# 南區頭痛讀書會

2010-10-31

嘉義基督教醫院 神經內科 吳啓順醫師

## Patient Profile

- Name: 張王□□
- Age: 68 y/o
- Sex: Female
- Date of hospitalization:

2010-06-10 --- 2010-06-21

# Chief Complaint

 Chronic right temporal throbbing headache for more than 6 months

### Present illness

- She began to have right temporal throbbing headache since 2009-10.
- Severity: moderate to severe
- Frequency: nearly daily, several times/day
- Duration: 30min to 2hrs/attack
- Nausea/Vomiting: (+)
- Associated S/S: right eye tearing and redness
- S/S aggravated since 2010-04

# Past History

- Hypertension(+)
- Type 2 DM(+)
- Hyperlipidemia(+)
- Smoking(-)/Alcohol(-)
- Drug allergy(-)

## Neurological Examination

- Cons: clear
- High cortical function: intact
- Cranial Nerve: Normal
- MP: 5 / 5 TR: symmetric Babinski sign:-/-
- Sensory: symmetric and intact
- Cerebellar sign: (-)
- EPS :tremor(-), rigidity(-), bradykinesia(-)
- Gait: fair
- Sphincter function: OK

## Neurological Examination

- Bilateral temporal artery pulse: + / +
- Right eye conjunctiva and sclera congestion with tearing while headache attack.

# Lab data

03 01	CBC 全套血液檢查				
03 05	WBC 白血球計數		8.39	3.5~9.9	x10^3/u
03 04	RBC 紅血球計數	H	5.96	4.2~5.4	x10^6/u
<b>03 06</b>	Hb 血色素		12.5	12~16	g/dl
03 07	Ht 血球容積比		38.3	37~47	8
■ 0307A	MCU 平均紅血球容積	L	64.3	81∼99	f1
■ 0307B	MCH 平均紅血球血色素	L	21.0	27~31	pg
■ 03 07 C	MCHC 平均紅血球血色	L	32.6	33~37	g/dl
03 08	Platelet 血小板計數		238	130~400	x10^3/u
■ 0301C	RDW-SD 紅血球分佈寬《	L	33.0	38~48	f1
03 01 B	RDW-CV 紅血球分佈質《		14.3	12~17	%
03 01E	MPU 平均血小板容積		10.4	9∼13	f1
03 03	WBC DC 白血球分類				
03 03 G	Segment 分葉細胞		59.1	50∼65	%
03 03 H	Lymphocyte 淋巴球		32.1	20~40	%
03 03 I	Monocyte 單核球		6.0	4∼8	8
	Eosinophil 階酸性球		2.3	1~3	8
03 03K	Basophil 階鹼性球		0.5	0∼1	%

# Lab data

診斷 1 :頭 代 號	新 項 目	名	稱	H/L	檢	診斷 2 驗	: 値		<b>∌</b>	考	値	單	位
0415 0416 0420B 0421B 0426B 0427B ■0430	GPT 麩 BUN(B) Creati Na (B) K (B)	胺酸所 尿素 nine( 動 鉀		胺 酐	19 12 14.8 1.0 138 3.89 212					4 3 ~1.5 ~150 ~5		U/L U/L mg/c mg/c mmo: mmo:	d1 1/L 1/L
簽收時間	葉雅蓉	(	99/06/0	7 19:	95:17	列印	  時間:	吳啓順		099/1	0/29	16:2	4:53
■0448	HbA1c	糖化	血色素	Н	7.4				4~	6		%	
簽收時間	:葉雅蓉	#	099/06/	12 07:	16:47	列	抑時間	:吳啓順	į	099/	10/29	16:2	6:47
診斷1:頭 診斷3:本	痛 態性高[	fot leak				診斷 2 診斷 4	·第二型	型或未明	月示型	型糖尿	病未	是及付	ff
111 5.5	濱 冒	答	稱	H/L	檢	田分四日 田前 別が	値		參	考	偤	単	位
0317	ESR 煮工[	<u>但球</u> 炎	<b>郡</b> 建度		17				Ø~2	9		pun/	lır
※収時間: 数告時間:	 葉雅蓉 陳美输		99/06/1 <sup>-</sup> 99/06/1 <sup>-</sup>		12:14 10:29	 列b 	 	 吳啓順 		099/(	39/2 <sup>1</sup>	18:5	7:11

## Trigeminal autonomic cephalalgias

 The trigeminal autonomic cephalalgias (TACs) are a group of primary headache disorders characterized by

- 1. Unilateral trigeminal distribution pain
- Occurs in association with ipsilateral cranial autonomic features

# Differential Diagnosis

Clinical features of the trigeminal autonomic cephalalgias

	Cluster headache	Paroxysmal hemicrania	SUNCT		
Sex (female:male)	1:3 to 1:7	1:1 to 2.7:1	1:1.5		
Pain					
Туре	Stabbing, boring	Sharp, stabbing, throbbing	Burning, stabbing, sharp		
Severity	Excruciating	Excruciating	Severe to excruciating		
Site	Orbit, temple	Orbit, temple	Periorbital		
Attack frequency	1 every other day to 8 per day	1 to 40 a day (>5 per day for more than half the time)	3 to 200 per day		
Duration of attack	15 to 180 minutes	2 to 30 minutes	5 to 240 seconds		
Autonomic features	Yes	Yes	Yes (prominent conjunctival injection and lacrimation)		
Migrainous features (nausea, photophobia or phonophobia)	Yes	Yes	Rare		
Alcohol trigger	Yes	Occasional	No		
Cutaneous triggers	No	Rare	Yes		
Indomethacin effect	None	Absolute	None		
Abortive treatment	Sumatriptan injection or nasal spray Oxygen	Nil	Nil		
Prophylactic treatment	Verapamil Methysergide Lithium	Indomethacin	Lamotrigine Topiramate Gabapentin		

SUNCT: short-lasting unilateral neuralgiform pain with conjunctival injection and tearing.

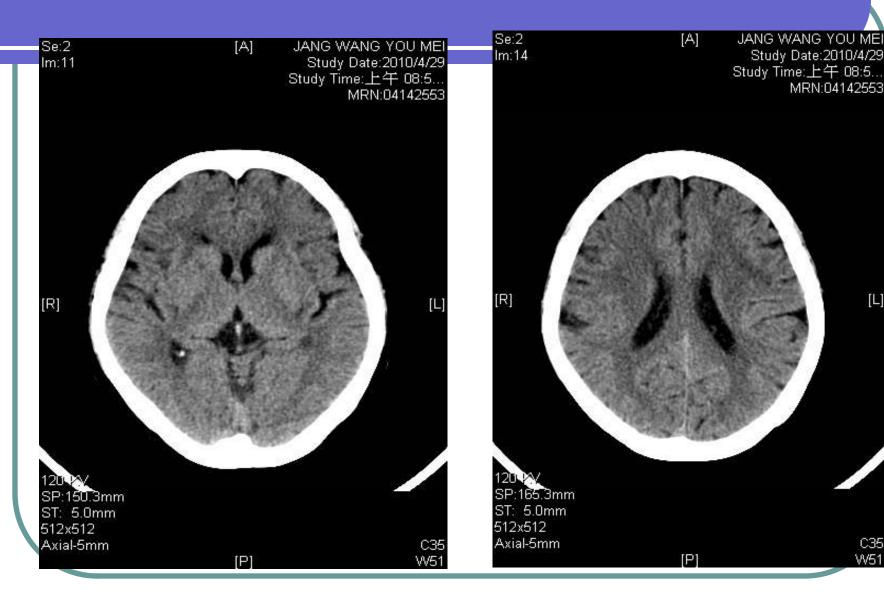
Uptodate 2010

## Non-Contrast Brain CT(2010-04-29)





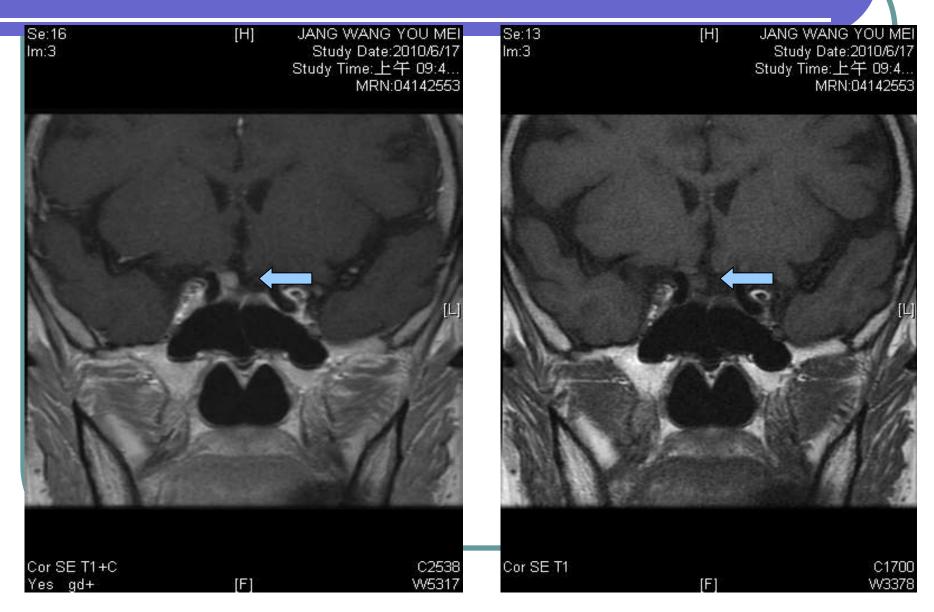
# Non-Contrast Brain CT(2010-04-29)



## **Treatment Course**

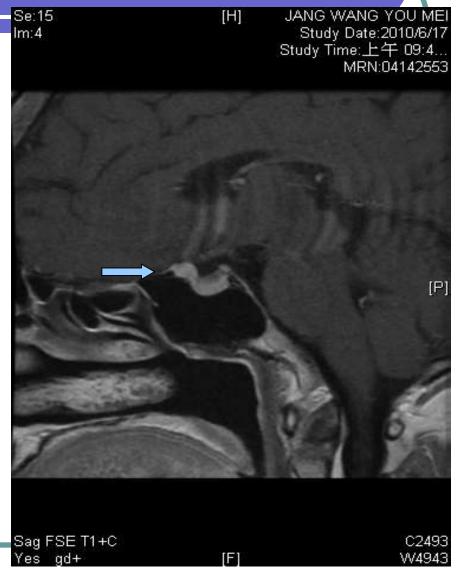
- Oral indomethacin trial
  - → Fail to relieve headache with dose up to indomethacin(50) 2# tid po
- 100% O2 inhalation therapy
  - → No obvious effect to relieve acute headache attack
- Partial response to oral Prednisolone 45mg/day
- Arrange brain MRI/MRA study to rule out structural lesion.

# Brain MRI/MRA with enhancement 2010-06-17

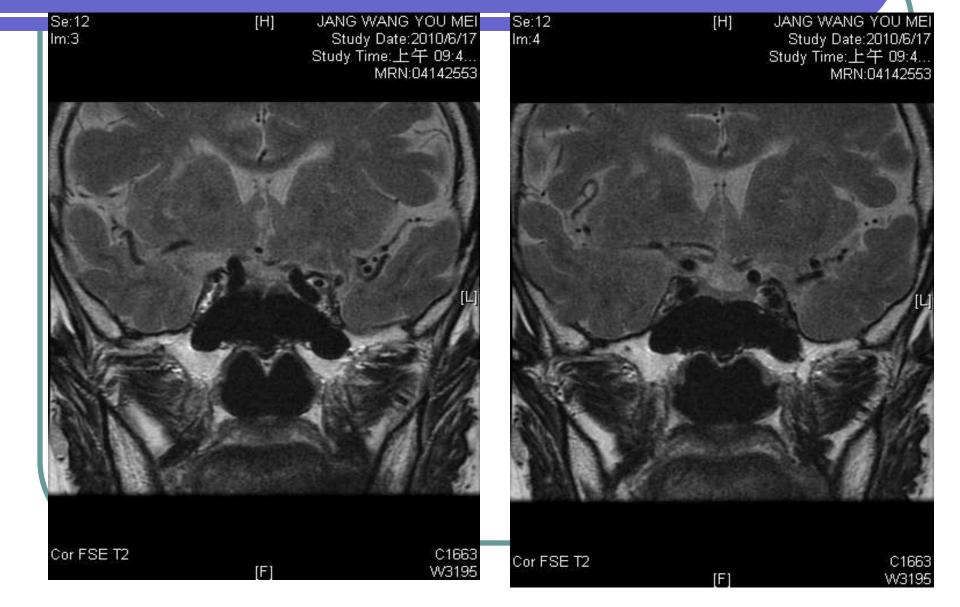


# Brain MRI/MRA with enhancement 2010-06-17

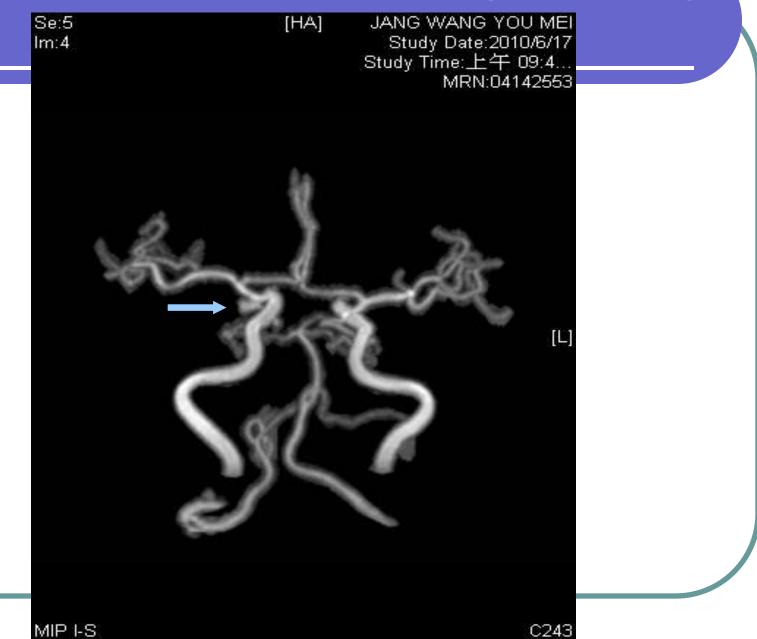




# Brain MRI/MRA with enhancement 2010-06-17



#### Brain MRI/MRA with enhancement(2010-06-17)



[FP]

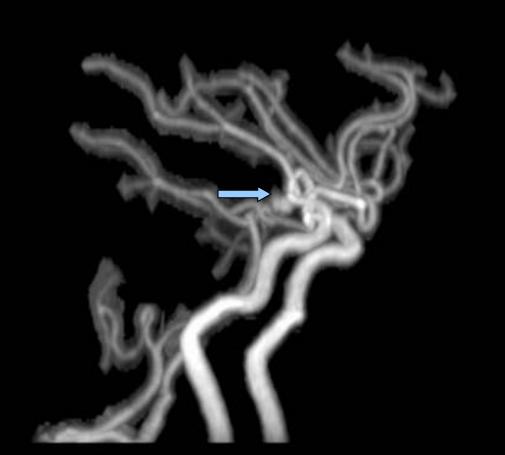
W482

#### Brain MRI/MRA with enhancement(2010-06-17)

Se:4 lm:7

[H]

JANG WANG YOU MEI Study Date:2010/6/17 Study Time:上午 09:47:44 MRN:04142553



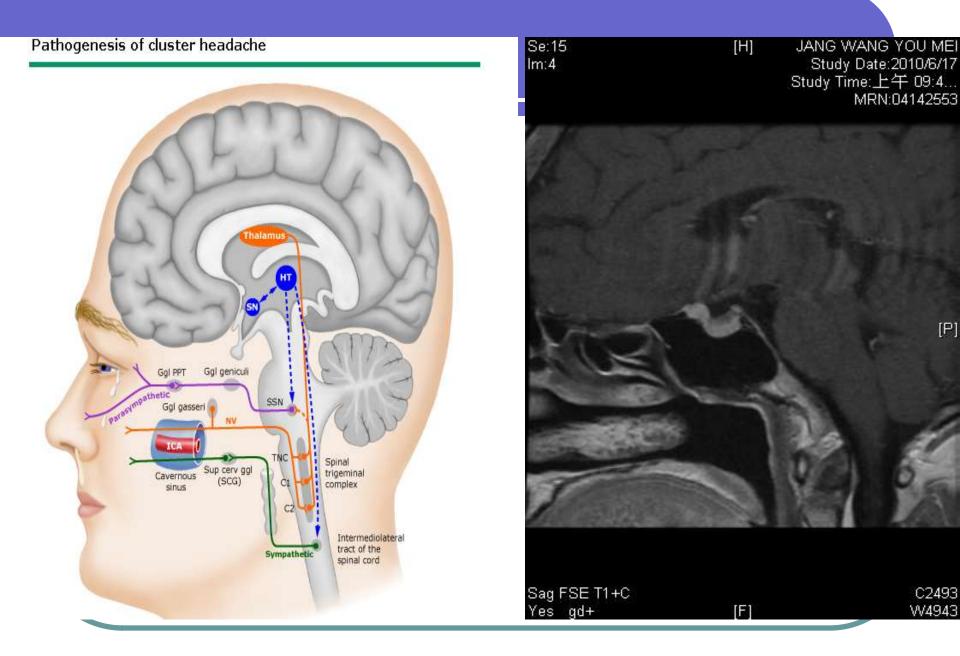
[AL]

## Brain MRI/MRA Report

- One 5\*4mm Rt Pcom artery aneurysm with posterolateral tilting.(Se/Im:5/4, 6/74) In favor of Rt fetal PCA noted.
- >No obvious focal abnormal enhanced lesion in the brain parenchyma.
- >No definite hyperintense areas on DWI in the brain.
- >Several tiny hyperintense areas in the periventricular white matters on T2WI and FLAIR noted, probabbly due to gliosis, demyelination, or old tiny ischemic infarctions.
- >The ventricular systmem and cortical sulci showed no dilatation.
- >There was no mass effect or midline shift.

#### Imp:

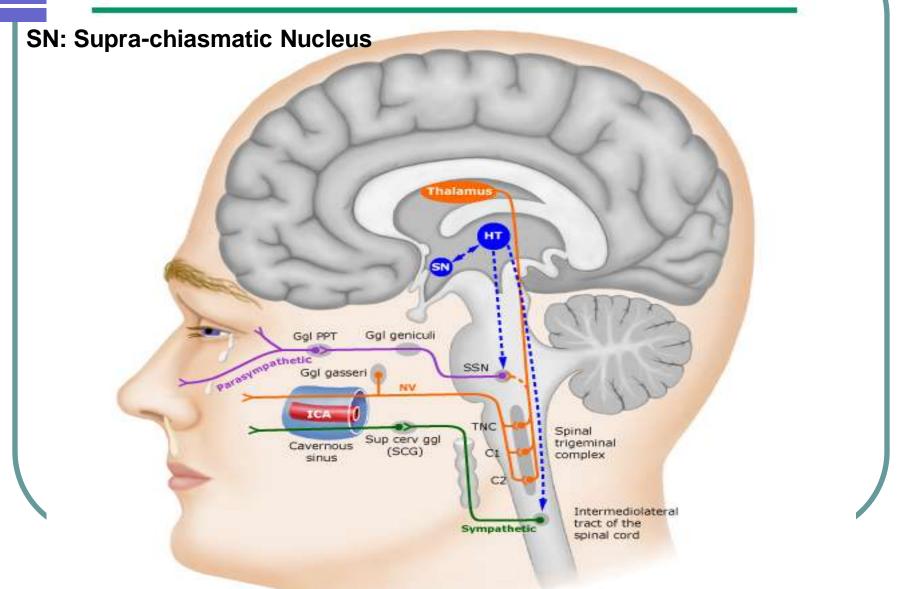
- 1)One 5\*4mm Rt Pcom artery saccular aneurysm with posterolateral tilting.
- (Se/Im:5/4,6/74). In favor of Rt fetal PCA noted.
- 2)One 5mm suprasellar enhanced nodule with connection to Rt pituitary gland ,adjacent to Rt optic nerve and cavernous Rt ICA. (Se/Im:16/3,15/4) Nature?
- Please correlation with clinical status and further management.



Cluster headache: Pathogenesis, diagnosis and management. Lacet 2005; 366:847

# Cluster headache: Pathogenesis, diagnosis and management. Lacet 2005; 366:847

Pathogenesis of cluster headache



## Pathogenesis of Cluster Headache

Schematic model showing most of the putative actors in CH pathogenesis. Pain afferents from the trigeminovascular system. synapse on the trigeminocervical complex (TNC), and then project to the thalamus and lead to activation in cortical areas known to be involved in pain transmission. Either a direct influence of the hypothalamus or a reflex activation of the parasympathetic outflow from the superior salivatory nucleus (SSN) predominately through the pterygopalatine (sphenopalatine) ganglion, leads to the parasympathetic symptoms ipsilateral to the pain. A third-order sympathetic nerve lesion, thought to be caused by vascular changes in the cavernous sinus loggia with subsequent irritation of the local plexus of nerve fibers, results in a partial Horner's syndrome. The key site in the CNS for triggering the pain and controlling the cycling aspects is in the posterior hypothalamic grey matter region, modulated by phase-shifting in the suprachiasmatic nuclei. Abbreviations: Gql = qanqlion, HT = hypothalamus, ICA = internal carotid artery, NV = trigeminal nerve, PPT = pterygopalatine, SCG = superior cervical ganglion, SN = suprachiasmatic nucleus, SSN = superior salivatory nucleus, TNC = trigeminal nucleus caudalis Modified from: May A. Cluster headache: pathogenesis, diagnosis, and

management, Lancet 2005; 366:847.

## Symptomatic Trigeminal Autonomic Cephalalgia

The Neurologist • Volume 15, Number 6, November 2009

- The majority of the cases of TACs are idiopathic or primary.
- There are numerous case reports of symptomatic cases in the literature, though a causal relationship with the underlying structural lesion is uncertain in many cases.
- This raises the issue of diagnostic evaluation required in this patient group to identify those with a causal underlying structural lesion.

#### **SYMPTOMATIC Cluster Headche**

- 24 case reports of symptomatic cluster headache (CH) were indentified from literature review.
- The meam age at the onset of symptoms was 39 years. (range: 14–68 years).
- 16 were male and 8 were female.
- The mean time to diagnosis from onset of symptoms was 7 years (range: 2 weeks to 31 years).
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## **SYMPTOMATIC Cluster Headche**

#### Underlying Lesion

- In 8 patients (33%), a vascular abnormality was identified including arteriovenous malformations, internal carotid artery dissections, cerebral venous sinus thrombosis, and subclavian steal syndrome.
- Twelve patients (50%) had a tumor, including 7 with pituitary tumors (29%).
- 1 case report of idiopathic granulomatous hypophysitis.
- Other lesions identified included a clival epidermoid cyst, sphenoidal aspergilloma, foreign body in the maxillary sinus in 1 patient each.
- Two patients had a normal computed tomography (CT) study but a subsequent abnormal magnetic resonance imaging (MRI) study.
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#### Response to CH Abortive and Preventive Treatments

- Eight (33%) patients failed to respond to the abortive and preventive treatments tried.
- Twelve patients (50%) responded with partial or complete benefit with at least 1 abortive or preventive treatment.
- Four patients (17%) did not have any preventive treatments.
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#### Response to Treatment of Pathology

- All the patients were reported as showing complete resolution of the headache syndrome after treatment of the underlying pathology.
- Though the follow-up period was not stated in 6 cases and was fairly short in several other cases.

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## Conclusion

 A remarkable finding of this review was that a fairly high proportion of trigeminal autonomic cephalalgia syndromes were
 secondary to pituitary lesions

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### Conclusion

- The purpose of this review was to identify the clinical features that may be pointers for distinguishing symptomatic cases of TACs from primary ones.
- It is difficult to draw definitive guidelines from this retrospective review, especially given the small number of cases identified.
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## Conclusion

- An atypical clinical phenotype or abnormal examination was present in at least half of the symptomatic cases of CH and SUNCT, and all cases of PH.
- Hence, the presence of atypical symptomatology or an abnormal examination at presentation or their development in the subsequent course of the disorder should prompt further investigations.

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THANKS FOR YOUR ATTENTION !!