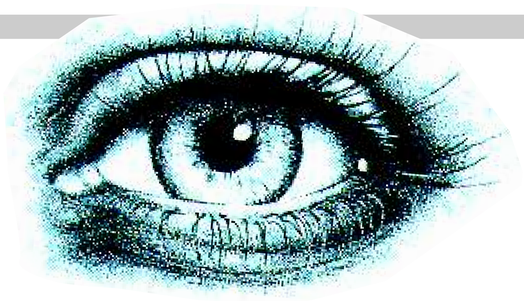

INTRACRANIAL IDIOPATHIC HIPERTENSION OR PSEUDOTUMOR CEREBRI WITH ANTIPHOSPHOLIPID ANTIBODIES PRESENT TREATED WITH LOW MOLECULE WEIGHT HEPARIN - ORAL FORMULA

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ALTERNATIVE NAME

- Idiopathic intracranial hypertension
- Benign intracranial hypertension
- Pseudotumor cerebri

Although often considered to be "idiopathtic" detailed investigation has reveealed a high incidence of outflow abnormalities in PTC syndrom patients. Paul W. Brazis,2004

THE DIAGNOSTIC CRITERIA for idiopathic cases should include the following:

1)if symptoms are present, they may only reflect those of generalized intracranial hypertension or papilledema;

2)if signs are present, they may only reflect those of generalized intracranial hypertension or papilledema;

3)documented elevated ICP measured with the patient in the lateral decubitus position;

4) normal CSF composition;

5)no evidence of mass, structural, or vascular lesionon magnetic resonance imaging (MRI) or contrast-enhanced computed tomography (CT) for typical patients, and MRI and MR venography for all others;

6) no other cause of intracranial hypertension identiffied FRIEDMAN DI.2002

PATHOGENIC MECHANISM

in all our cases of presumed PTC, obstruction or impairment of intracranial venous drainage must be considered as a mechanism for cerebral edema with incresed ICP and papilledema.

Venous sinus thrombosis may be the mechanism of PTC reported with systemic lupus erythematosus, essential thrombocccythhhhhemia, proteinnS deficiency, antithrombin III deficiency, the antiphospholipid antibody syndrome, paroxysmal nocturnal hemoglobinuria, Behcet"s disease, meningeal sarcoidosis, hypervitaminosis A, mastoiditis, and other disease processes (Sareen D, Castillo IG, Celebisoy N, 2002)

1887 Quincke was the first describe the condition known today as pseudotumor cerebri

1904 Nonne introduced the term pseudotumor cerebri

1955 Foley introduced the term begin intracranial pressure

2005 Irrespective of the term used the problems connected to this entity remain in the focus of nowadays research...

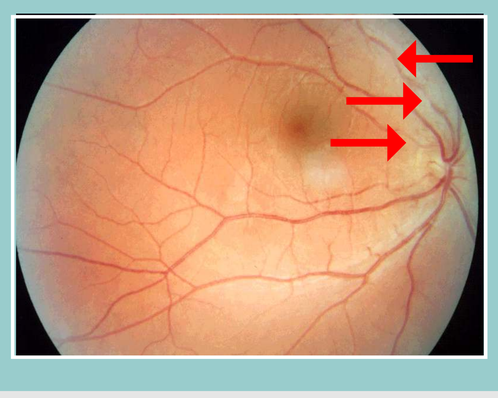
MATERIAL AND METODH

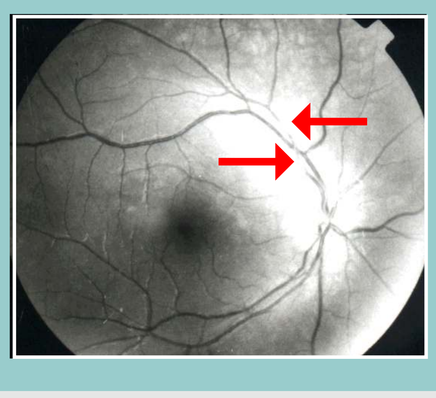
PATIENT	AGE/YEARS	HEADACHE	TRANSIENT VISUAL OBSCURA-TIONS	DPLOPIA	DIZZNES	NAUSEA	VOMITING	TINITUS	PAPILLEDEMA
1	24	●	✸	✱	■	▲	▽	◆	
2	24	●	✸	✱	■	▲	▽	◆	✖
3	27	●	✸		■	▲	▽	◆	
4	31	●	✸		■	▲	▽		✖
5	33	●	✸		■	▲	▽		✖
6	34	●	✸		■			◆	
7	41	●	✸			▲	▽		
8	47	●	✸		■			◆	
9	49	●	✸		■	▲	▽		✖
10	49	●		✱	■	▲	▽	◆	

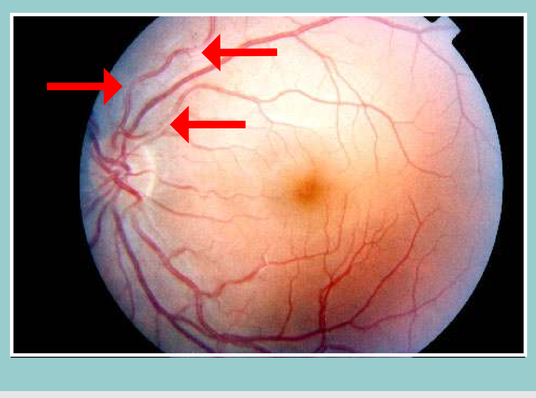
ACL	LA	β2GP I	PUNCTURE LUMBAR	CT- scan	MRI
+ + + + +	–	p	PL	CT	
+ + + +	–			CT	MRI
+ + + +	+		PL	CT	
+ + + + +	–	p		CT	MRI
+ + + + +	+			CT	MRI
+ + + + +	–			CT	
+ + + + +	–	p	PL		MRI
+ + + +	+		PL		MRI
+ + + + +	+			CT	MRI
+ + + +	+			CT	

RESULTATS AFTER TREATMENT WITH SULODEXID

TREATMENT WITH SULODEXID	1 MONTH	3 MONTHS	6 MONTHS	9 MONTHS	12 MONTH
1	● ■ ◆	● ◆	◗		
2	● ✱ ■ ◆ ✖	● ■	◆ ◗	◆	◗
3	● ■ ◆	● ◆	◗		◗
4	● ■ ✖	◗	◗		
5	● ✖	◗	◗		
6	● ■ ◆	● ■ ◆	◗		
7	● ◆	● ◆	●	◆	◗
8	● ■	◗	◗		◗
9	● ■ ✖	◗	◗		
10	● ◆	● ◆	●	◆	◗







Follow-up treatment with orally anticoagulant therapy:

- over 10 month : 6 cases
- 8 months: 1 case
- 7 months: 1 case
- stopped over 6 month : 1 case
- over 12 months : 1 case

Heparin produces its anticoagulant effects by binding to antithrombin III and inhibiting thrombogenesis primarly through inactivation of factors IIa and Xa. On theoretical ground there is a potential for the prevention and treatment of cerebrovascular diseases, to this purpose drugs interfering with hemostasis could be of interest. *Cerletii C., 1994*

Sulodexide: The additive effect of fast moving heparin and dermatan sulfate on thrombin inhibition is the mechanism of the anticoagulant action of natural mixture of glycosaminoglycans:sulodexide. *Pancani C., 2000*

We suggest that the treatment with sulodexid should be used in HII when the described aspect of the retinal vessels exists together with the antiphhospholipid anttibodies.