



DERİN BOYUN ENFEKSİYONLARI

Prof. Dr. Ergin ÇİFTÇİ

**Ankara Üniversitesi Tıp Fakültesi
Çocuk Enfeksiyon Hastalıkları Bilim Dalı**

www.erginciftci.com

12 Haziran 2025

İNSAN ve MİKROP

Bitmeyen Kavga...

ETKENLER



Deep Breath ©Melanie Weidner 2005 www.ListenForJoy.com

ETKENLER

Virüsler

Bakteriler

Mantarlar

Parazitler

AKUT TONSİLLOFARENJİT

Etiyoloji

ETKENLER			
BAKTERİLER	VİRÜSLER	ATİPİK ETKENLER	DİĞER NEDENLER
<i>Streptococcus pyogenes</i> (A grubu) B, C ve G grubu streptokoklar <i>Arcanobacterium haemolyticum</i> <i>Corynebacterium diphtheriae</i> <i>Fusobacterium necrophorum</i> <i>Francisella tularensis</i> <i>Neisseria gonorrhoeae</i> <i>Neisseria meningitidis</i> <i>Salmonella typhi</i> <i>Leptospira</i> spp. <i>Borelia</i> spp. <i>Yersinia enterocolitica</i>	<i>Epstein-Barr virüs</i> <i>Adenovirüs</i> <i>Herpes simpleks virüs</i> <i>İnfluenza virüs</i> <i>Parainfluenza virüs</i> RSV <i>Enterovirüsler</i> <i>Kızamık virüsü</i> <i>Kızamıkçık virüsü</i> <i>Sitomegalovirüs</i> <i>Rhinovirüs</i> <i>Coronavirus</i> <i>Human metapneumovirus</i> HIV	<i>Mycoplasma pneumoniae</i> <i>Mycoplasma hominis</i> <i>Chlamydia pneumoniae</i> <i>Chlamydia trachomatis</i> <i>Legionella pneumophila</i> <i>Coxiella burnetti</i> <i>Candida</i> <i>Toxoplasma gondii</i>	<i>Behçet sendromu</i> <i>Kawasaki hastalığı</i> PFAPA <i>Aftöz stomatit</i> <i>Steven-Johnson sendromu</i> <i>Nötropeni</i> <i>Kemoterapi</i>

AKUT TONSİLLOFARENJİT

GAS

TONSİLLOFARENJİT

En sık **5-15 yaş**larda görülür

Damlacık, yakın temas ve gıda yoluyla bulaşır

Kuluçka dönemi **2-5 gündür**

Ateş

Boğaz ağrısı

Baş ağrısı

Karın ağrısı

Kusma

TONSİLLOFARENJİT

Farenkste hiperemi, ödem, eksuda görülür

Çene açısındaki lenf bezleri büyük ve ağrılıdır

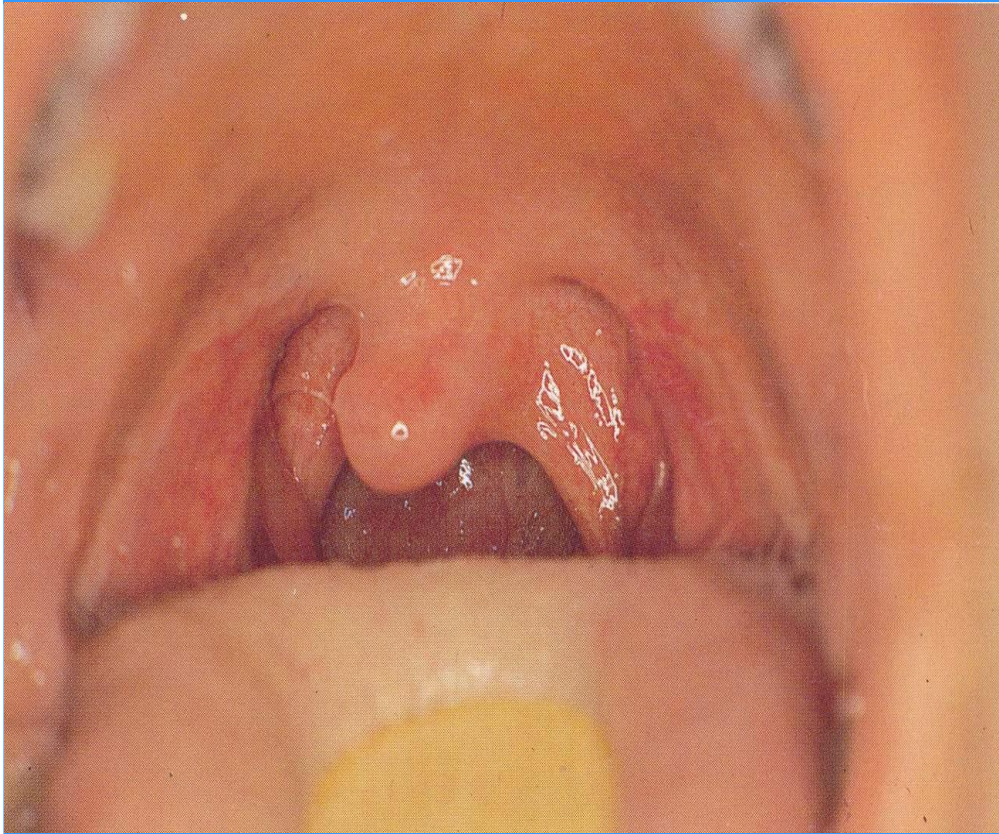
Hastalık kendiliğinden iyileşir



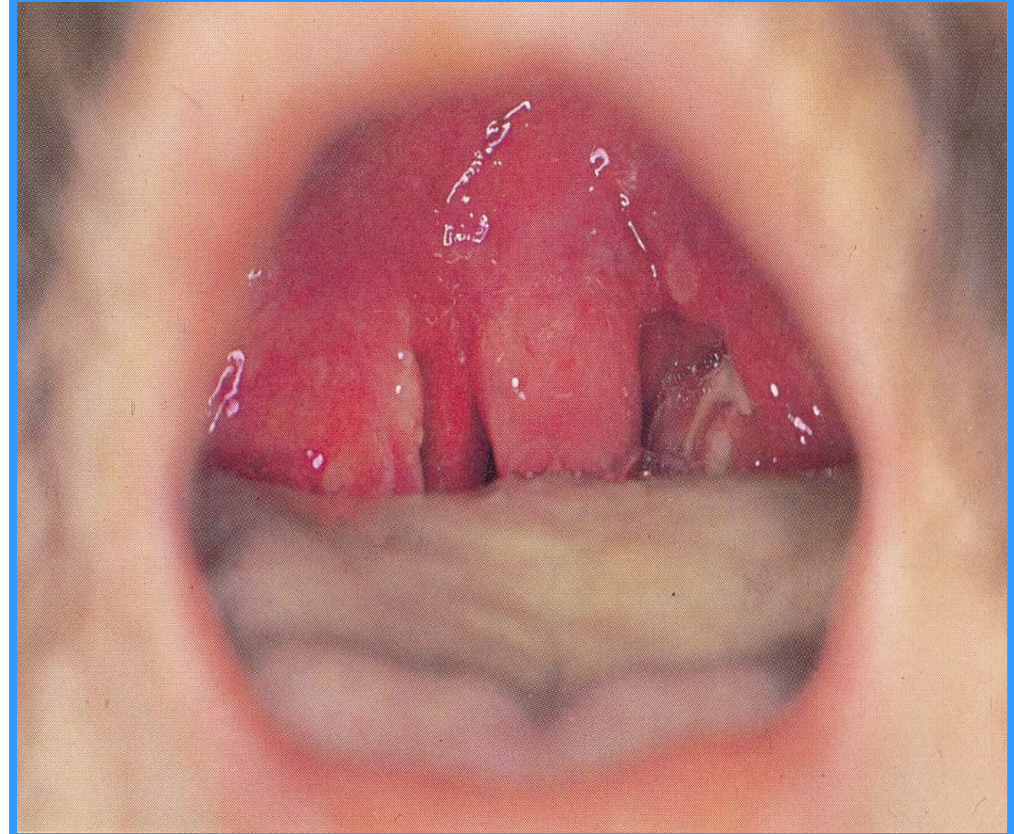
AKUT TONSİLLOFARENJIT

GAS

TONSİLLOFARENJIT



TONSİLLOFARENJIT



AKUT TONSİLLOFARENJIT

GAS

TONSİLLOFARENJIT



TONSİLLOFARENJIT



AKUT TONSİLLOFARENJIT

GAS

TONSİLLOFARENJIT



TONSİLLOFARENJIT





AKUT TONSİLLOFARENJİT

GAS

TONSİLLOFARENJİT

Akut faz reaktanları

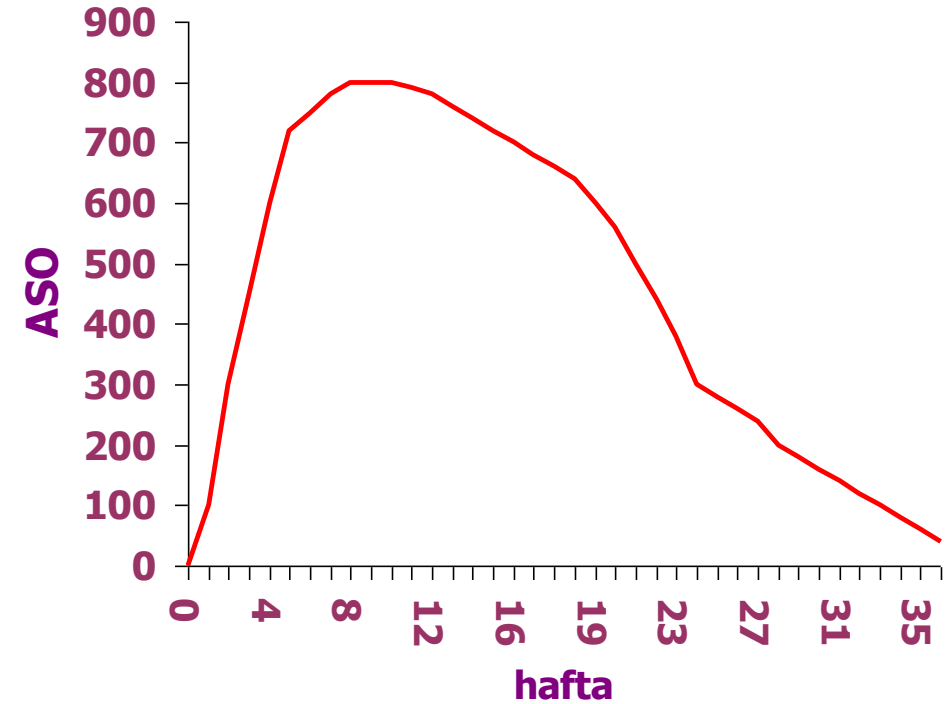
ASO

Hızlı antijen testi

Boğaz kültürü

**ALTIN STANDART TANI YÖNTEMİ
BOĞAZ KÜLTÜRÜDÜR**

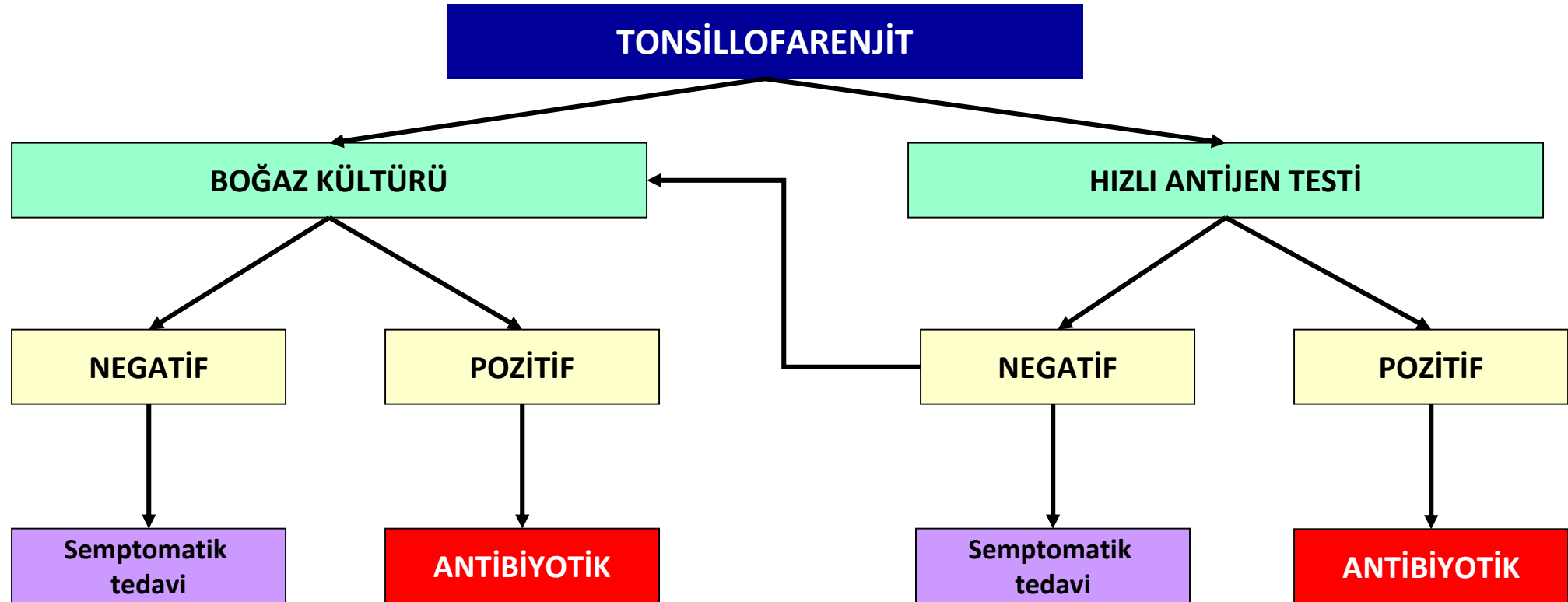
TONSİLLOFARENJİT



AKUT TONSİLLOFARENJİT

GAS

HIZLI ANTİJEN TESTLERİ



AKUT TONSİLLOFARENJİT

GAS

TONSİLLOFARENJİT

CENTOR KRİTERLERİ

1. Ateş $>38^{\circ}\text{C}$
2. Öksürük olmaması
3. Ağrılı ön servikal adenopati
4. Tonsillerde şişlik veya eksuda

Her birine 1 puan ver.

0-1 Puan Test ve antibiyotik gereksiz
2-3 Puan Test yap, sonucuna göre tedavi ver
4 Puan Test yapmadan antibiyotik ver

TONSİLLOFARENJİT

McISAAC KRİTERLERİ

1. Ateş $>38^{\circ}\text{C}$
2. Öksürük olmaması
3. Ağrılı ön servikal adenopati
4. Tonsillerde şişlik veya eksuda

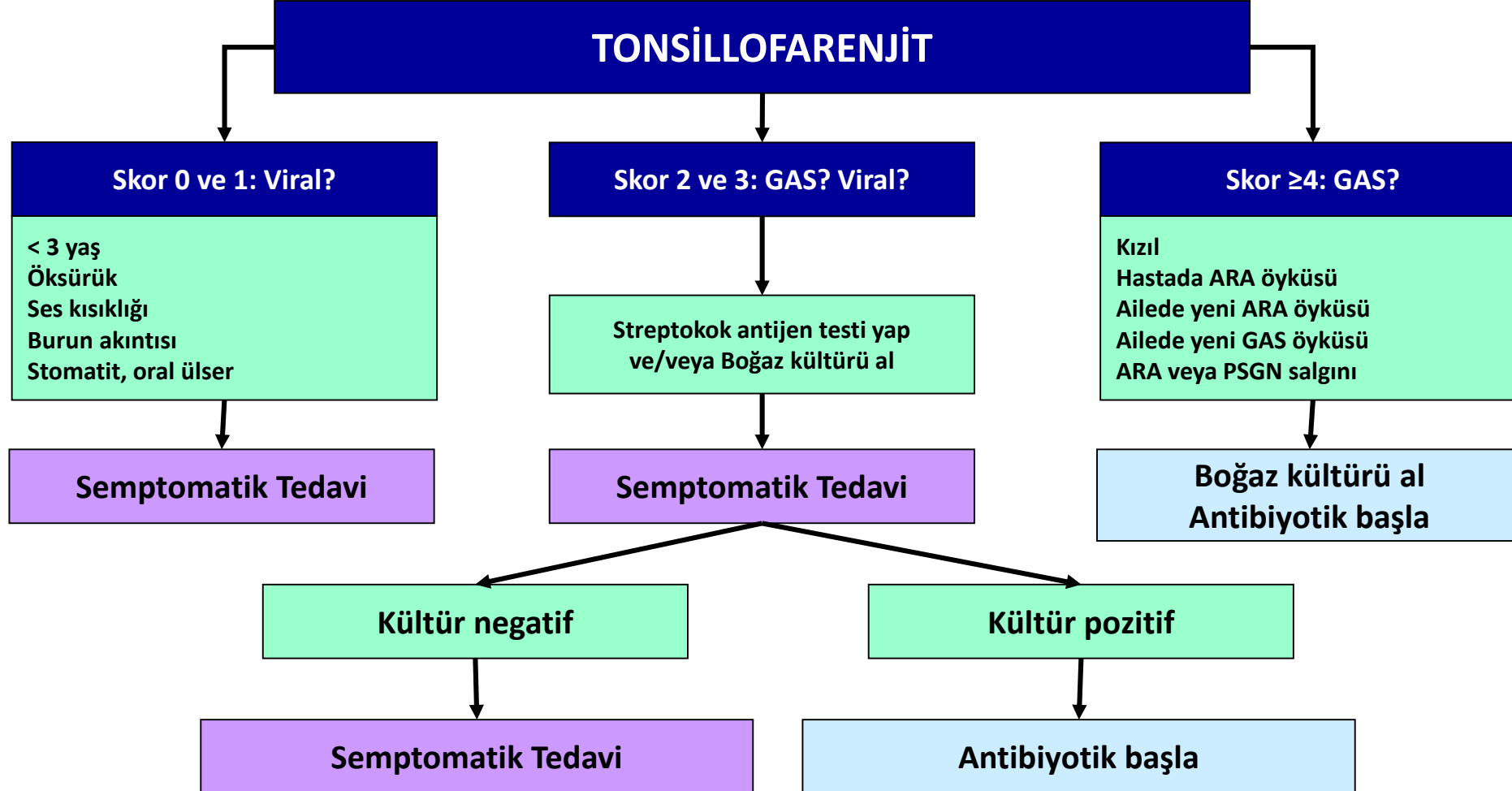
Her birine 1 puan ver.

3-14 yaş 1 puan ekle
 ≥ 45 yaş 1 puan çıkar

0-1 Puan Test ve antibiyotik gereksiz
2-3 Puan Test yap, sonucuna göre tedavi ver
4-5 Puan Test yapmadan antibiyotik ver

AKUT TONSİLLOFARENJİT

Antibiyotik Başlama Kararının Verilmesi



Klinik GAS düşündürüyor ve kültür olanağı yoksa antibiyotik başla

AKUT TONSİLLOFARENJİT

Tedavi

TEDAVİ				
Antibiyotik	Doz	Doz sayısı	Veriliş yolu	Süre
<i>Benzatin penisilin G</i>	600 000 Ü (≤27 Kg) 1 200 000 Ü (>27 Kg)	1	IM	Tek doz
<i>Penisilin V</i>	250 mg (400 000 Ü)/doz (≤27 Kg) 500 mg (800 000 Ü) /doz (>27 Kg)	2-3	Oral	10 gün
<i>Amoksisilin</i>	50 mg/kg/doz (en çok 1000 mg/doz) 25 mg/kg/doz (en çok 500 mg/doz)	1 2	Oral	10 gün

AKUT TONSİLLOFARENJİT

Epstein-Barr Virüs Enfeksiyonu

TONSİLLOFARENJİT



AKUT TONSİLLOFARENJİT

PFAPA Sendromu

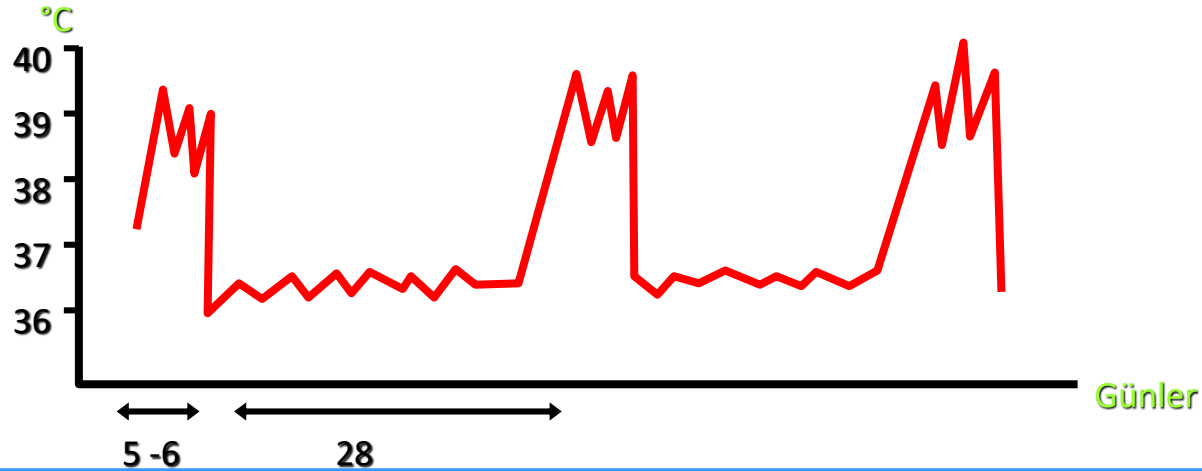
TONSİLLOFARENJİT

Türk Pediatri Arşivi 2004; 39: 36- 40

Periyodik ateş, aftöz stomatit, farenjit ve servikal adenit (PFAPA) sendromlu bir olgu

Ergin Çiftçi, Halil Özdemir, Sonay İncesoy, Erdal İnce, Ülker Doğru

ATEŞ ÇİZELGESİ



AKUT TONSİLLOFARENJİT

GAS

TONSİLLOFARENJİT

ERKEN KOMPLİKASYONLAR

Servikal lenfadenit

Peritonsiller apse

Parafarengeal apse

Retrofarengeal apse

Akut otitis media

Akut sinüzit

Bronkopnömoni

Menenjit

Beyin apsesi

Septik artrit

Osteomyelit

Endokardit

Sellülit

Nekrotizan fasiit

Bakteriyemi

Streptokoksik toksik şok sendromu

TONSİLLOFARENJİT

GEÇ KOMPLİKASYONLAR

Akut romatizmal ateş

Poststreptokoksik glomerülonefrit

Poststreptokoksik reaktif artrit

PANDAS

(Pediatric Autoimmune Neuropsychiatric Disorders

Associated with Streptococcal Infections)

DERİN BOYUN ENFEKSİYONLARI

Etiyoloji ve Epidemiyoloji

EPİDEMİYOLOJİ

ABD’de yılda 30/100 000 peritonsiller apse

**Pittsburg Çocuk Hastanesi
1986-1992 arasında 117 derin boyun
enfeksiyonu**

**%49 peritonsiller enfeksiyon
%22 retrofaringeal enfeksiyon
%2.5 parafaringeal enfeksiyon**

**Peritonsiller enfeksiyonlar geç çocukluk ve
adölesanlarda sık**

**Retrofaringeal enfeksiyon erken çocuklukta
sık
Retrofaringeal lenf nodlarında 4 yaşından
sonra atrofisi**

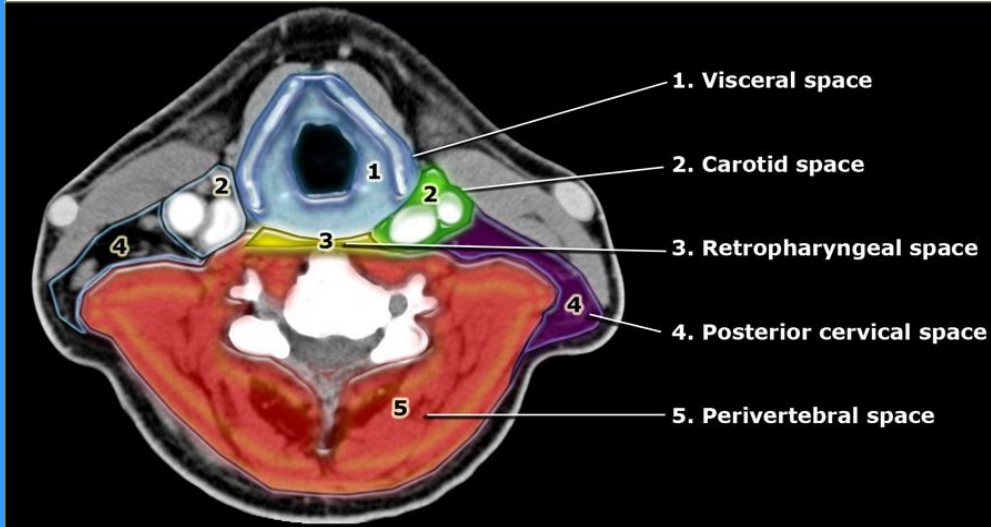
ETİYOLOJİ

**Tonsiller ve faringeal enfeksiyonlar
Dental enfeksiyonlar
Otit
Sinüzit
Tükrük bezi enfeksiyonları
Oral cerrahi girişimler
Oral kavite ve farenksin delici travmaları
Yabancı cisim aspirasyonu
Servikal lenfadenit
Özafagoskopi ve bronkoskopi
Tiroidit
Komşu kemik enfeksiyonları
Konjenital nedenler
**Brankial yarık anomalileri
Tiroglossal kist****

DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

DERİN BOYUN BOŞLUKLARI



DERİN BOYUN BOŞLUKLARI

Peritonsiller apse
Retrofaringeal apse

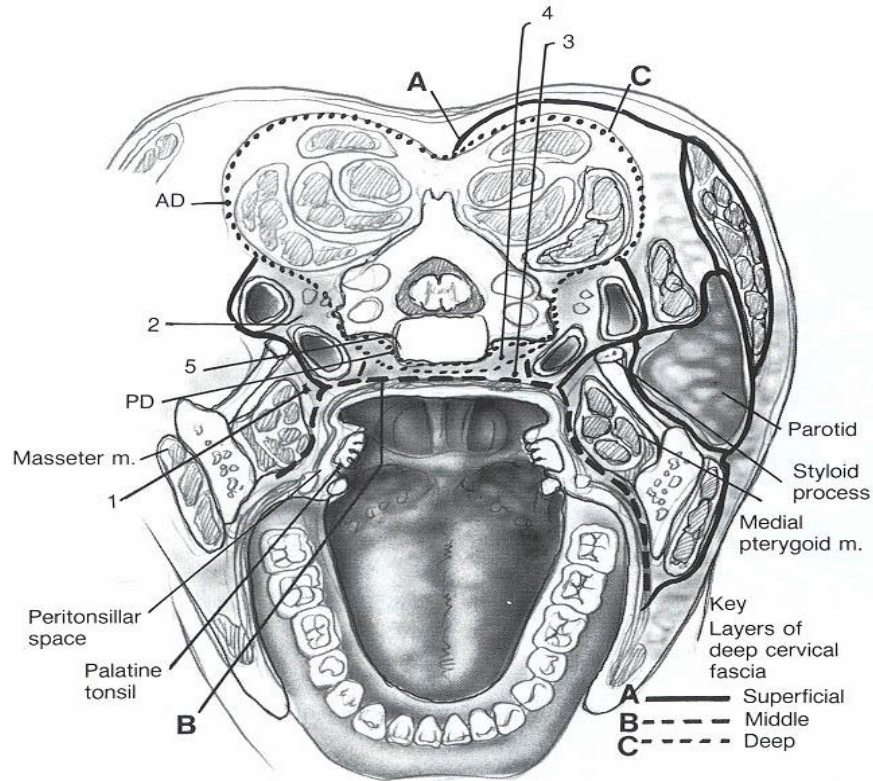
Parafaringeal apse

Prevertebral apse
Visseral vasküler alan apsesi
Submandibüler apse
Submental apse
Sublingual apse
Mastikattör alan apsesi
Pretrakeal apse

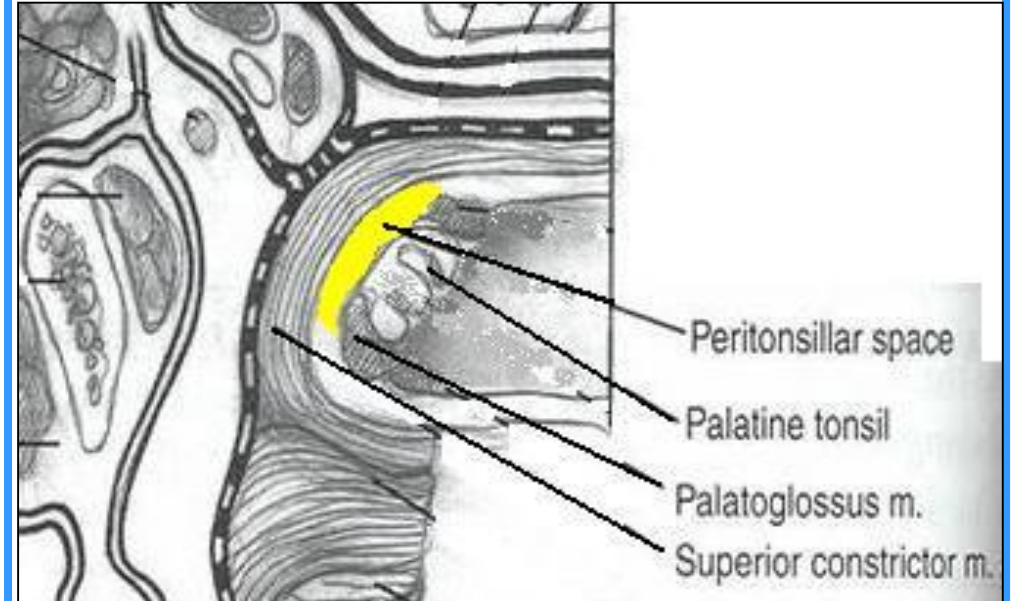
DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

PERİTONSİLLER BOŞLUK



PERİTONSİLLER BOŞLUK



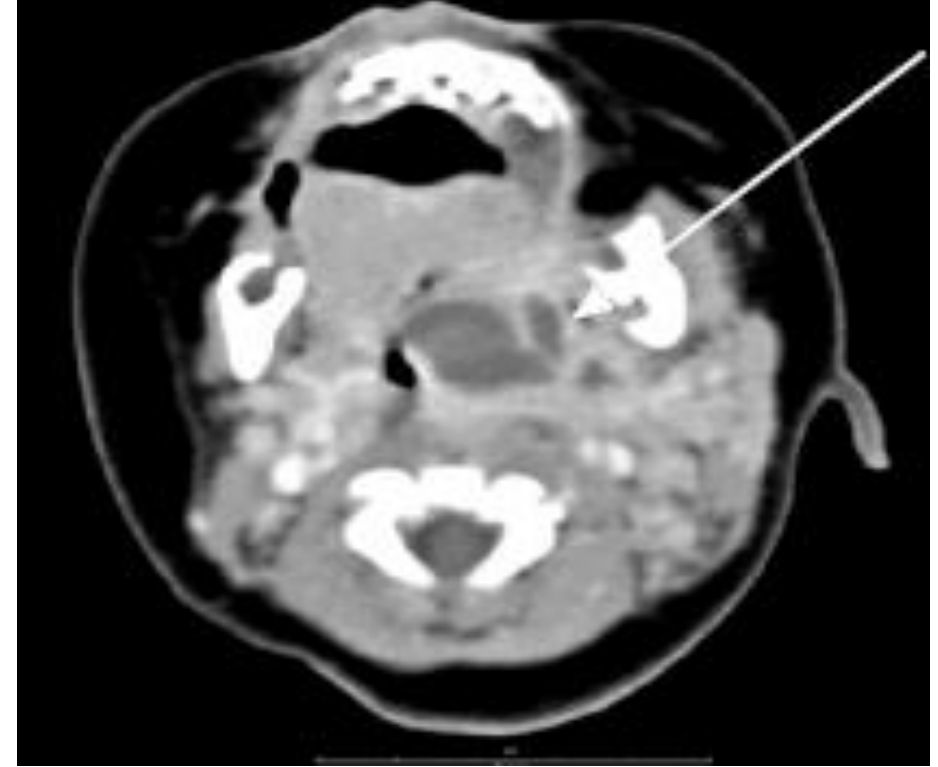
DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

PERİTONSİLLER ABSE



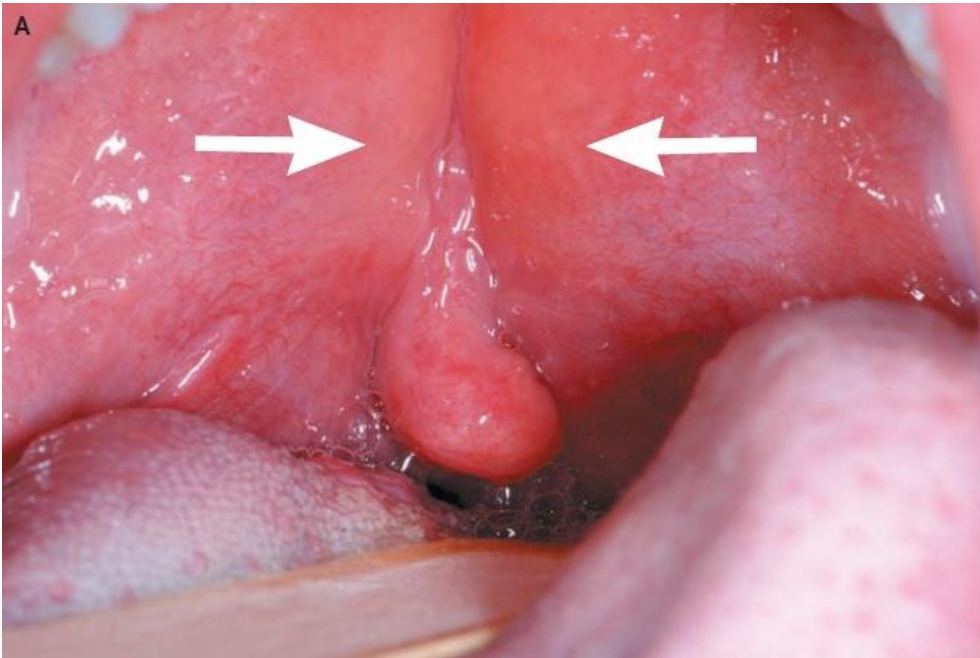
PERİTONSİLLER ABSE



DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

PERİTONSİLLER ABSE



PERİTONSİLLER ABSE

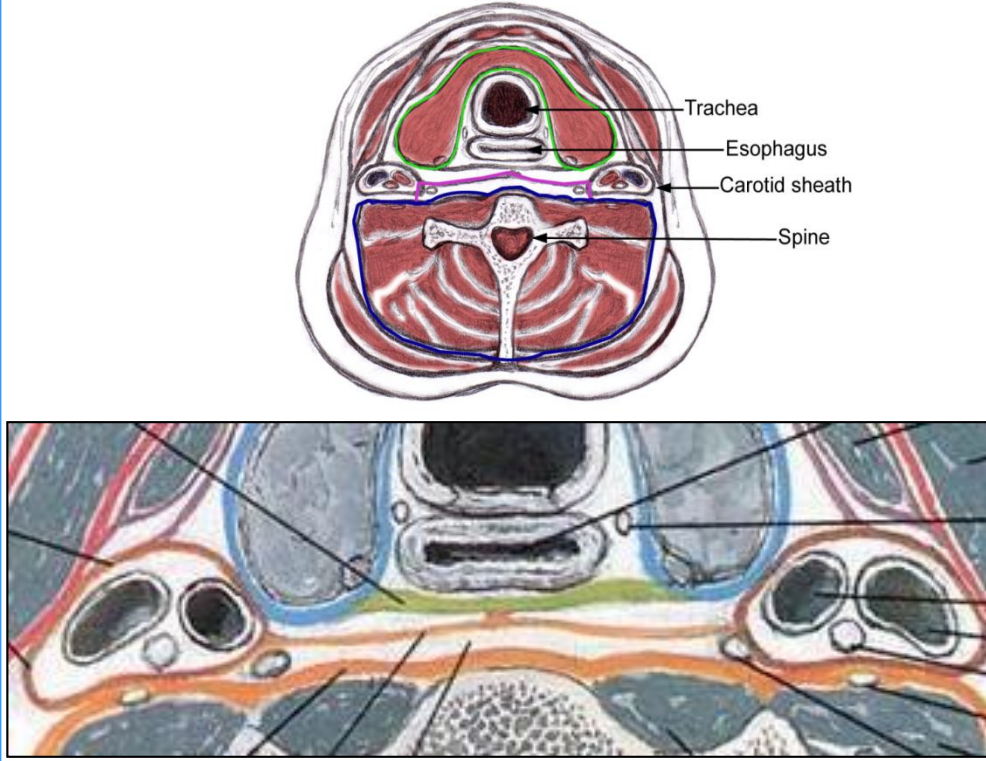


Fiechtl JF, Stack LB.
Bilateral peritonsillar abscesses.
N Engl J Med 2008; 358: e27.

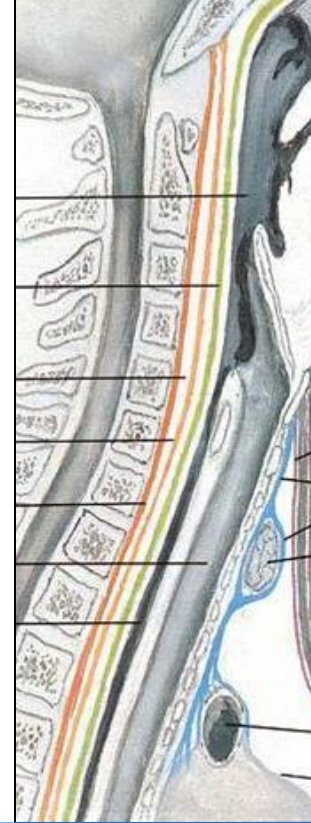
DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

RETROFARİNGEAL BOŞLUK



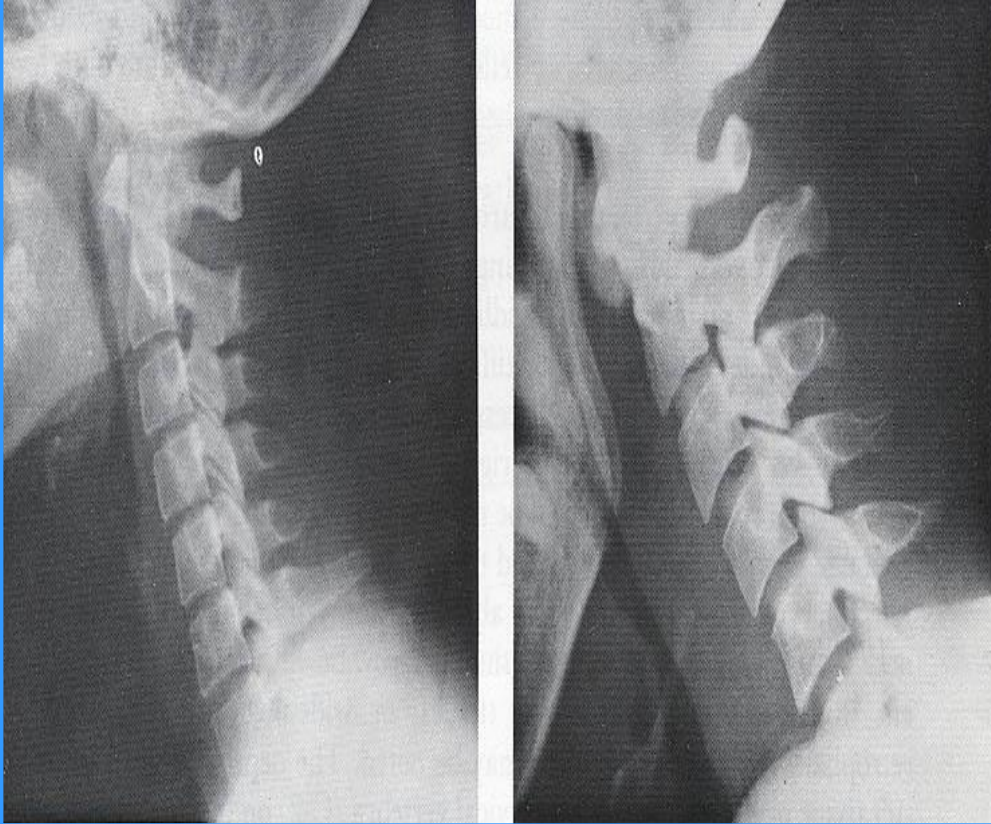
RETROFARİNGEAL BOŞLUK



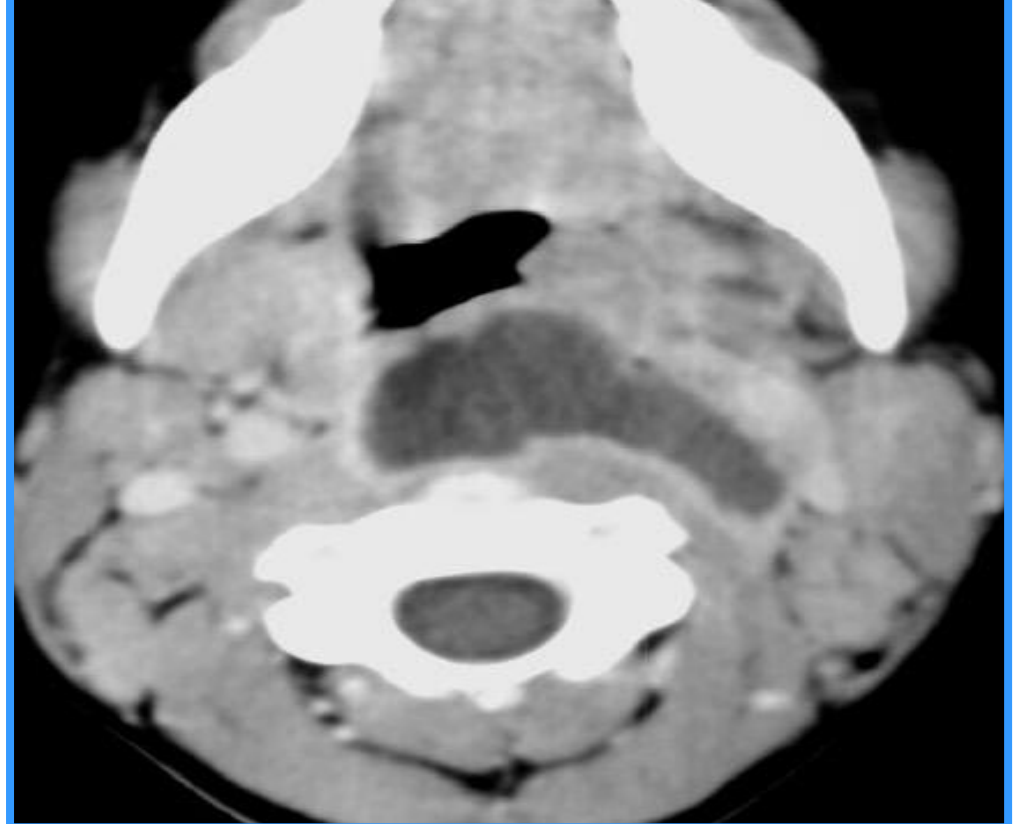
DERİN BOYUN ENFEKSİYONLARI

Derin Boyun Boşlukları

RETROFARENGEAL APSE



RETROFARENGEAL APSE



DERİN BOYUN ENFEKSİYONLARI

Semptom ve Bulgular

SEMPTOMLAR

Ateş
Halsizlik
İştahsızlık

Boğaz ağrısı
Yutma güçlüğü
Boğuk sesle konuşma
Ağzını tam açamama
Boyun hareketlerinde ağrı
Boyun ağrısı
Boyunda şişlik
Ses kısıklığı

Nefes darlığı

BULGULAR

Orta-şiddetli toksik görünüm
Ağızda kötü koku
Tonsillerde hiperemi, hipertrofi, kript
Tonsil ve uvulada itilme
Farinks arka duvarında şişlik
Farinks yan duvarında şişlik
Boyun hareketlerinde kısıtlılık
Ağrılı servikal LAP
Boyunda şişlik ve fluktuasyon
Trismus
Tortikollis

Solunum sıkıntısı

DERİN BOYUN ENFEKSİYONLARI

AÜTF Çocuk Enfeksiyon Deneyimi

DERİN BOYUN ENFEKSİYONLARI

Çocuklarda Derin Boyun Enfeksiyonları

Deep Neck Infections in Children

Nurşen Belet, Anıl Tapırsız, Yetiş Uçar, Ergin Çiftçi, Suat Fitöz*, Erdal İnce, Ülker Doğru
Ankara Üniversitesi Tıp Fakültesi Çocuk Sağlığı ve Hastalıkları Anabilim Dalı, Çocuk Enfeksiyon Hastalıkları Bilim Dalı
*Radyoloji Anabilim Dalı, Ankara, Türkiye

Özet

Amaç: Ocak 2000-Nisan 2007 arasında Ankara Üniversitesi Tıp Fakültesi Çocuk Enfeksiyon Hastalıkları Servisine kabul edilen derin boyun enfeksiyonlu hastaların klinik, mikrobiyolojik ve radyolojik özelliklerini ve tedavi sonuçlarını değerlendirmek.

Gereç ve Yöntem: Derin boyun enfeksiyonlu hastaların kayıtları demografik özellikler, klinik özellikler, mikrobiyolojik ve görüntüleme bulguları ve komplikasyonları açısından retrospektif olarak değerlendirildi.

Bulgular: Bu dönemde toplam derin boyun enfeksiyonu tanısı alan 12 hasta izlendi. Parafaringeal enfeksiyon en sık görülen (%75) derin boyun enfeksiyonu tipi idi. Ampisilin-sulbaktam tek başına veya klindamisin ile birlikte olmak üzere hastalara en sık verilen antibiyotik tedavisi idi. Antibiyotik tedaviye yanıt vermeyen üç hastaya (iki peritonsiller apse, bir retrofaringeal-parafaringeal apse) cerrahi drenaj uygulandı. On hasta (%83) yalnız antibiyotik tedavi ile düzeldi. Hastaların taburcu olduktan sonra iki ay ile 5 yıl arasındaki izlemünde mortalite, komplikasyon veya rekürrens görülmedi.

Sonuç: Derin boyun enfeksiyonlu hastaların çoğu intravenöz antibiyotikler ile tedavi edilebilir, fakat klinik düzelme yoksa cerrahi drenaj yapılması gerekmektedir. (Çocuk Enf Derg 2007; 1: 58-62)

Anahtar kelimeler: Derin boyun enfeksiyonları, peritonsiller apse, parafaringeal apse, retrofaringeal apse

Summary

Aim: To evaluate the clinical, microbiological and radiological findings and treatment results of patients with deep neck infections admitted to the Pediatric Infectious Diseases Clinics of Ankara University Medical School between January 2000 and April 2007.

Materials and Methods: The data of patients with deep neck infection was analyzed retrospectively for demographics, clinical features, microbiological and radiological findings and complications.

Results: During this period a total of 12 patients were followed and parapharyngeal infection was the most common deep neck infection (75%). Frequently intravenous ampicillin/sulbactam alone or in combination with clindamycin were given to the patients. Surgical drainage was performed in three patients unresponsive to antibiotic treatment (two peritonsillar abscess, one retropharyngeal/parapharyngeal abscess). Ten patients recovered with antibiotic treatment alone (83%). No mortality, complication or recurrence was observed during 2 months to 5 years after discharge.

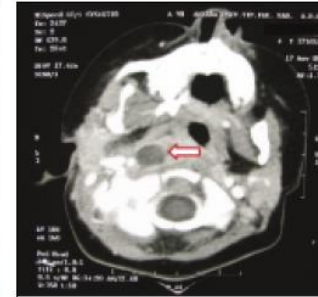
Conclusion: Most of the patients with deep neck infections can be treated with intravenous antibiotics, however if there is no clinical improvement, surgical drainage is required. (J Pediatr Inf 2007; 1: 58-62)

Key words: Deep neck infections, peritonsillar abscess, parapharyngeal abscess, retropharyngeal abscess

DERİN BOYUN ENFEKSİYONLARI

Olgu no	BT bulguları	Antibiyotik tedavisi	Cerrahi drenaj	Hastanede kalış süresi	Antibiyotik tedavi süresi (Parenteral/toplam)
1	-	Amp-sulb*	Evet	7	7/14
2	-	Amp-sulb	-	6	6/10
3	-	Amp-sulb + klindamisin	Evet	6	6/10
4	Retrofaringeal-parafaringeal apse	Amp-sulb + klindamisin	-	10	10/21
5	Retrofaringeal-parafaringeal apse	Amp-sulb + klindamisin	Evet	10	10/15
6	Retrofaringeal-parafaringeal apse	Amp-sulb + klindamisin	-	12	12/30
7	Parafaringeal apse	Amp-sulb + klindamisin	-	9	9/21
8	Parafaringeal apse	Seftriakson + klindamisin	-	17	17/24
9	Parafaringeal apse	Amp-sulb + klindamisin	-	9	9/38
10	Parafaringeal sellülit	Amp-sulb	-	8	8/14
11	Parafaringeal apse	Amp-sulb + klindamisin	-	16	16/23
12	Parafaringeal sellülit	Amp-sulb + klindamisin	-	7	7/12

*Ampisilin-sulbaktam



Belet N, Tapırsız A, Uçar Y, Çiftçi E, Fitöz S, İnce E, Doğru Ü.
Çocuklarda derin boyun enfeksiyonları.
Çocuk Enf Derg 2007; 1: 1-5.

DERİN BOYUN ENFEKSİYONLARI

Etkenler

MİKROBİYOLOJİ

AEROB

Streptococcus pyogenes

Staphylococcus aureus

Haemophilus influenzae

MİKROBİYOLOJİ

ANAEROB

Bacteroides

Prevotella

Fusobacterium

Peptostreptococcus

DERİN BOYUN ENFEKSİYONLARI

Komplikasyonlar

KOMPLİKASYONLAR

Hava yolu obstrüksiyonu

**Apsenin spontan veya muayene sırasında
hava yoluna açılması ve aspirasyon
pnömonisi**

KOMPLİKASYONLAR

**Diğer derin boyun kompartmanlara yayılım
Nekrotizan fasiit
Mediastinit, ampiyem
Retroperitoneal enfeksiyon
Karotis arter trombozu, erezyonu, yırtılması,
→hemipleji**

**İnternal juguler ven trombozu
Lateral ve kavernöz sinüs trombozları
Septik trombüs embolileri ve çoklu organ
apseleri
Kranial sinir tutulumu (IX,X,XI,XII)
Yumuşak damak, larinks, dil paralizileri
Horner sendromu
Septisemi**

DERİN BOYUN ENFEKSİYONLARI

Tedavi

TEDAVİ

Hospitalizasyon
Hava yolu açıklığı
Analjezik
İV antibiyotik
 Ampisilin-sulbaktam
 Klindamisin
 Sefaperazon-sulbaktam
 Piperasilin-tazobaktam
 Tikarsilin-klavulonat
 Sefalosporin+klindamisin/metranidazol
 Karbapenem
KBB konsültasyonu
Cerrahi drenaj
İğne aspirasyonu
İnsizyon
Akut-elektif tonsillektomi
Ağız içi drenaj
Eksternal drenaj

TEDAVİ



Loftis L.
Acute infectious upper airway obstructions in children.
Semin Pediatr Infect Dis 2006; 17:5-10.

The Eagle Effect Revisited: Efficacy of Clindamycin, Erythromycin, and Penicillin in the Treatment of Streptococcal Myositis

Dennis L. Stevens, Amy E. Gibbons,
Roberta Bergstrom, and Virginia Winn

*From the Infectious Disease Service, Department of
Medicine, Veterans Administration Medical Center, Boise,
Idaho; and the Department of Medicine, University of
Washington, Seattle, Washington*

We investigated the relative efficacies of penicillin, clindamycin, and erythromycin in a mouse model of myositis due to *Streptococcus pyogenes*. Penicillin was ineffective unless given at the time of bacterial injection, and treatment delays of 2 h reduced its efficacy such that survival was no better than that of untreated control animals ($P > .05$). Survival of erythromycin-treated mice was greater than that of both penicillin-treated mice and untreated controls, but only if treatment was begun within 2 h. Mice receiving clindamycin, however, had survival rates of 100%, 100%, 80%, and 70% even if treatment was delayed 0, 2, 6, and 16.5 h, respectively. Thus, clindamycin demonstrated superior efficacy to penicillin among all the various treatment groups ($P < .05$). Our results corroborate the failure of penicillin in this model of streptococcal infection and suggest that, unlike penicillin, the efficacy of clindamycin is not adversely altered by the "Eagle effect."

Dr. Harry Eagle Is Dead at 86; Formulated Cell-Growth Medium

By BRUCE LAMBERT

Dr. Harry Eagle, a medical scientist whose many discoveries included a method for growing cells in test tubes that was a breakthrough for biological research, died on Friday at United Hospital in Port Chester, N.Y., where he was admitted three weeks ago. He was 86 years old and lived in Monmouth, N.Y.

He died of cancer, said Arthur Ostrow, a spokesman for the Albert Einstein College of Medicine, from which Dr. Eagle was retired.

Dr. Eagle's career included executive posts at several major academic, research and government institutions. In 1967, President Ronald Reagan awarded him the National Medal of Science, the country's highest scientific honor.

His best-known achievement was his formulation in 1955 of the essential compounds needed to sustain the reproduction of human and other mammalian cells in the laboratory, a mixture that became known as Eagle's growth medium. Its development was

penicillin prevents gonorrhea when taken soon after exposure to infection.

Dr. Eagle published widely in scientific journals. Among his many honors, he won the Waterford International Biomedical Award and Eli Lilly Award in Bacteriology and citations from the American Association of Medical Colleges and the American Society of Cell Biology.

He was a past president of the Society of American Microbiologists, the American Association of Immunology and the Society of Experimental Biology and Medicine. He was also a former chairman of the Cold Spring Harbor Laboratory.

Dr. Eagle was born in New York City and grew up in Baltimore. He graduated from Johns Hopkins University in 1925 and its medical school in 1927.

A Career in Teething

For most of the next 20 years he did teaching and research at Johns Hopkins. He was the director of its Venereal Disease Research Laboratory and Laboratory of Experimental Therapeutics. He was also a commanding officer in the United States Public Health Service program at Johns Hopkins.

From 1947 until 1963, Dr. Eagle worked at the National Institutes of Health. He served as the scientific director of the National Cancer Institute from 1947-63, then chief of the Experimental Therapeutics Section of the National Microbiological Institute from 1963-70 and chief of the Cell Biology Laboratory of the National Institute of Allergy and Infectious Diseases from 1970-81.

He joined the Albert Einstein College of Medicine in the Bronx in 1981 and became a professor and chairman of its cell biology department, founding chairman of its Biological Sciences Division, associate dean for Scientific Affairs and founding director of its Cancer Research Center.

He retired as the center's director in 1988 but remained active until his illness last year.

Surviving are his wife of 44 years, the former Jean Lane; three sons, W. Thomas Jr. of Berkeley, Calif.; John C. of Lafayette, Calif.; and Peter E. of



Dr. Harry Eagle

Walter Wells Sr., 64, Insurance Executive

Walter Thomas Wells Sr., who was a leading executive in the marine insurance industry, died on Friday at his home in Moraga, Calif. He was 64 years old.

He died of cancer, his family said.

Mr. Wells retired earlier this year as the senior vice president and longest-serving employee of the Marine Office of America Corporation, a division of Continental Insurance and the largest marine insurance company in North America.

Born in Brooklyn, Mr. Wells grew up in Ridgewood, N.J. He started at Marine as an underwriter trainee in 1950 at its Manhattan office. He took a leave to study at the New York State Maritime Academy and after graduation served as a lieutenant in the United States Navy from 1952 to 1954.

After returning to Marine, he held a series of posts, including senior ocean cargo underwriter, vice president of business production at the home office and director of recruiting and training.

Surviving are his wife of 42 years, the former Jean Lane; three sons, W. Thomas Jr. of Berkeley, Calif.; John C. of Lafayette, Calif.; and Peter E. of

Clindamycin in the treatment of group G β -haemolytic streptococcal infections

A. Pillai^a, S. Thomas^{b,*}, C. Williams^a

^aDumfries and Galloway Royal Infirmary, Dumfries, DG1 4AP, UK

^bWycombe Hospital, Buckinghamshire, UK

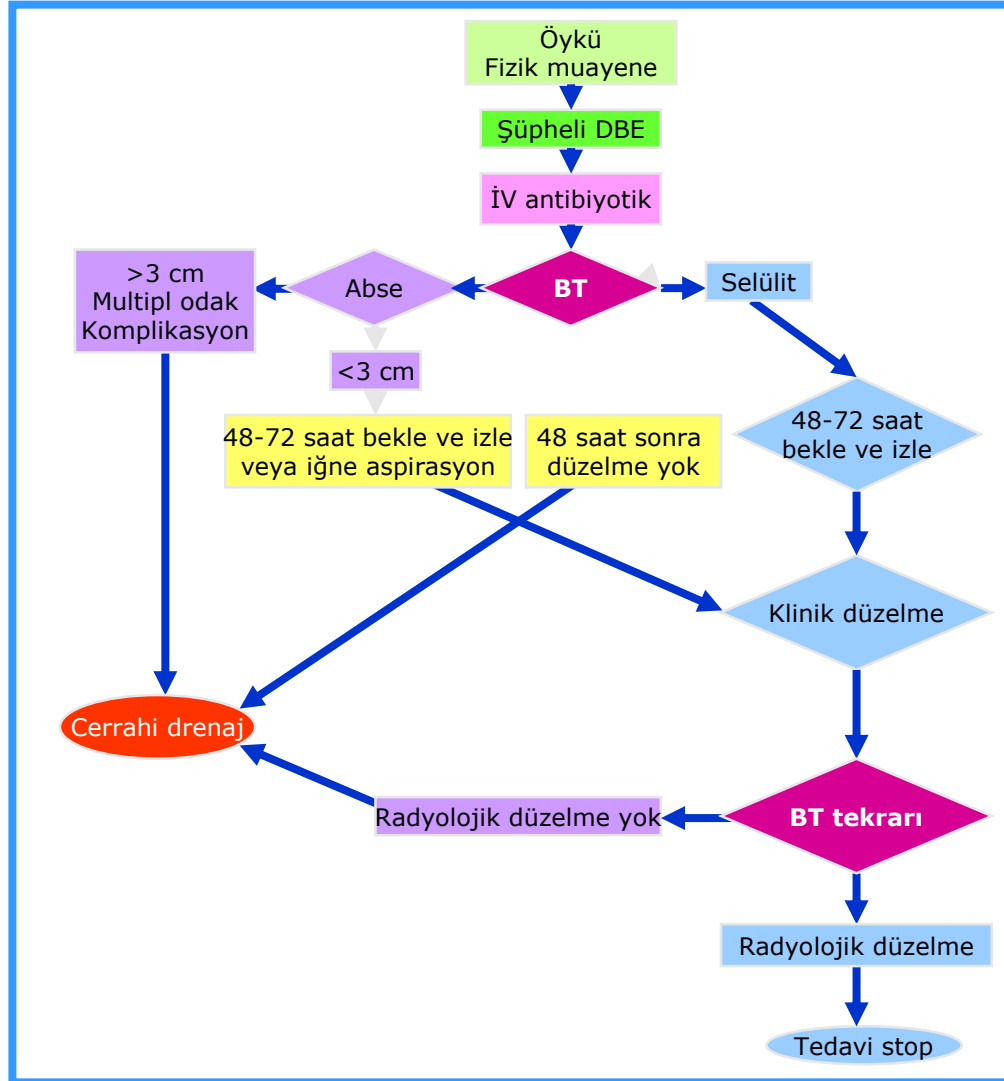
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Abstract We report a case of severe streptococcal cellulitis in a healthy 47 year old male, where the sole microbial isolate was a β -haemolytic group G *Streptococcus*. Treatment failure with high dose penicillin was observed despite in vitro sensitivity. The addition of clindamycin resulted in dramatic clinical improvement. This may indicate an Eagle-type effect (whereby antibiotics exhibit paradoxically reduced bactericidal activities at high drug concentrations), in group G β -haemolytic infections. Although well documented with group A streptococcal infections, this phenomenon has not been fully recognised with group G β -haemolytic streptococcal infections. This may have important implications for clinical management.

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