski↑
- ✓ Pentastarch kümülatif dozda renal replasman tedavisi ve 90 günlük mortalitede↑

Schortgen F, Lacherade JC, Bruneel F, et al:Effects of Hydroxyethyl starch and gelatin on renal function in severe sepsis: A multicentre randomised study. Lancet 2001; 357:911–916
Sakr Y, Payen D, Reinhart K, et al: Effects ofhydroxyethyl starch administration on renal function in critically ill patients. Br J Anaesth 2007; 98:216–224

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Comparison of Albumin and Saline for Fluid Resuscitation in the Intensive Care Unit

The SAFE Study Investigators*

Resüsitasyon için albümin

veya salin verilebilir.

BACKGROUN
It remains u
care units (IC
trial to comp
heterogeneo

METHODS
We random
4 percent alb
28 days. The
period after

RESULTS
Of the 6997 patients who underwent randomization, 3497 were assigned to receive albumin and 3500 to receive saline; the two groups had similar baseline characteristics. There were 726 deaths in the albumin group, as compared with 729 deaths in the saline group (relative risk of death, 0.99; 95 percent confidence interval, 0.91 to 1.09; P=0.87). The proportion of patients with new single-organ and multiple-organ failure was similar in the two groups (P=0.85). There were no significant differences between the groups in the mean (±SD) numbers of days spent in the ICU (6.5±6.6 in the albumin group and 6.2±6.2 in the saline group, P=0.44), days spent in the hospital (15.3±9.6 and 15.6±9.6, respectively; P=0.30), days of mechanical ventilation (4.5±6.1 and 4.3±5.7, respectively; P=0.74), or days of renal-replacement therapy (0.5±2.3 and 0.4±2.0, respectively; P=0.41).

CONCLUSIONS
In patients in the ICU, use of either 4 percent albumin or normal saline for fluid resuscitation results in similar outcomes at 28 days.

aline versus Albumin Fluid Evalua-
SAFE) Study is a collaboration of the
lian and New Zealand Intensive Care
y Clinical Trials Group, the Australian
ross Blood Service, and the George
ute for International Health. The writ-
mmittee (Simon Finfer, M.B., B.S.,
lo Bellomo, M.B., B.S., M.D., Neil
M.B., B.S., Ph.D., Julie French, R.N.,
Myburgh, M.B., B.Ch., Ph.D., and Ro-
orton, Ph.D., M.P.H.) takes respon-
for the content of this article. Address
t requests to Dr. Finfer at ANZICS
Level 3, 10 Ievers St., Carlton, VIC
Australia, or at ctg@anzics.com.au.

*The Saline versus Albumin Fluid Evaluation (SAFE) Study investigators are listed in the Appendix.

N Engl J Med 2004;350:2247-56.
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- ✓ MAP<65 mmHg vazopressör tedavi
- ✓ İlk seçenek dopamin veya norepinefrin
- ✓ Epinefrin,vazopressin ilk seçenek değil
- ✓ VASST çalışması norepinefrin ile norepinefrin+vazopressin
- ✓ Terlipressin????
- ✓ Renal doz dopamin…….
- ✓ Miyokard disfonksiyonu ve kardiyak indeks ↓→dobutamin

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- ✓ $Scvo_2$ veya SVO_2 < % 70 eritrosit süspansiyonu (Hct > % 70)
- ✓ Dobutamin infüzyonu

Septik şokta resüsitasyonun erken başlanması (Acil Servis'te) Erken hedefe yönelik tedavi

Rivers et al. NEJM 2001;345:1368

Erken hedefe yönelik tedavi

Early goal-directed therapy in severe sepsis and septic shock: a contemporary review of the literature

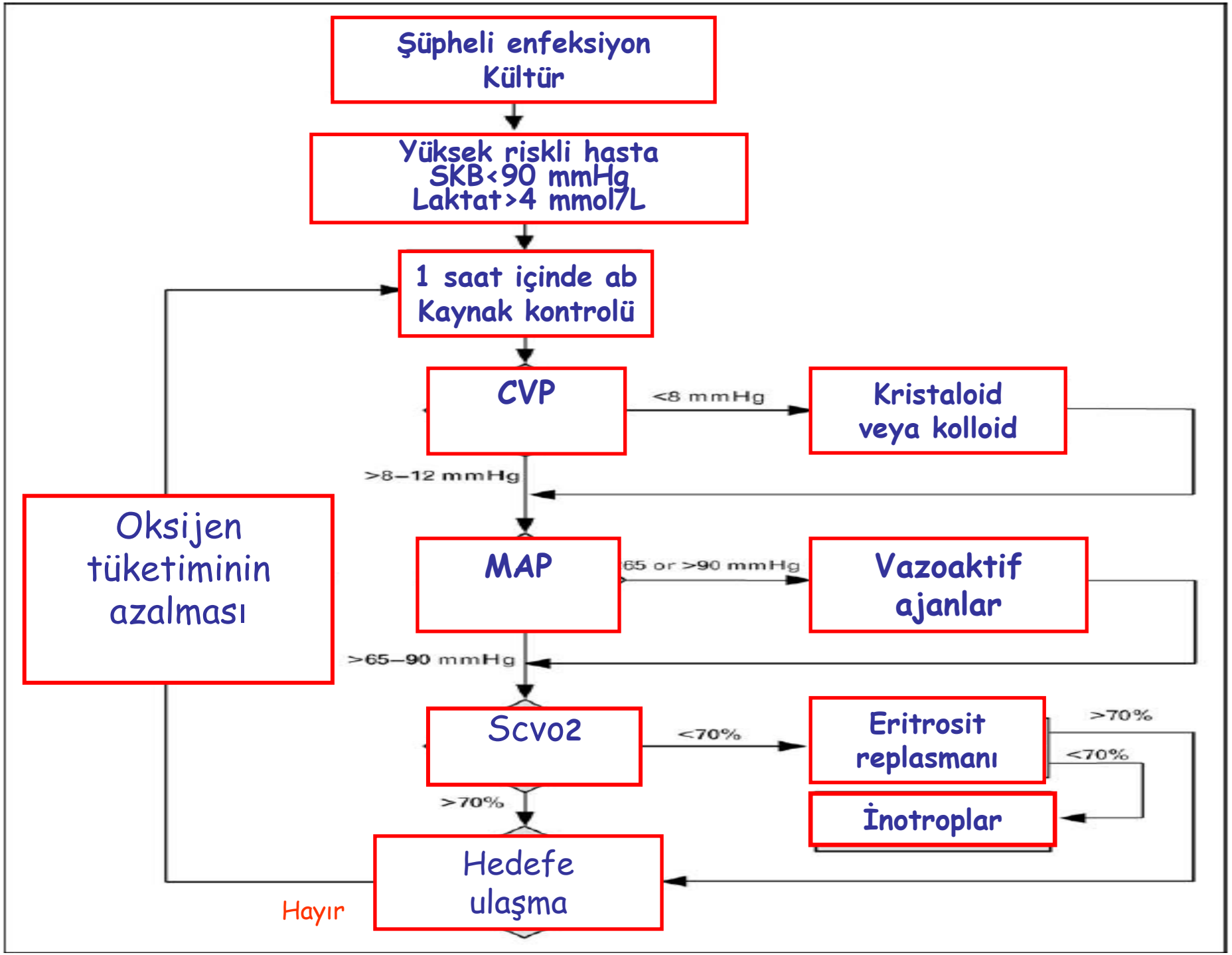
Emanuel P. Rivers[a,b], Victor Coba[b] and Melissa Whitmill[b]

[a]Department of Emergency Medicine and
[b]Department of Surgery, Henry Ford Health Systems, Detroit, Michigan, USA

Correspondence to Emanuel P. Rivers, MD, MPH, IOM, Department of Emergency Medicine and Surgery, Henry Ford Hospital, 2799 West Grand Boulevard, 270-Clara Ford Pavillon, Detroit, MI 48202, USA
Tel: +1 313 916 1557; fax: +1 313 916 8675; e-mail: erivers1@hfhs.org

Current Opinion in Anesthesiology 2008, 21:128–140

Purpose of review
Aggressive approaches to acute diseases such as acute myocardial infarction, trauma, and stroke have improved outcomes. Early goal-directed therapy for severe sepsis and septic shock represents a similar approach. An analysis of the literature assessing external validity and generalizability of this intervention is lacking.
Recent findings
Eleven peer-reviewed publications (1569 patients) and 28 abstracts (4429 patients) after the original early goal-directed therapy study were identified from academic, community and international settings. These publications total 5998 patients (3042 before and 2956 after early goal-directed therapy). The mean age, sex, APACHE II scores and mortality were similar across all studies. The mean relative and absolute risk reduction was 0.46 ± 26% and 20.3 ± 12.7%, respectively. These findings are superior to the original early goal-directed therapy trial which showed figures of 34% and 16%, respectively. A consistent and similar decrease in healthcare resource consumption was also found.
Summary
Early goal-directed therapy modulates systemic inflammation and results in significant reductions in morbidity, mortality, and healthcare resource consumption. Early goal-directed therapy has been externally validated and is generalizable across multiple healthcare settings. Because of these robust findings, further emphasis should be placed on overcoming logistical, institutional, and professional barriers to implementation which can save the life of one of every six patients presenting with severe sepsis and septic shock.


Şüpheli enfeksiyon
Kültür
Yüksek riskli hasta
SKB<90 mmHg
Laktat>4 mmol/L
1 saat içinde ab
Kaynak kontrolü
CVP
<8 mmHg
Kristaloid
veya kolloid
>8–12 mmHg
MAP
65 or >90 mmHg
Vazoaktif
ajanlar
>65–90 mmHg
Scvo2
<70%
Eritrosit
replasmanı
>70%
<70%
İnotroplar
>70%
Hedefe
ulaşma
Hayır
Oksijen
tüketiminin
azalması

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1. SIRS nedeninin infeksiyon olduğunun saptanması
2. İnfeksiyon yerinin belirlenmesi ve mikrobiyolojik bulguların yorumlanması
3. Antibiyotik tedavisi
4. İnfeksiyon kaynağının kontrol edilmesi

1.SIRS Nedeninin İnfeksiyon Olduğunun Saptanması

DOKU HARABİYETİ:
Cerrahi / travma
Hematom / venöz tromboz
Miyokard / pulmoner infarkt
Transplant rejeksiyonu
Pankreatit
Eritrodermi

METABOLİK:
Tiroid krizi
Akut adrenal yetmezliği

MALİGNİTE:
Hipernefroma
Lenfoma
Tümör lizis sendromu

TEDAVİ İLE İLİŞKİLİ:
Kan ürünleri
Sitokinler (GM-CSF)
Malign hipertermi (halotan)
Nöroleptik malign sendrom (haloperidol)
Opiat / benzodiazepinler

NÖROLOJİK:
Subaraknoid kanama

2.İnfeksiyon yerinin belirlenmesi ve mikrobiyolojik bulguların yorumlanması

- ✓ Kültürlerin alınması
- ✓ Mikrobiyolojik tetkiklerin yapılması
- ✓ Mikrobiyolojik bulguların yorumlanması
  - -Kolonizasyon-infeksiyon
  - -Önceden antibiyotik tedavisi
  - -İyi bilinmeyen mikroorganizmaların varlığı

3.Antibiyotik tedavisi

- ✓ Septik şokun tanısından itibaren ilk 1 saat içinde
- ✓ Ampirik antibiyotik tedavisi:İnfeksiyonun kaynağı ve olası mikroorganizmalar
- ✓ Gram boyama sonuçları
- ✓ Hastane mikrobiyal florası
- ✓ Konakçının durumu
  - -immunsupresyon
  - -hepatorenal disfonksiyon
  - -gebelik
  - -hastaneye yatış hikayesi
  - -ilaç intoleransı

Duyarlı Patojen

Bulaşın Önlenmesi

İnfeksiyonun Önlenmesi

Antibiyotik Direnci

İnfeksiyon

Etkili Tanı ve Tedavi

Uygun Antibiyotik Kullanımı

Antibiyotik Kullanımı

CDC

Acil Tıp Uzmanları Derneği

2008

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- ✓ Başlangıçta antibiyotik seçimi empiriktir.
- ✓ Etken saptanana dek geniş-spektrumlu antibiyotik tedavisine devam edilmeli, günlük değerlendirme ile tedavi gözden geçirilmeli ve daraltılmalıdır. (Deeskalasyon)
- ✓ Tedavi 7-10 gün olmalıdır.
- ✓ SIRS tablosunun infeksiyöz nedenli olmadığı saptanırsa direnç gelişim riskini en aza indirmek için antibiyotik tedavisi kesilmelidir

Boş İnançlar

- ✓ Geniş spektrum iyidir.
- ✓ Yanıtsızlık her zaman spektrum dışında kalan bir etken sonucudur.
- ✓ Şüphede kalındığında ilaçlar değiştirilmeldir
- ✓ Daha fazla hastalık daha fazla ilaç gerektirir.
- ✓ Yanıt, tanının doğru olduğu anlamına gelir.
- ✓ Daha ciddi hastalık daha ciddi ve daha yeni antibiyotik gerektirir.

The Cochrane Collaboration,2006

- 64 çalışma,7586 hasta
- 44 çalışma→Yeni, geniş-spektrumlu BL ile daha eski, daha az potent BL + AG
- Monoterapide ölüm % 15 daha az
- 20 çalışma→Aynı BL ile BL + AG
- Mortalitede fark yok
- Tüm nedenlere bağlı ölümde fark yok
- Hastanede yatış süresi fark yok

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- ✓ Uzmanların çoğu, ağır sepsis veya septik şoktaki nötropenik hastalarda kombinasyon tedavisini tercih ederler
- ✓ Pseudomonas ile infekte hastalarda kombinasyon tedavisini tercih edilmelidir.
- ✓ Kombinasyon tedavisine 3-5 günden uzun devam etmemelidir

4.İnfeksiyon kaynağının kontrol edilmesi

- ✓ İlk 6 saat içinde infeksiyon kaynağı uygun yöntemlerle araştırılmalı ve uygun tedavi seçilmelidir.

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Source Control

Source Control Technique	Examples
Drainage	Intra-abdominal abscess
	Thoracic empyema
	Septic arthritis
Debridement	Pyelonephritis, cholangitis
	Infected pancreatic necrosis
	Intestinal infarction
	Mediastinitis
Device removal	Infected vascular catheter
	Urinary catheter
	Infected intrauterine contraceptive device
Definitive control	Sigmoid resection for diverticulitis
	Cholecystectomy for gangrenous cholecystitis
	Amputation for clostridial myonecrosis

Sepsis ~ Adrenal Yetmezlik

- ✓ Relatif adrenal yetmezlik (~ %50)
  - ✓ Glukokortikoid reseptör direnci
  - ✓ Adrenal hemoraji / invazyon
  - ✓ Kortizol sentezinde azalma
- ✓ Diürnal ritimde bozukluk

- ACTH stimülasyon testi / Herhangi bir zamanda alınan kortizol değeri ?
  - 30 –60. dk kortizol değeri < 18 μg/dL veya < 9 μg/dL artış → adrenal yetmezlik

Sepsiste Steroid Tedavisi

- ✓ *Annane JAMA 2002*

  Relatif adrenal yetm olanda 200mg hidrokortizon+mineralokortikoid→28 günlük mortalite ↓

- ✓ *Annane BMJ 2004,Meta-analiz: 16 çalışma*

  ≤ 300 mg hidrokortizon eş-değeri steroid dozunda ve ≥ 5 gün uygulamada 28 günlük ve hastane mortalitesinde, ve şok süresinde azalma var

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Hydrocortisone Therapy for Patients with Septic Shock

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Şoktan çıkışı hızlandırıyor

BACKGROUND

Hydrocortisone is widely used in patients with septic shock even though a survival benefit has been reported only in patients who remained hypotensive after fluid and vasopressor resuscitation and whose plasma cortisol levels did not rise appropriately after the administration of corticotropin.

METHODS

In this multicenter, randomized, double-blind, placebo-controlled trial, we assigned 251 patients to receive 50 mg of intravenous hydrocortisone and 248 patients to receive placebo every 6 hours for 5 days; the dose was then tapered during a 6-day period. At

Sağkalım üzerie etkisi yok

have a res

RESULTS

Of the 499 patients in the study, 233 (46.7%) did not have a response to corticotropin (125 in the hydrocortisone group and 108 in the placebo group). At 28 days, there was no significant difference in mortality between patients in the two study groups who did not have a response to corticotropin (39.2% in the hydrocortisone group and 36.1% in the placebo group, P=0.69) or between those who had a response to corticotropin (28.8% in the hydrocortisone group and 28.7% in the placebo group, P=1.00). At 28 days, 86 of 251 patients in the hydrocortisone group (34.3%) and 78 of 248 patients in the placebo group (31.5%) had died (P=0.51). In the hydrocortisone group, shock was reversed more quickly than in the placebo group. However, there were more episodes of superinfection, including new sepsis and septic shock.

CONCLUSIONS

Hydrocortisone did not improve survival or reversal of shock in patients with septic shock, either overall or in patients who did not have a response to corticotropin, although hydrocortisone hastened reversal of shock in patients in whom shock was reversed. (ClinicalTrials.gov number, NCT00147004.)

From Hadassah Hebrew University Medical Center, Jerusalem (C.L.S., Y.G.W., J. Benbenishty); Raymond Poincaré Hospital, University of Versailles, UniverSud Paris, Garches, France (D.A.); Charité Universitätsmedizin Berlin, Campus Virchow-Klinikum, Berlin (D.K.); Hospital de St. António dos Capuchos, Centro Hospitalar de Lisboa Central, Lisbon, Portugal (R.M.); Bloomsbury Institute of Intensive Care Medicine, University College London, London (M.S.); Analytica International, Loerrach, Germany (K.F.); Klinikum Mannheim, Mannheim, Germany (A.K.); Zentralklinikum Augsburg, Augsburg, Germany (H.F.); St. Luc University Hospital, Université Catholique de Louvain, Brussels (P.-F.L.); Friedrich Schiller Universität, Jena, Germany (K.R.); Health Services Research Unit, University of Aberdeen, Aberdeen, United Kingdom (B.H.C.); Hôpital Lariboisière, Denis Diderot University of Paris, Paris (D.P.); and Klinikum der Universität, Ludwig Maximilians Universität, Munich, Germany (J. Briegel). Address reprint requests to Dr. Sprung at the General Intensive Care Unit, Department of Anesthesiology and Critical Care Medicine, Hadassah Hebrew University Medical Center, P.O. Box 12000, Jerusalem, Israel 91120, or at sprung@cc.huji.ac.il.

*Members of the Corticosteroid Therapy of Septic Shock (CORTICUS) study group are listed in the Appendix.

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- ✓ Sıvı ve vazopressör tedaviye dirençli septik şok
- ✓ A CTH stimülasyon testi yapma
- ✓ Hidrokortizon varken deksametazon yapma
- ✓ Mineralokortikoid etkisi olmayan steroid veriliyorsa 50 mg fludrokortizon eklenmeli
- ✓ Hidrokortizon dozu 300 mg üzerinde olmamalı

Rekombinan Human Aktive Protein C

- ✓ Endojen APC`nin rekombinant formu
- ✓ İnsan böbrek 293 hücre
- ✓ PROWESS (PROtein C Worldwide Evaluation in Severe Sepsis)
- ✓ Prospektif, randomize, çift kör, uluslararası, çok merkezli, 1690 hasta
- ✓ 28 günlük mortalite; plasebo %31 APC %25
- ✓ ADDRESS,ENHANCE

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- ✓ APACHE II >25
- ✓ MOF
- ✓ Ölüm riski ↑

Recombinant Activated Protein C Nominal Group Vote

Strong for use, 6
Weak for use, 15
Neutral, 1
Weak for not using, 0
Strong for not using, 0

Kontrendikasyonlar

Aktif iç kanama

İntrakranial patoloji, neoplazm ya da serebral herni bulgusu olan hastalar

≥ 15 IU/kg/saat ya da daha yüksek dozda eşzamanlı heparin tedavisi

Sepsise bağlı akut koagülopati dışında bilinen kanama diyatezi

Kronik ağır karaciğer hastalığı

Trombosit sayısı <30.000x106/L

Kanama riski yüksek olan hastalar

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Kan ürünleri

- ✓ Hb < 7 olmadıkça eritrosit replasmanı yapma
- ✓ Kanama ve koagülasyon testlerinin bozukluğu durumunda invaziv işlem öncesi ve kanama haricinde rutin taze donmuş plazma kullanımından kaçınılması
- ✓ Antitrombin III uygulanmaması
- ✓ Trombosit
  - < 5000/mm³
  - 5000-30.000 /mm³ kanama riski varlığında
  - >50.000 /mm³ invaziv işlem veya cerrahi

Waiting
for the
Perfect
man

Vaka

- ✓ 63 yaşında erkek hasta
- ✓ Sol yüz yarısında uyuşma ve ağrı
- ✓ Sol gözde şişlik,görme kaybı,sol göz kapağında düşme
- ✓ İTP,Steroid induced DM
- ✓ Ateş: 36,6 °C,Nabız: 92/dak.,TA:120/80 mmHg,
- ✓ Fizik muayene: Sol periorbital dokuda şişlik

Acil Servis Bulgular

Nörolojik muayene

- ✓ Sol gözde direkt ve indirekt ışık refleksi yok
- ✓ Sol gözde tam görme kaybı
- ✓ Solda optik disk soluk
- ✓ Sol gözde pitoz ve ekzoftalmus
- ✓ Sol göz orta hatta ve hareketleri her yöne tam kısıtlı

Acil Servis Bulgular

Nörolojik muayene

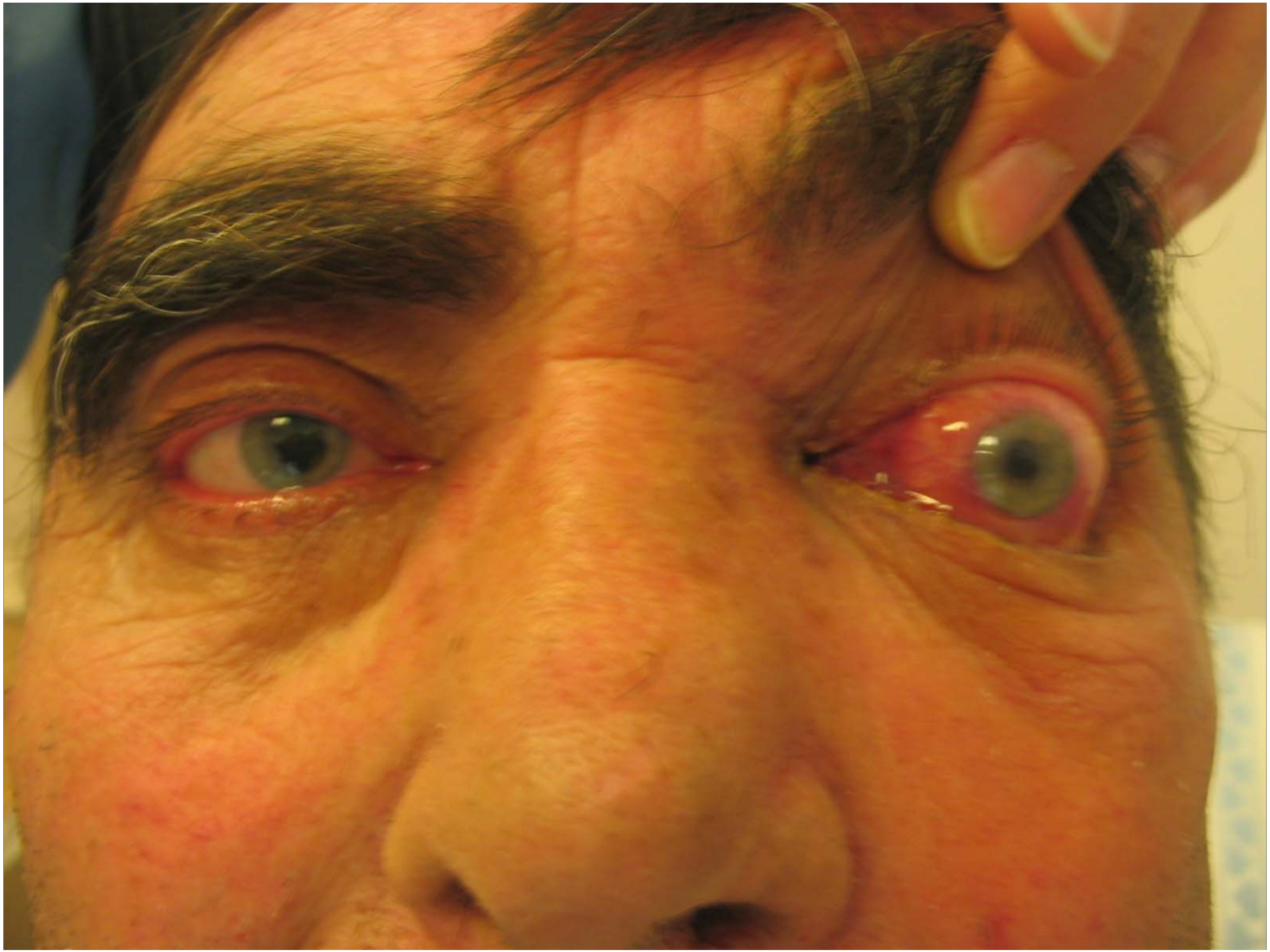
- ✓ Sol V1, V2, V3′de hipoestezi
- ✓ Solda kornea refleksi alınamıyor
- ✓ Sol nazolabial oluk silik (yanak ödemine bağlı mekanik kısıtlılık)
- ✓ Diğer NM bulguları normal

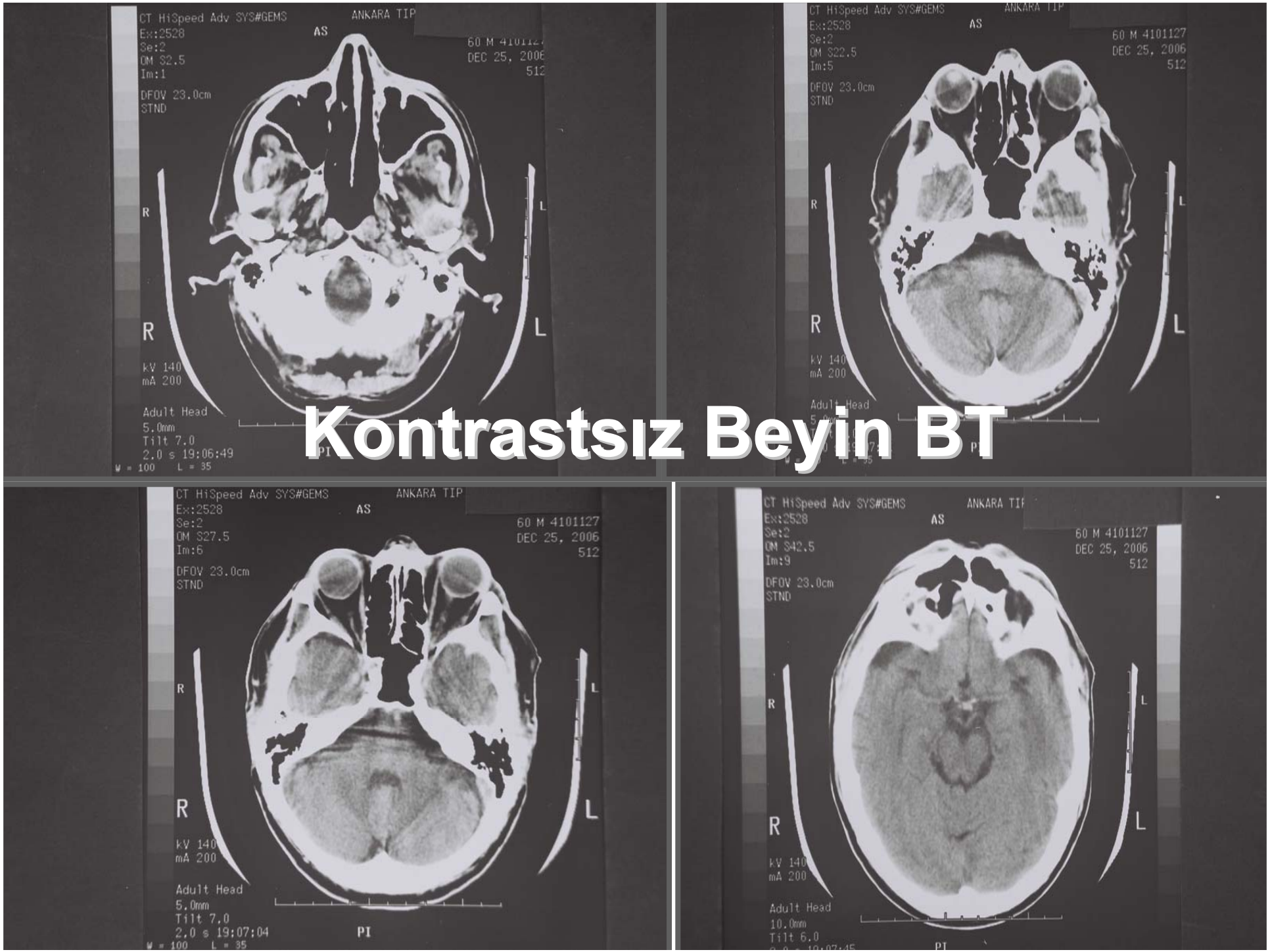
* Kontrastsız beyin BT

Acil Servis Bulgular

- ✓ BK: 20.000/mm^3
- ✓ Trombosit: 18.000/mm^3
- ✓ AKŞ: 213 mg/dL
- ✓ Na: 125 mEq/L
- ✓ Protein 30 mg/dL
- ✓ Glukoz 250 m/dL
- ✓ Keton (-)

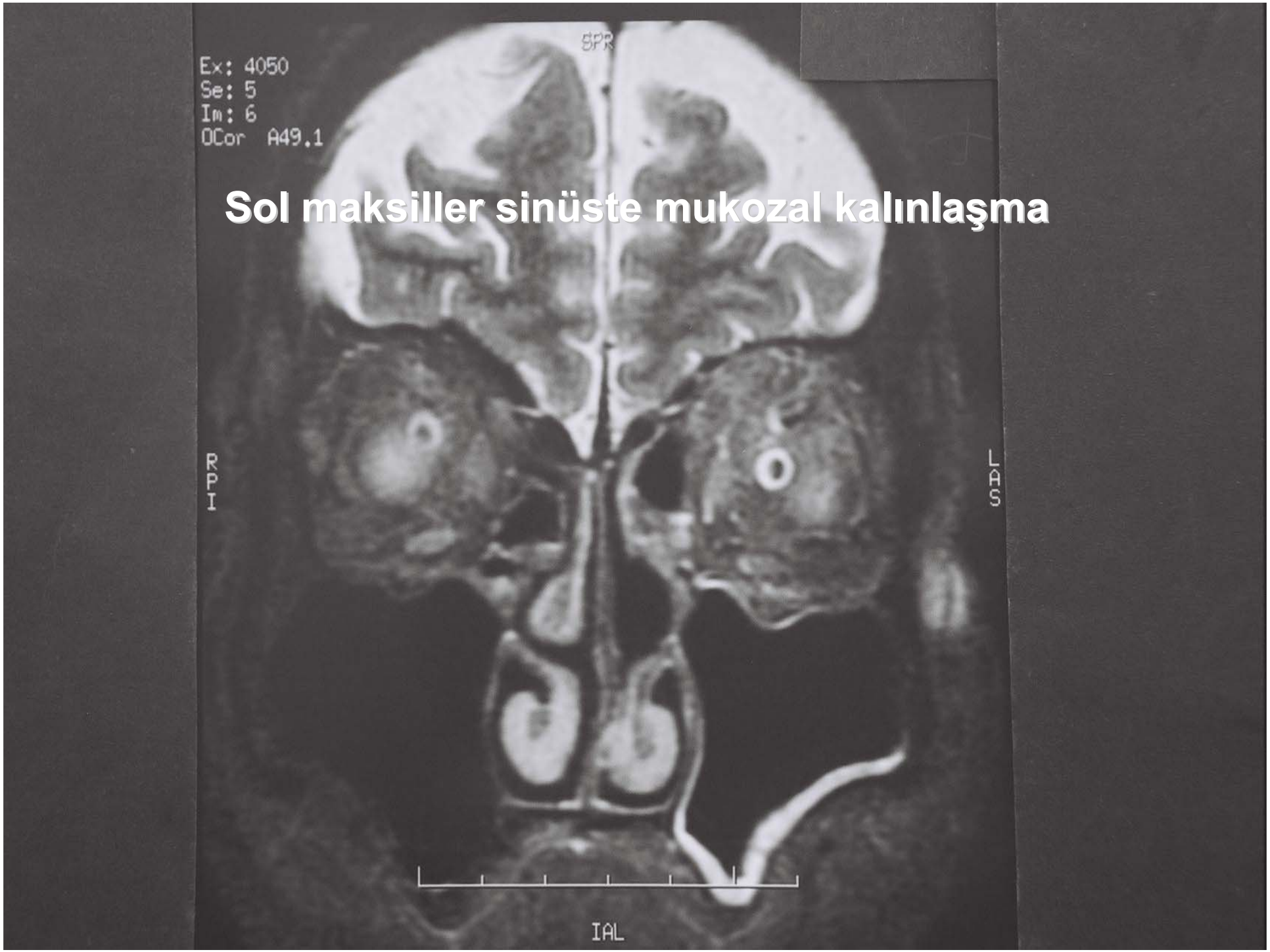
} TİT





Kontrastsız Beyin BT

Sol retrobulber yağ doku artışı,yağ dokuda kirlenme
Kanama? Enfeksiyon?



Sol maksiller sinüste mukozal kalınlaşma

Acil Servis Bulgular (3.gün)

Nörolojik Muayene

- ✓ Sol V1, V2'de hipoestezi anesteziye ilerledi
- ✓ Diğer NM bulgularında değişiklik yok

  * Yumuşak doku enfeksiyonu?

    Sulbaktam ampisilin+Siprofloksasin

Acil Servis Bulgular (3.gün)

*LP: Basınç: 20.5 cmH_2O, berrak, renksiz

Glukoz: 139 mg/dl (KŞ: 200 mg/dl)

Protein: 36 mg/dL, klor: 117 mEq/L

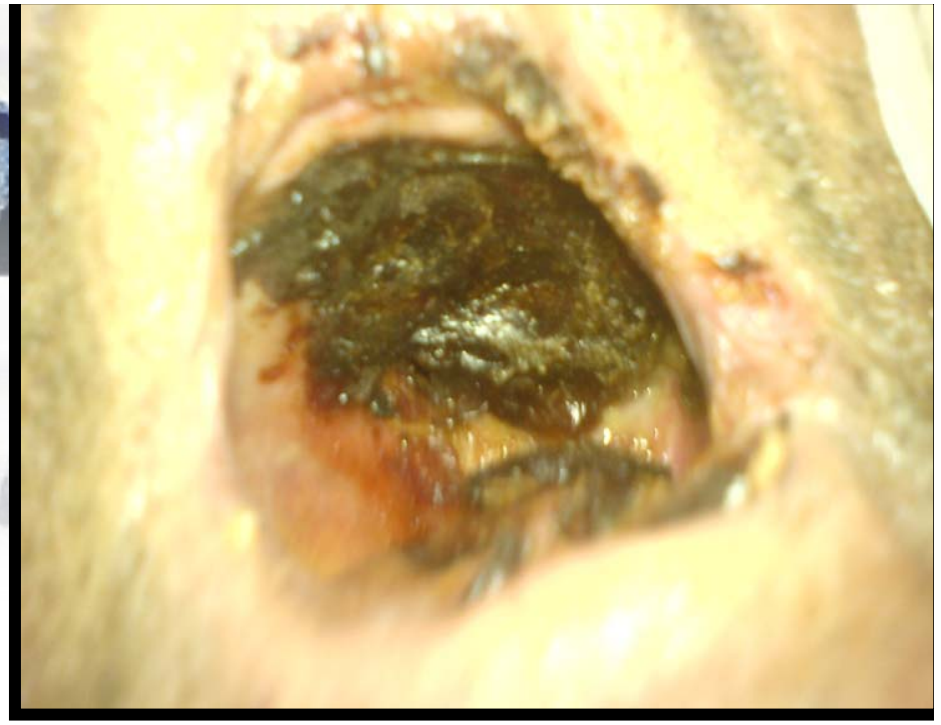
LDH: 79 IU/L

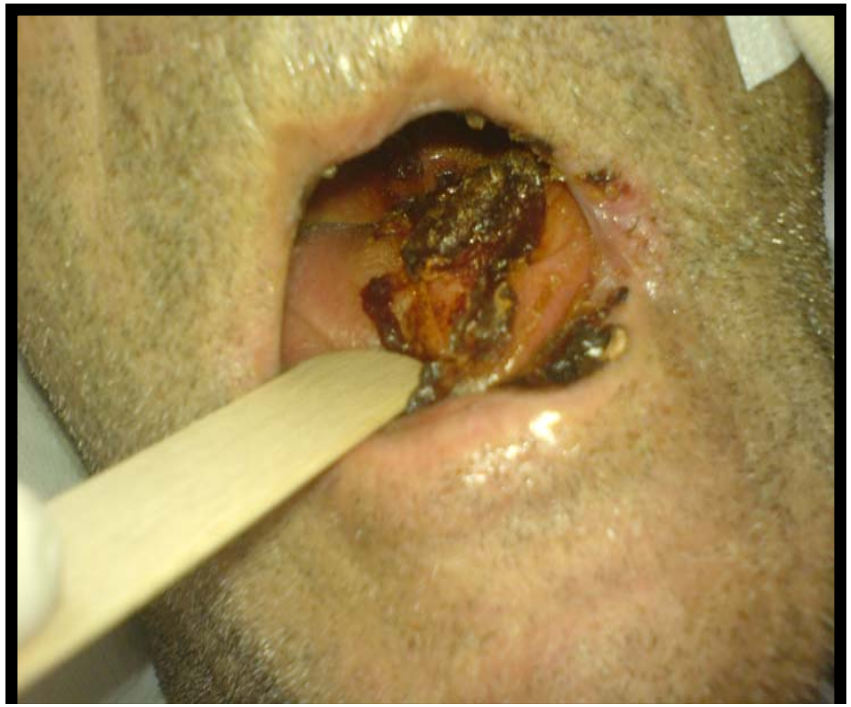
Hücre: 30 lökosit

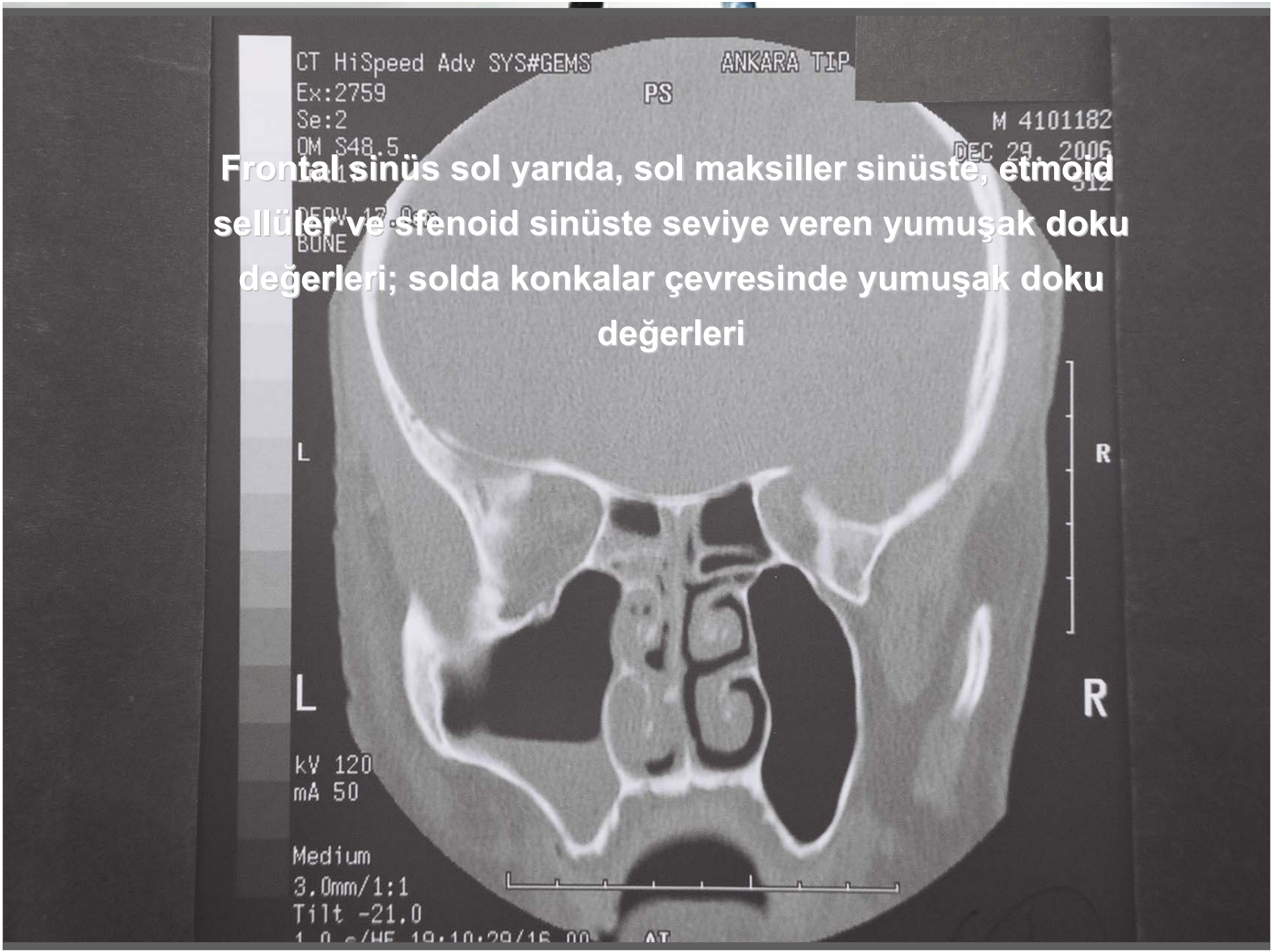
Mantar, ARB, kültür, gram boyama:(-)

Acil Servis Bulgular (5.gün)

- ✓ Amfoterisin B başlanması → infeksiyon hastalıkları konsültasyonu
- ✓ Prednolün kesilmesi
- ✓ Burun kavitesinde krutlar, sol taraf sert damakta 2 cm çapta, orta hattı geçmeyen, beyaz-gri plaklar (nekrotize doku), ülserasyon yok, sol yumuşak damak pitotik → KBB Konsültasyonu
- ✓ Paranazal sinüs BT





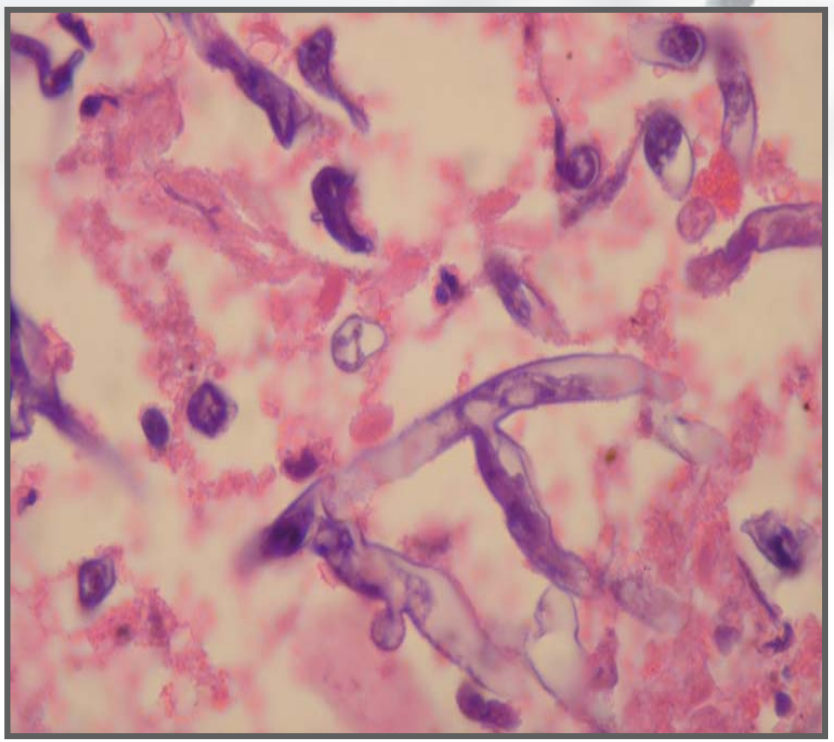


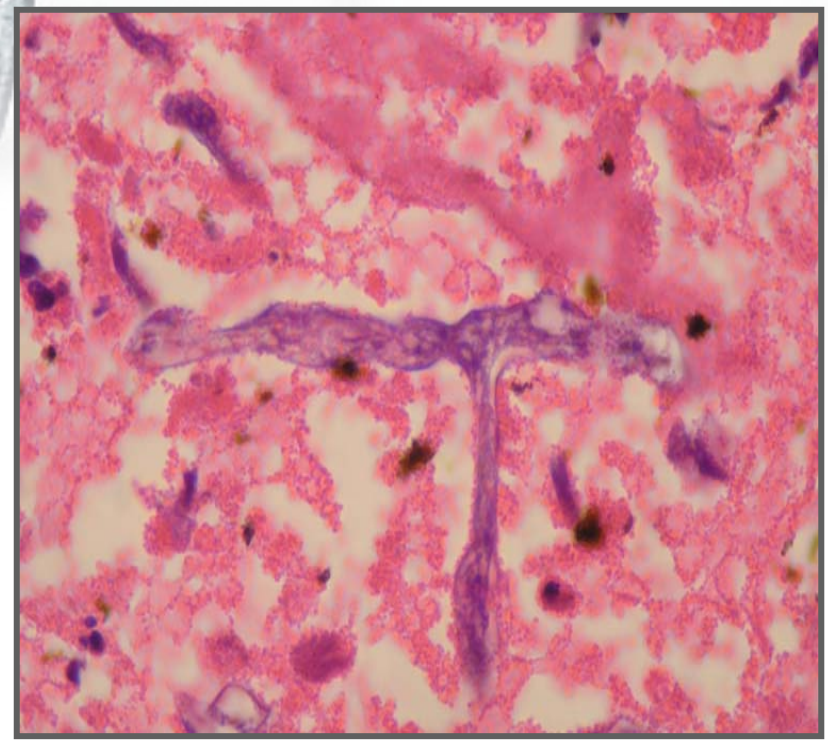
Frontal sinüs sol yarıda, sol maksiller sinüste, etmoid sellüler ve sfenoid sinüste seviye veren yumuşak doku değerleri; solda konkalar çevresinde yumuşak doku değerleri

Acil Servis Bulgular (5.gün)

- ✓ İnfeksiyon hast Kons: Amfoterisin B 3mg/kg/gün iv
- ✓ Hasta Nöroloji Anabilim Dalı'na yatırıldı
- ✓ Nazal kavite ve sert damak mukozasından histopatolojik tanı için biyopsi alındı → Rinoorbital fungal enfeksiyon (Mukormikozis ?)

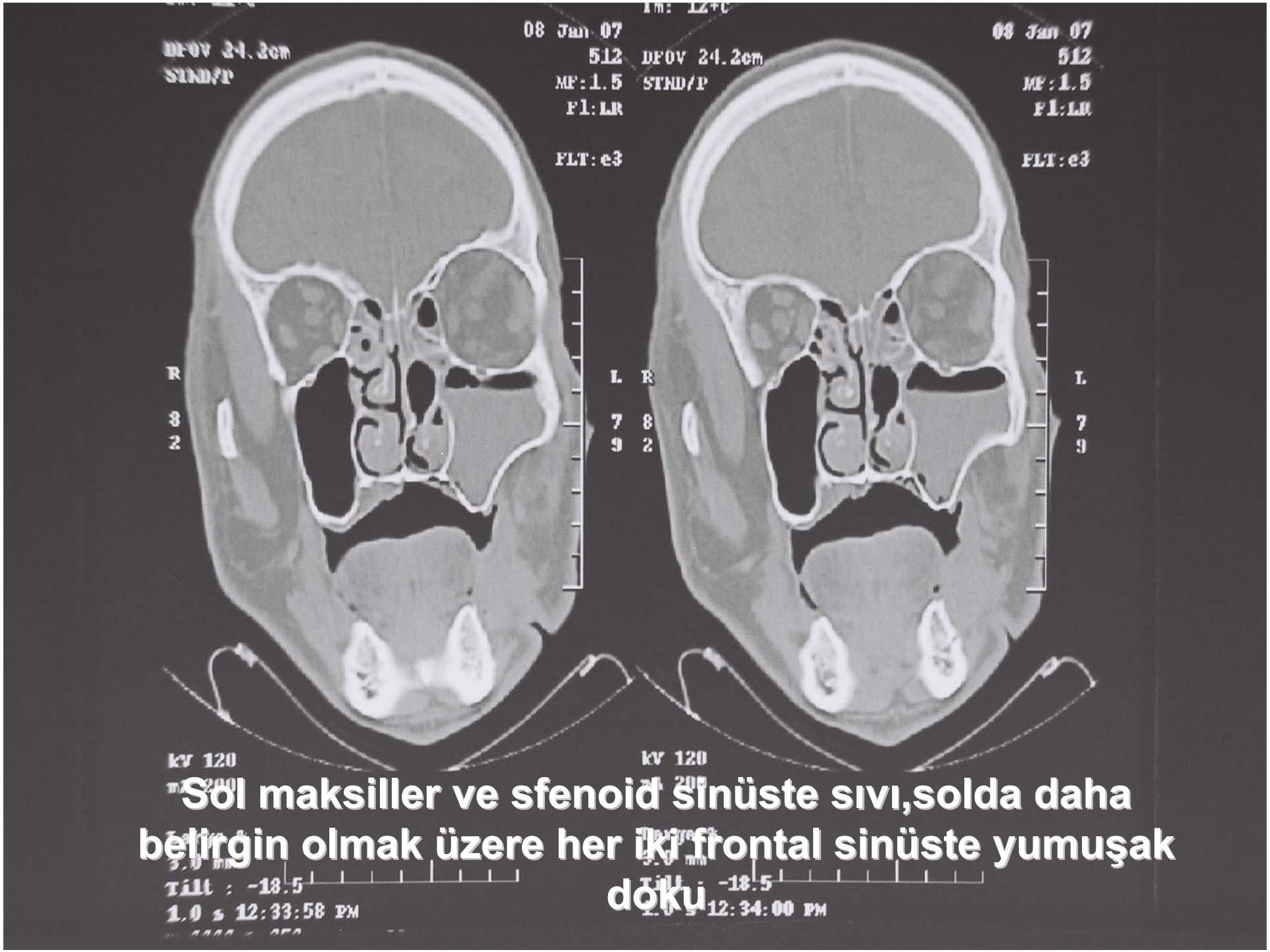
10.gün



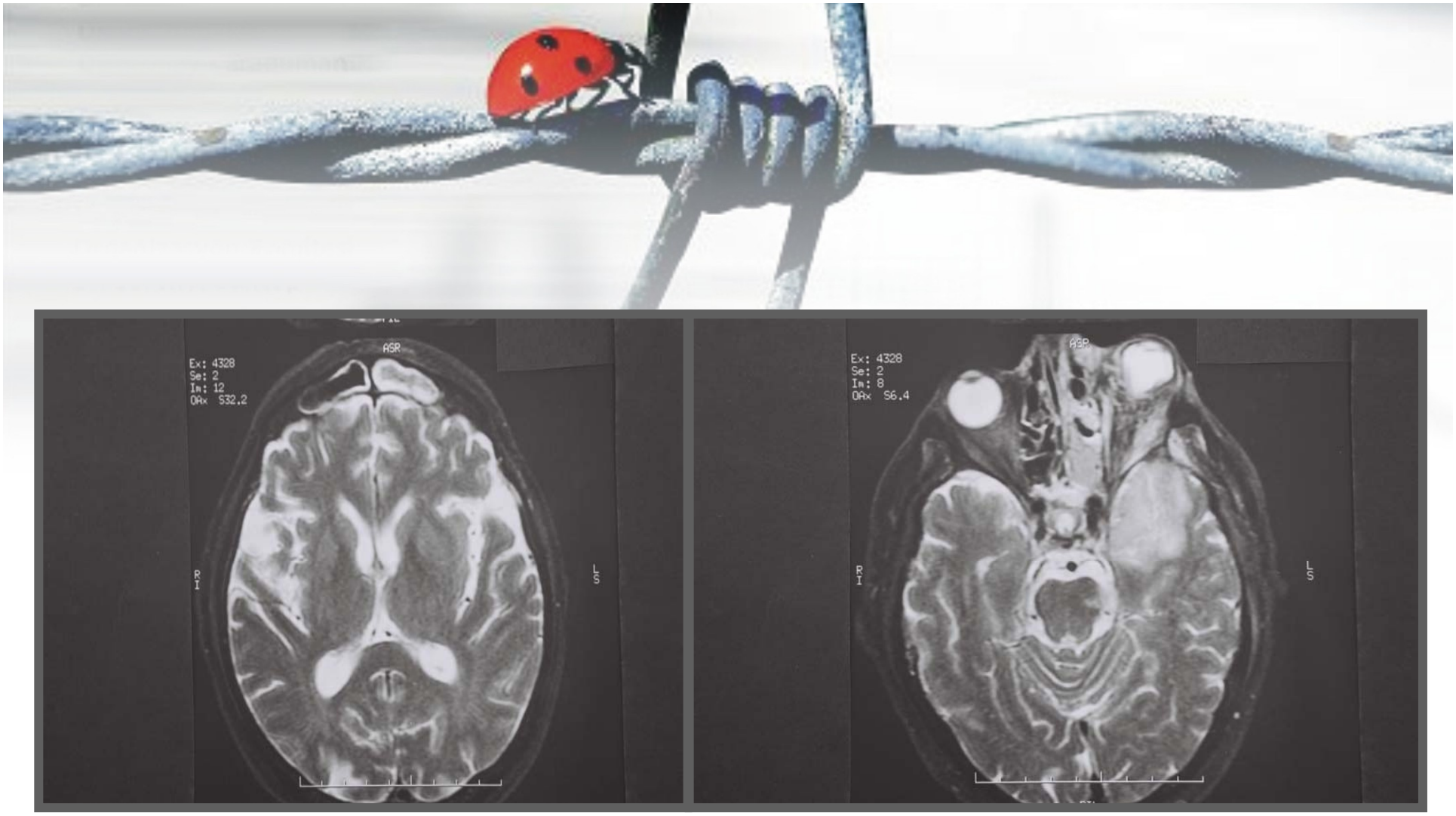


Mukormikozis ile uyumlu nekrotizan mantar infeksiyonu

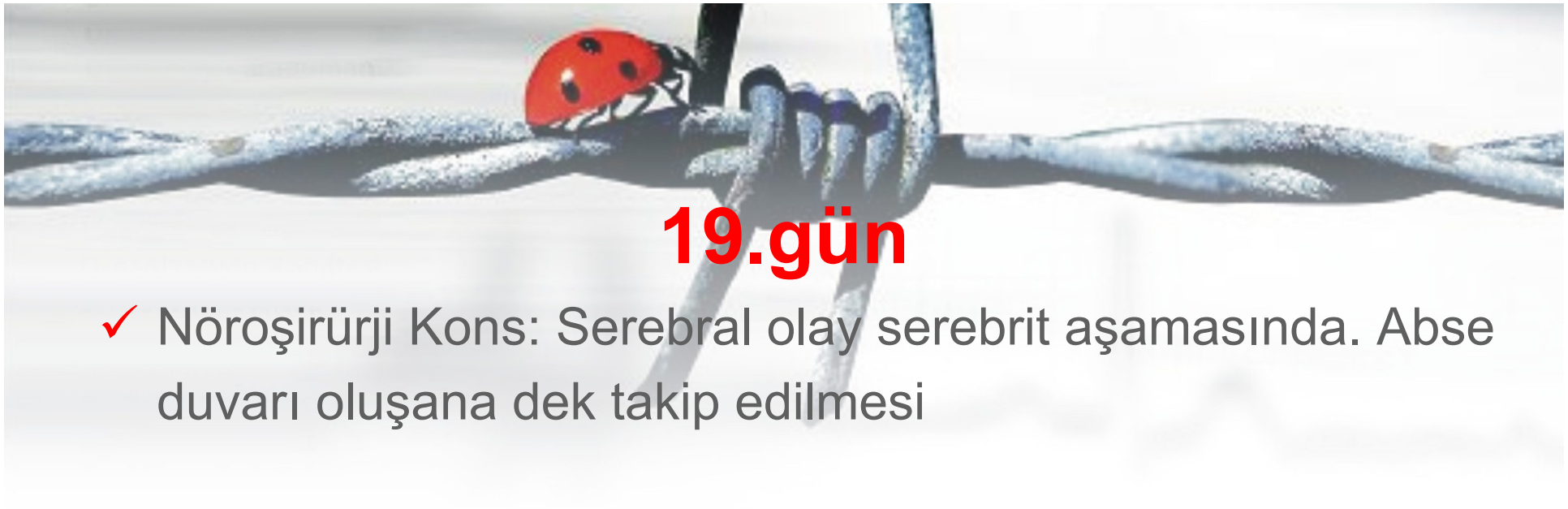
10.gün



Sol maksiller ve sfenoid sinüste sıvı,solda daha belirgin olmak üzere her iki frontal sinüste yumuşak doku



Bilateral frontoparietalde iskemik gliottik alanlar
Sol temporal lobda opaklaşmalı alan



19.gün

- ✓ Nöroşirürji Kons: Serebral olay serebrit aşamasında. Abse duvarı oluşana dek takip edilmesi

- ✓ KBB+Göz operasyonu:Glob egzanterasyonu + subtotal maksillektomi + etmoidektomi + sert damak sol yarı eksizyonu

- ✓ Progresyon nedeniyle Lipozomal Amfoterisin B 7 mg/kg + Kaspofungin asetat 50 mg/gün tedavisine geçildi

Orbital Apeks Sendromu

- ✓ Eksternal oküler kasları innerve eden 3 kraniyal sinirin paralizisi (III, IV, VI)
- ✓ Trigeminal sinirin V1 dalı
- ✓ Optik sinir lezyonu
- ✓ Mukormikozis

Mukormikozis

✓ Taksonomik sınıflandırma

➡Sınıf: Zygomycetes

➡Aile: a) Entomophthorales
b) Mucorales

➡Tür: 1) Rhizopus
2) Mucor
3) Absidia

Mukormikozis

- ✓ Mukormikozis hızlı progresyon gösteren ve oldukça fatal seyreden fungal infeksiyondur
- ✓ Doğada yaygın olarak bulunur ve sağlıklı insanların nazofaringeal mukozalarında kolonize olabilir
- ✓ Mukormikozis özellikle immünsupresyondaki veya diabetik hastalarda görülür

Mukormikozis

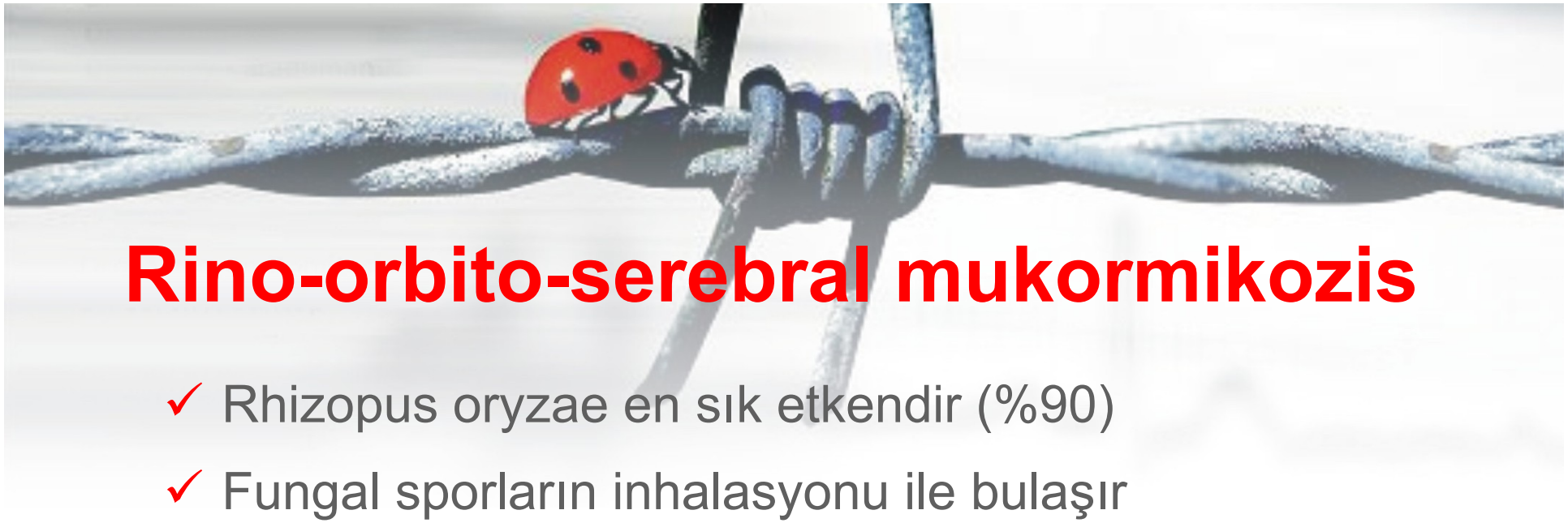
Predispozan faktörler

- ✓ Diabetes mellitus (%60-80)
- ✓ Hematolojik maligniteler
- ✓ Organ transplantasyonu
- ✓ Malnutrisyon
- ✓ Renal yetmezlik
- ✓ AIDS
- ✓ İntravenöz madde bağımlılığı
- ✓ Desferroksamin tedavisi
- ✓ İmmünsupresif ilaç kullanımı

Mukormikozis

Klinik formlar

- ✓ Rino-orbito-serebral (en sık)
- ✓ Pulmoner
- ✓ Gastrointestinal
- ✓ Kutanöz
- ✓ Dissemine



Rino-orbito-serebral mukormikozis

- ✓ Rhizopus oryzae en sık etkendir (%90)
- ✓ Fungal sporların inhalasyonu ile bulaşır
- ✓ Arterlere afinite gösterir ve internal elastik lamina içerisinde gelişerek tromboz ve infarktlara yol açar
- ✓ Arter invazyonu ve trombozis antifungal ilaçların dokuya penetrasyonunu azaltır

Rino-orbito-serebral mukormikozis

- ✓ Nazal mukoza ve sinüslerin lokal infeksiyonu
- ✓ Orbital tutulum
  - * Orbital sellülit
  - * Oküler invazyon
  - * Orbital-periorbital kranial sinirlerin iskemisi
  - * 'Orbital apeks sendromu (II, III, IV, VI, V 1)'
- ✓ İntrakraniyal tutulum

* Komşuluk / Hematojen / Perinöral (?) yayılım
  - * İskemi (karotis interna, kavernöz sinüs)

Rino-orbito-serebral mukormikozis

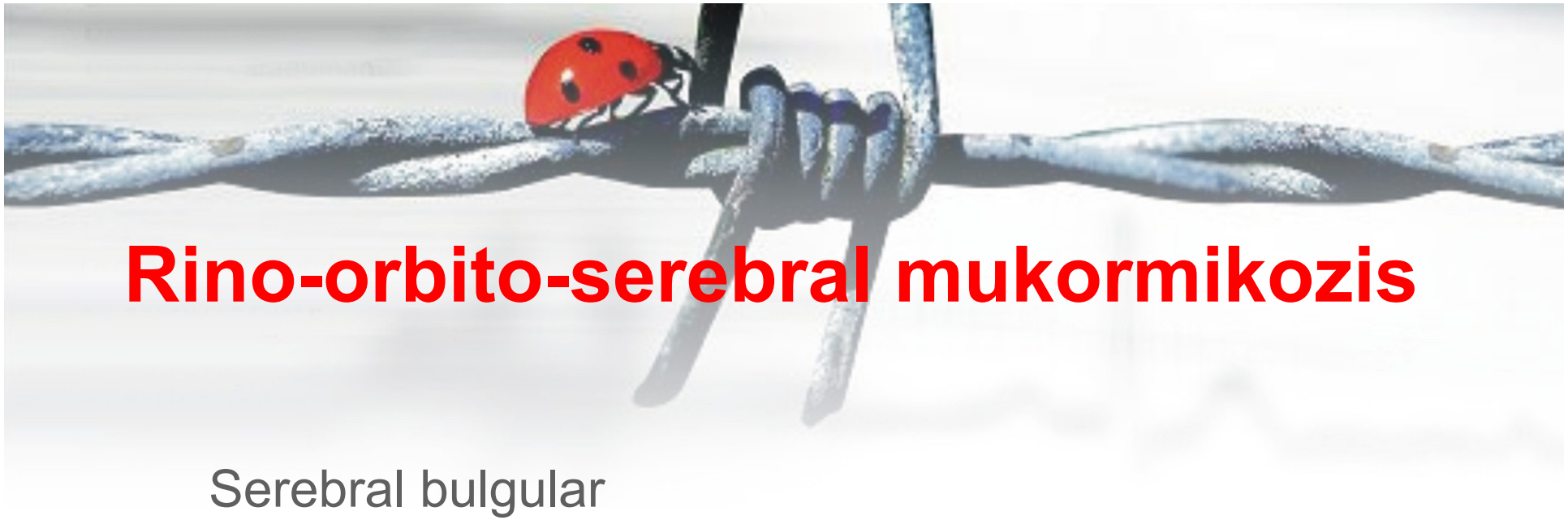
İlk 72 saatteki semptomlar

- ✓ Ateş (% 44)
- ✓ Nazal ülserasyon ve nekroz (siyah) (% 38)
- ✓ Periorbital / fasial ödem (% 34)
- ✓ Sinüzit (% 26)
- ✓ Baş / Yüz ağrısı (% 25-22)
- ✓ Hemorajik nazal akıntı

Rino-orbito-serebral mukormikozis

Göz semptomları

- ✓ Eksternal oftalmopleji (% 67)
- ✓ Görme keskinliğinde azalma (% 65)
- ✓ Proptozis (% 64)
- ✓ İnternal oftalmopleji (% 43)
- ✓ Konjonktival hiperemi (% 19)
- ✓ Dilate ve ışık yanıtı olmayan pupil
- ✓ Korneal anestezi



Rino-orbito-serebral mukormikozis

Serebral bulgular

- ✓ Bilinç bozukluğu (% 22)
- ✓ Fokal nörolojik defisit

Laboratuvar

- ✓ Nötrofilik lökositoz görülür ve tedaviye olan yanıtın değerlendirmesinde kullanılır
- ✓ BOS bulguları non-spesifiktir (nötrofilik pleositoz ve protein artışı olabilir)
- ✓ BT ile sinüzit saptanabilir ancak benign mukozal hastalıklardan ayrımı zordur
- ✓ Kranial ve orbita MRG ile lezyonlar kolaylıkla gösterilebilir

Tanı

- ✓ Histopatolojik inceleme
  - * Geniş, irregüler, non-septat ve dik açıyla dallanan hifaların görülmesi

- ✓ Fungal kültür (Sabouraud's agar)
  - * Kültürde üreme olmaması tanıyı ekarte ettirmez
  - * Kültür sonucunu beklemek tedaviyi geciktirir

Tedavi

- ✓ Hiperglisemi ve asidozun düzeltilmesi
- ✓ Amfoterisin B (lipozomal)
- ✓ Cerrahi debridman
- ✓ Hiperbarik oksijen (?)

Kötü prognoz göstergeleri

- ✓ Tanı ve tedavide gecikme
- ✓ Altta yatan hematolojik, renal hastalık
- ✓ Bilateral sinüs tutulumu
- ✓ Fasiyal / Göz kapağı nekrozu
- ✓ Palatal nekroz
- ✓ Fokal sensorial-motor defisit
- ✓ Sağ kalım: %50-60

Sonuç

- ✓ Diabetik veya immünsupresif hastalarda akut olarak ortaya çıkan ağrılı oftalmoplejide mukormikozis düşünülmelidir

- ✓ Mukormikoziste tedavi başarısı, erken tanı ve erken agresif medikal, cerrahi tedaviye bağlıdır

SABRINIZ İÇİN TEŞEKKÜRLER