



Kocaeli Üniversitesi Tıp Fakültesi Çocuk Sağlığı ve Hastalıkları Anabilim Dalı

Onkoloji Bilim Dalı
Olgu Sunumu

28 Nisan 2016 Perşembe

İnt. Dr. Erdi Karahan

Prof. Dr. Funda Çorapçıoğlu



KOCAELİ ÜNİVERSİTESİ TIP FAKÜLTESİ ÇOCUK SAĞLIĞI VE HASTALIKLARI ANABİLİM DALI

ÇOCUK ONKOLOJİ BİLİM DALI OLGU SUNUMU

İNT. DR. ERDİ KARAHAN

28 NİSAN 2016

11 YAŞINaDA KIZ HASTA

- **ŞİKAYET** : Gülememe, görmede azalma, boyunda kitle
- **ÖYKÜSÜ** : Nisan 2015'te akalazya nedeniyle opere edilen hastanın operasyondan 1 ay sonra uyku haliyle birlikte güçsüzlük, gözlerde pitoz, görmede bulanıklık şikayetleri başlamış. Dış merkezde muayenede herhangi bir patoloji saptanmamış. Çocuk psikiyatrisine yönlendirilmiş. Aile daha sonra çocuk nörolojisine başvurmuş, çekilen kranium mr'da boyun bölgesinde görülen laplar o esnada saçlı derideki zonaya bağlanmış. Daha sonra göz doktoruna başvurmuşlar. Gözlerde midriyazis saptanan hasta çocuk onkolojiye yönlendirilmiş. Fakat hasta kontrole gelmemiş. Boynundaki şişlik son 1 aydır varmış.

- **ÖZGEÇMİŞ** : özellik yok

1. *Çocuk* : 13 yaş erkek SS
2. *Çocuk* : Hastamız
3. *Çocuk* : 4 yaş erkek SS

- **SOYGEÇMİŞ** : Anne 31 yaş, SS

Baba 40 yaş, SS

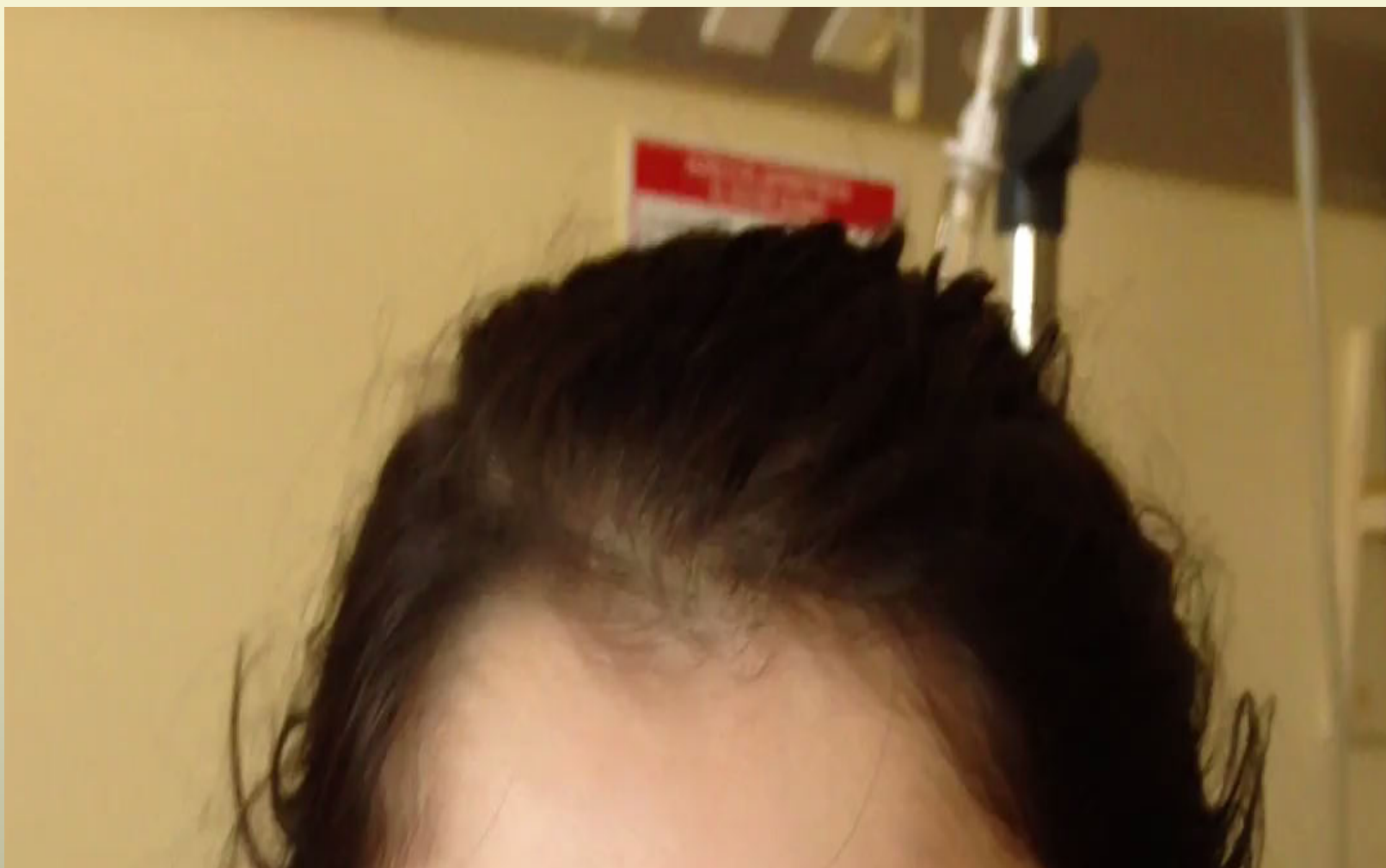
Ailede nörolojik hastalık öyküsü yok.

VİTAL BULGULAR

- Ateş : 36.7 C
- Solunum : 22 /dk
- Nabız : 78 /dk
- Kan Basıncı : 110/70 mmHg
- sPO2 : %100

FİZİK MUAYENE

- Boyun sağ tarafta 5x5 cm boyutlarında sert, fikse kitle.
- Bilateral pitozis, midriyazis. Işık refleksleri zayıf.
- Sağ fasiyal paralizi.
- AC sesleri doğal. Ral-ronküs yok.
- S1S2 + . Ek ses yok. Üfürüm yok.
- Batın rahat. Defans-rebound yok.
- Kas gücü 4 ekstremitede 5/5
- DTR normoaktif. Patolojik refleks yok.

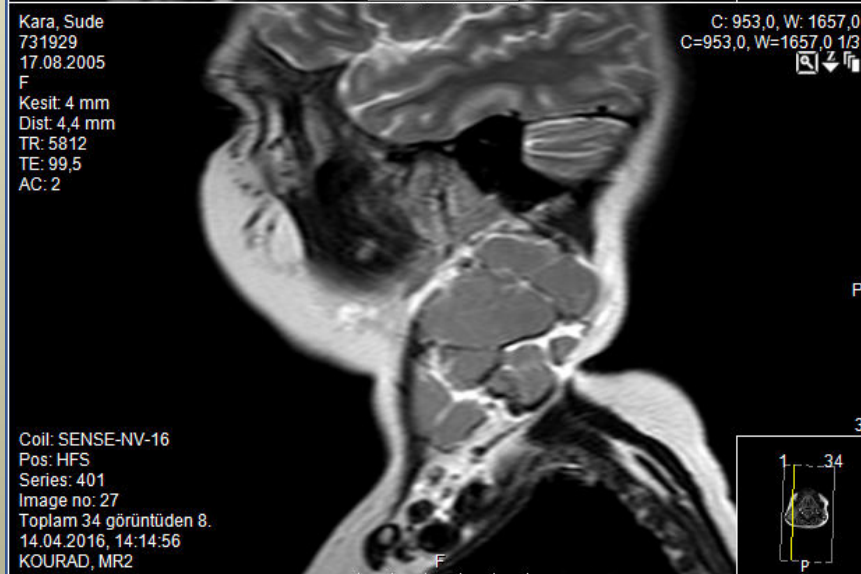
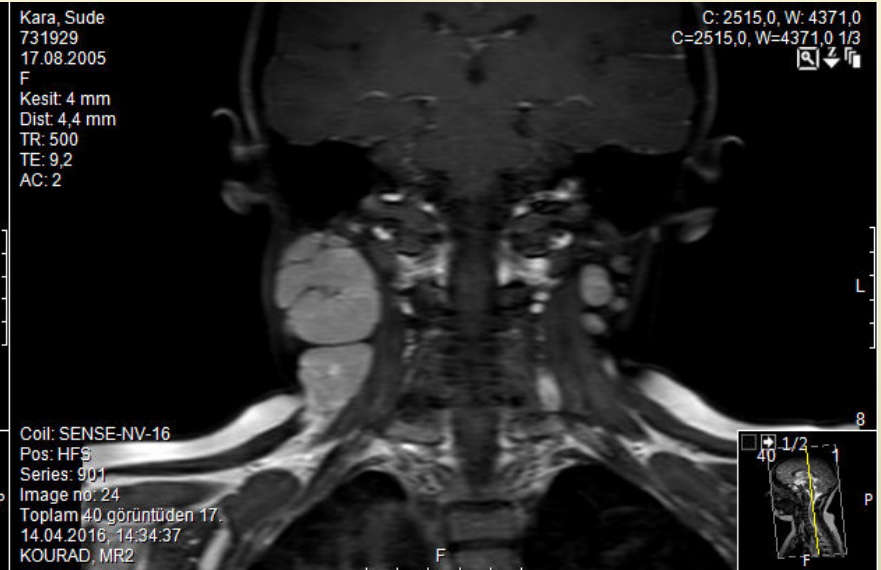


LABORATUVAR

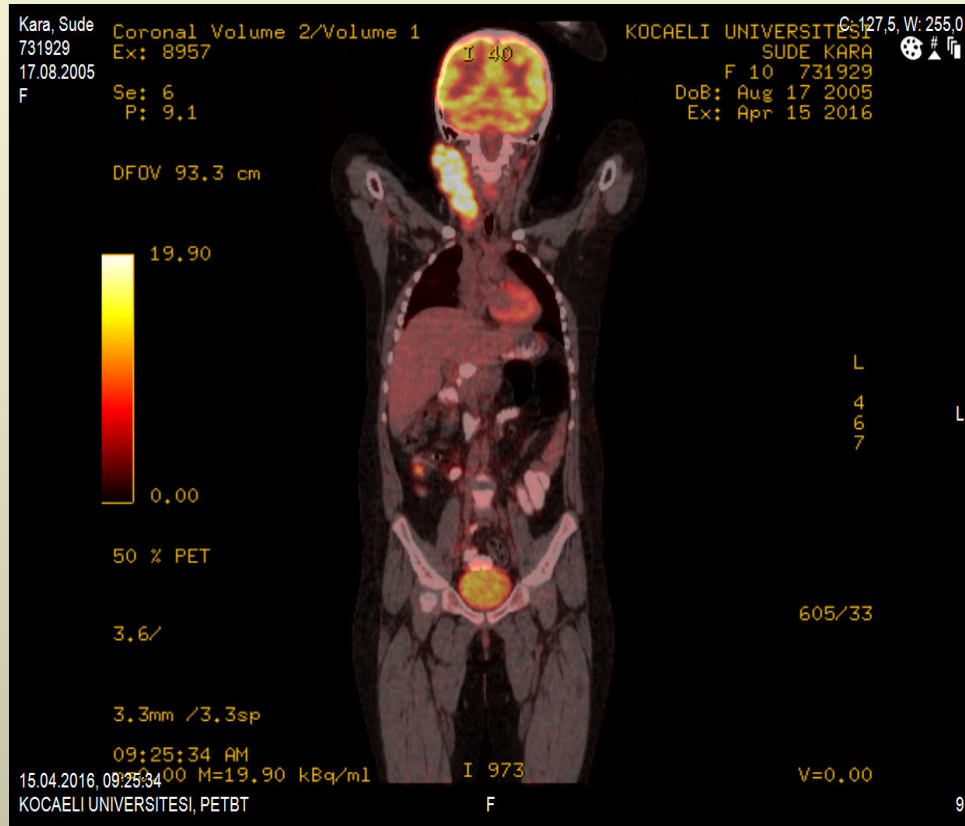
Rutin Biyokimya		
GLUKOZ(AKS)	115	▲ 74 - 106 (mg/dL)
ÜREA	21	16.6 - 48.5 (mg/dL)
BUN	10.0	6 - 20 (mg/dL)
KREATİNİN	0.57	(mg/dL)
CKD-EPI	143.2	(mL/dk/1.73m ²)
MDRD	152.7	(mL/dk/1.73m ²)
BUN / KREATİNİN ORANI	17.54	12 - 20 ()
TOTAL BİLİRUBİN	0.22	< 1.2 (mg/dL)
DİREKT BİLİRUBİN	0.12	< 0.30 (mg/dL)
İNDİREKT BİLİRUBİN	0.10	< 0.9 (mg/dL)
AST (SGOT)	32	(U/L)
ALT (SGPT)	30	(U/L)
GGT	11	(U/L)
LDH	313	(U/L)
ALKALEN FOSFATAZ	158	(U/L)
TOTAL PROTEİN	7.4	6.6 - 8.7 (g/dL)
ALBUMİN	4.8	3.97 - 4.94 (g/dL)
GLOBULİN	2.6	1.1 - 3.5 (g/dL)
ALBUMİN / GLOBULİN ORANI	1.8	0.8 - 2 (g/dL)
KALSİYUM FOSFOR ORANI	37.7	()
DÜZELTİLMİŞ KALSİYUM	8.6	()
DÜZELTİLMİŞ SODYUM	141.2	()
SODYUM	141	136 - 145 (mEq/L)
POTASYUM	4.13	3.5 - 5.1 (mEq/L)
KLOR	102	98 - 107 (mEq/L)
KALSİYUM	9.2	8.6 - 10.2 (mg/dL)
MAGNEZYUM	2.02	1.6 - 2.6 (mg/dL)
İNORGANİK FOSFOR	4.4	2.5 - 4.5 (mg/dL)
OZMOLARİTE(SERUMDA)	292	275 - 300 (mOsm/L)

Hematolojik Tetkikler		
PERİFERİK YAYMA	İADE	()
SEDİMENTASYON	13	(mm/h)
WBC	14.4	▲ 4.60 - 10.2 (x 10³/L)
NEU	10.700	▲ 2.00 - 6.90 (x 10³/L)
NEU%	74.400	37.0 - 80.0 (%)
LYM	2.840	0.600 - 3.40 (x 10 ³ /L)
LYM%	19.700	10.0 - 50.0 (%)
MONO	0.542	0.00 - 0.900 (x 10 ³ /L)
MONO%	3.760	0.00 - 12.0 (%)
EOS	0.230	0.00 - 0.700 (x 10 ³ /L)
EOS%	1.590	0.00 - 7.00 (%)
BASO	0.074	0.00 - 0.200 (x 10 ³ /L)
BASO%	0.514	0.00 - 2.50 (%)
RBC	4.27	4.04 - 6.19 (x 10 ⁶ /uL)
HGB	11.6	▼ 12.2 - 18.1 (g/dL)
HCT	36.5	▼ 37.7 - 53.7 (%)
MCV	85.5	80.0 - 97.0 (fL)
MCH	27.200	27.0 - 31.2 (pg)
MCHC	31.800	31.8 - 35.4 (g/dL)
PLT	320	142 - 424 (x 10 ³ /uL)
MPV	5.760	0.00 - 99.9 (fL)
PCT	0.184	(%)
PDW	16.200	()
RDW	15.200	12.2 - 17.2 ()
Türbidometrik Tetkikler		
CRP	0.12	< 0.5 (mg/dl)

GÖRÜNTÜLEME



PET



SONUÇ ve YORUM:

- Sağ servikal bölgede supraklavikular bölgeye uzanan malignite düzeyinde yoğun artmış metabolizma gösteren konglomere lenfadenopati izlendi (metastaz?/primer?). Histopatolojik doğrulama önerilir.
- Sol üst arka üçgen (5A) lenfatik lojda artmış metabolizma gösteren birkaç adet lenfadenopati, metastaz ile uyumlu değerlendirildi.
- Adenoidal bölgede diffüz artmış metabolizma gözlemlendi (fizyolojik?/primer?). Klinik, gereklilik halinde histopatolojik değerlendirme önerilir.
- Her iki akciğerde metabolizma göstermeyen ve hafif artmış metabolizma gösteren nodüller izlendi (metastaz?).

ÖN TANILARINIZ ?

ÖZET

- Boyunda kitle
- Bilateral pitozis, midriyazis, ışık refleksi - / -
- Sağ fasiyal paralizi
- Akalazya

- Kafa tabanı tutulumu ve LN metastazı ile prezente nazofarenks ca.
- Non-Hodgkin lenfoma SSS tutulumu.

Bu noktadan sonra ne (neler) istersiniz ?

- Nazofarenks , boyun, kranium MR : sağ servikal bölgede birden fazla lenfadenopati.
- PET : artmış metabolizma gösteren lenfadenopati.
- Diagnostik LP : hücre görülmedi.
- K. iliği aspirasyonu : anormal hücre görülmedi.
- LN biyopsisi : **Hodgkin Lenfoma**

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Achalasia of the esophagus associated with Hodgkin disease.

Peeples WJ, El-Mahdi AM, Rosato FE.

Abstract

Esophageal achalasia has not been reported in the literature as a complication of Hodgkin disease. Involvement of the esophagus is rare but when present, consists mostly of bleeding, ulceration or diverticuli. A case is reported in a fifty-one year old female with achalasia of the distal third of the esophagus associated with Hodgkin disease, Stage II B.

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[Allgrove syndrome (triple A). Finding of a mutation not described in the AAAS gene].

[Article in Spanish]

Capataz Ledesma M¹, Méndez Pérez P, Rodríguez López R, Galán Gómez E.

Author information

Abstract

Allgrove syndrome (triple A) is a rare autosomal recessive disease. The classic triad includes, congenital adrenal insufficiency due to ACTH resistance, achalasia of the cardia and alacrimia. Neurological abnormalities are associated with autonomic neuropathy, sensory and motor defects, deafness, mental retardation, Parkinsonism and dementia. The gene responsible is the ADRACALIN or AAAS encoding a protein called ALADIN. We report a case of a 19 year-old male, assessed when he was 10 years old in our department due to suspected storage disease. Mild mental and language retardation, hypernasal voice, sensory-motor neuropathy with autonomic involvement and signs of spastic paraparesis, alacrimia, gastroesophageal reflux, and achalasia. Molecular studies showed to mutations, the undescribed p.Tyr 19 Cys, and IVS14 +1 G.

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- + Hodgkin Lenfoma

➡ Tripple A (Allgrove) Sendromu?

➡ Hodgkin lenfoma

Paraneoplastik nörolojik tutulum
+ İnsidental Akalazya

> Paraneoplastik tutulumla bağlı akalazya

TRİPLE A (ALLGROVE) SENDROMU

- İlk kez 1978 yılında tanımlanan triple A sendromu seyrek görülen, otozomal resesif, ACTH dirençli adrenal yetmezlik, alakrimea ve akalazya ile karakterize bir hastalıktır.*1
- Ek olarak çeşitli nörolojik problemler santral, periferik ve otonomik sinir sistemini etkileyebilir, dermatolojik bulgular görülebilir.*2
- Bu sendromdan sorumlu gen kromozom 12q13 bölgesinde AAAS geni olarak tanımlanmıştır. Triple A sendromlu hastalarda AAAS geninde Çocuk Sağlığı ve Hastalıkları Dergisi 2014; 57: 195-199 Vaka Takdimi farklı klinik tablolara neden olan birçok farklı mutasyon bildirilmiştir.*3

HODGKİN HASTALIĞI

Patofizyoloji

Hodgkin lenfoma retiküloendotelyal ve lenfatik sistemi etkileyen germinal merkez B hüclerinin bir kanseridir. HL' nin orjinini oluşturan Reed-Sternberg hücreleri yüksek oranda mutasyon içeren, işlevselliğini kaybetmiş ve apoptoz özelliğini yitirmiş B lenfosit kökenli hücrelerdir.

Etiyoloji

HL'nın etyolojisi tam olarak bilinmemekle beraber %30 kadar vakada Epstein-Barr virüs (EBV) pozitif saptanmıştır.

Epidemiyolojik veriler çevresel, genetik, ve immünolojik faktörlerin HL gelişiminde rol oynadığını düşündürmektedir.

Epidemiyoloji

HL genel olarak tüm yaş gruplarında görülmekle beraber genç erişkiler de daha sık rastlanmıştır.

HODGKIN LENFOMADA HİSTOPATOLOJİK SINIFLAMA (DSÖ)

1- Klasik Hodgkin Lenfoma

- A) Noduler Sklerozan HL
- B) Lenfositlen Fakir HL
- C) Mikst Sellüler HL
- D) Lenfositlen Zengin HL

2- Noduler Lenfosit Predominant Hodgkin Lenfoma

HODGKİN LENFOMADA PARANEOPLASTİK SENDROMLAR

21

PARANEOPLASTIC NEUROLOGICAL SYNDROMES IN LYMPHOMAS 3231

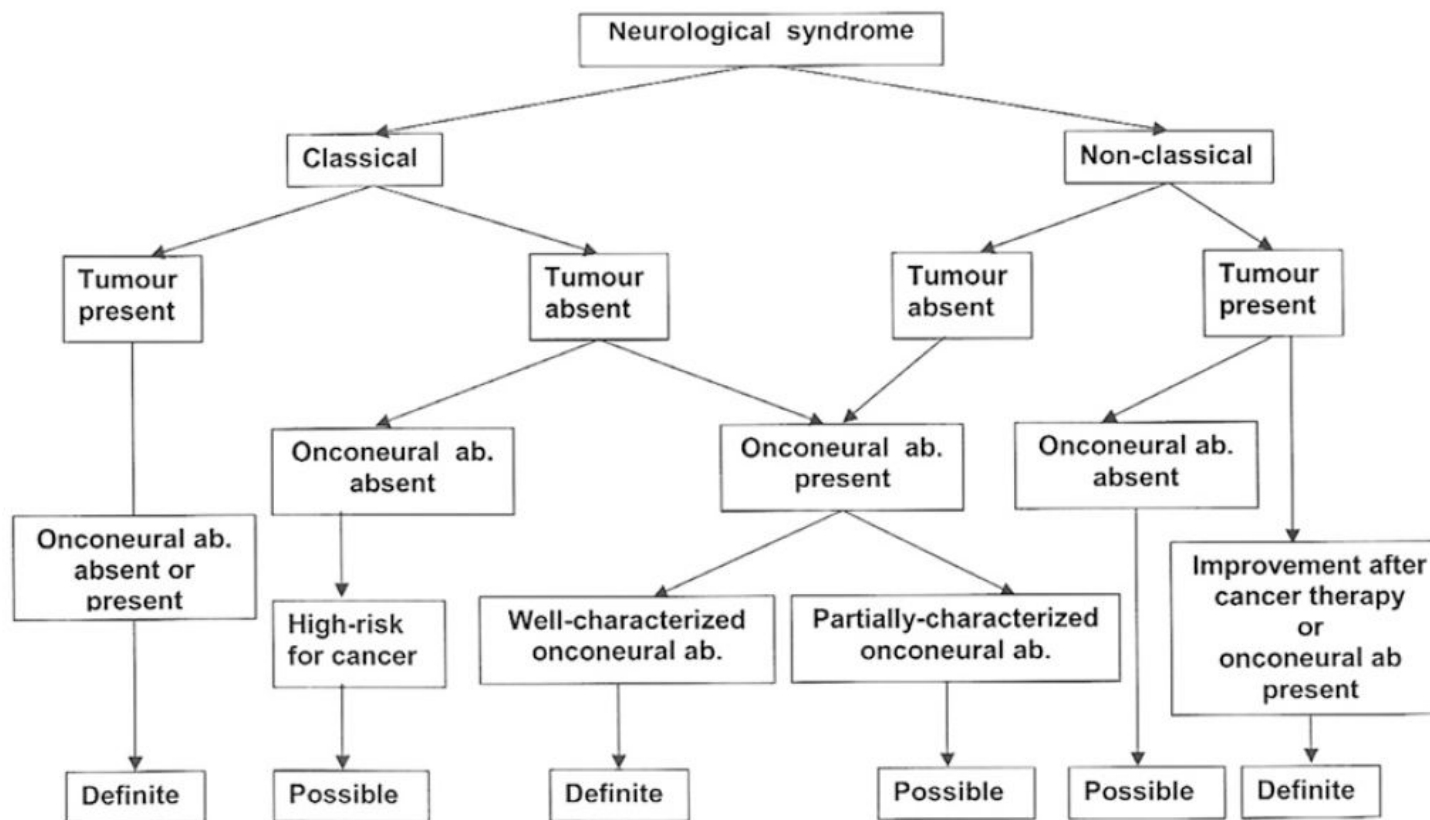


Table 1. Paraneoplastic neurological syndromes

Syndrome	Associated antibodies	Predominant lymphoma type	Selected references
<u>LE</u>	mGluR5	HL	16
Granulomatous angiitis	None	HL	40, 41
<u>Cerebellar degeneration</u>	Tr (DNER)	HL	5, 6
Paraneoplastic chorea	CV2/ CRMP5*	<10 cases (NHL in 4)	57, 58
<u>Opsoclonus-myoclonus</u>	None	<10 cases (NHL in 3)	64, 66
Stiff-person syndrome	None	HL	67, 72
Paraneoplastic myelopathy	None	HL and NHL	73, 74
Motor neuronopathy	None	HL	79, 80
<u>Sensory neuronopathy</u>	None†	<10 cases (5 with HL)	81, 82
Autonomic ganglionopathy	nAChR‡	<10 cases (HL, NHL)	87, 88
Sensorimotor neuropathy	None	HL and NHL	94
Vasculitic neuropathy	None	NHL	97
Neuromyotonia	None	<10 cases (HL, NHL)	104
<u>Lambert-Eaton myasthenic syndrome</u>	VGCC‡	<10 cases (NHL)	102
Myasthenia	AChR‡	HL and NHL	105
<u>Dermatomyositis</u>	p155	NHL	99

Classical syndromes are underlined. nAChR, nicotinic acetylcholine receptor; VGCC, voltage-gated calcium channel.

*Not present in all cases.

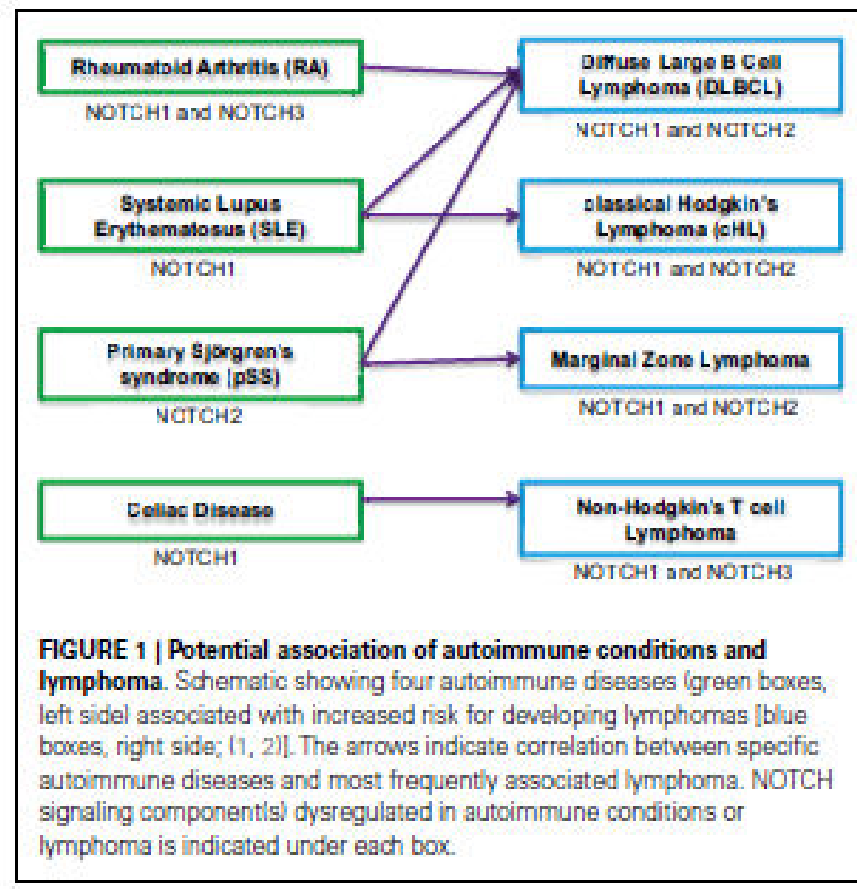
†One patient with NHL and Ma2 antibodies (not published).

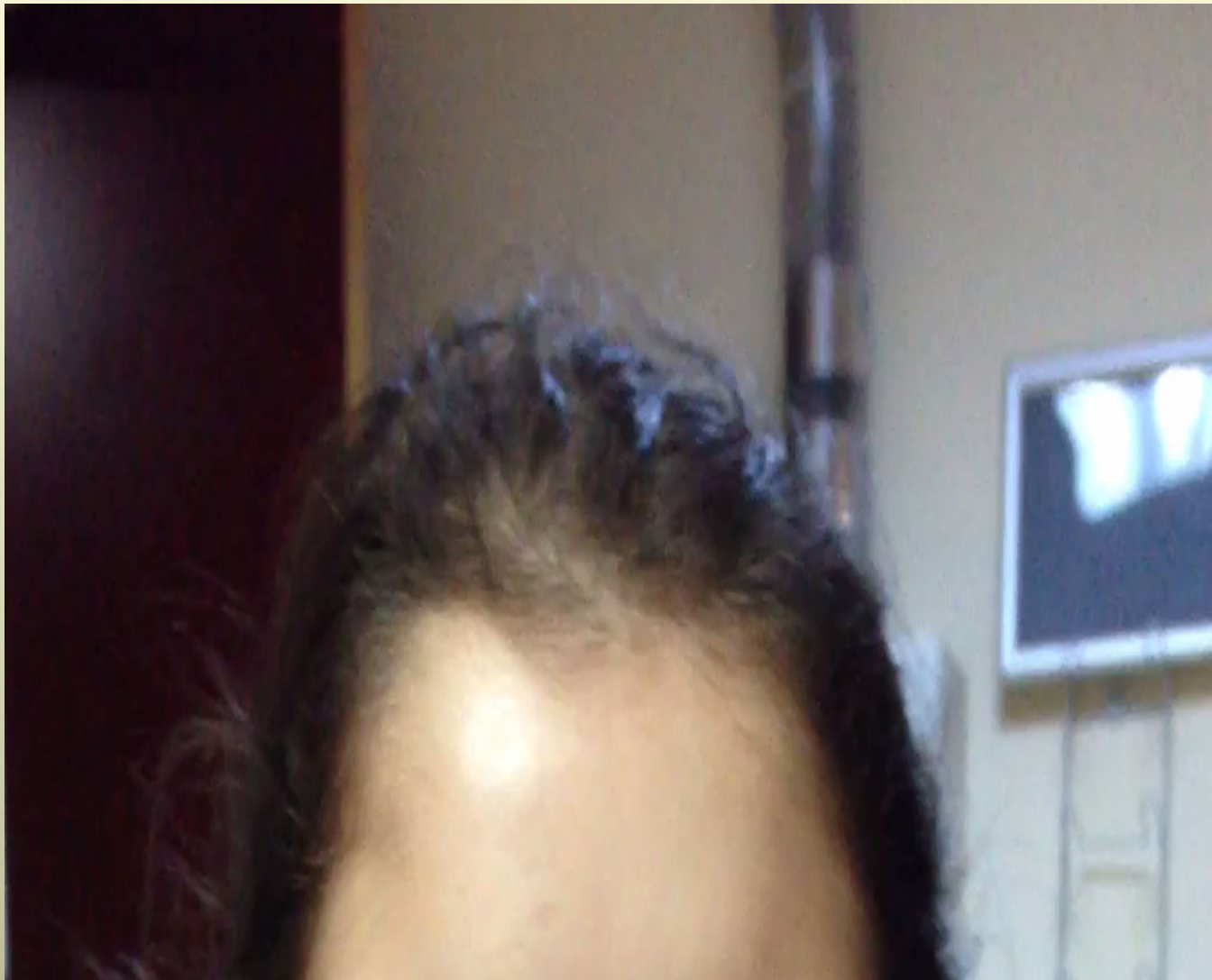
‡Marker of the syndrome, not predictor of cancer.

HL - otoimmünite

- Otoimmün hastalıklar
 - Nefrotik sendrom
 - Otoimmün hemolitik anemi
 - Otoimmün nötropeni
 - ITP

The link between autoimmunity and lymphoma: does NOTCH signaling play a contributing role?





KAYNAKLAR

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