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Hyponatremia of 100 mEq./l tr. Associated with Pituitary Adenoma. A Case Report.

Robert H. Kyle, M.D., Hwa Dong, Lee M.D., and Byung Chan Jeon M.D.

Department of Neurosurgery, Kosin Medical College, Pusan, Korea

=요 약=

본 44세 남자환자로 내원 12개월 전부터 성욕감퇴, 20일 전부터 식욕감퇴, 오심, 구토를 주소로 입원했다.

내원 당시 탈수현상을 보였으며 2일후 의식의 변화(혼미)와 한차례의 대발작증세를 보였다.

뇨와 혈청 소견상 혈중 나트륨치가 100mEq/L, 혈중 Cortisol치가 1.1 mEq/24hrs, 요비중은 변동이 있었으나 1.004까지 보였고, 그 외는 정상범위였다.

단순두개골 방사선학적 소견으로는 터키 안장부위가 커져있고 부분결손을 보였으며 뇌 전산화 단층 촬영 소견으로는 터어키 안장상부 및 왼쪽에 팽대된 뇌하수체종양을 보였다.

수술요법과 방사선요법을 받았으며, 현재 투약없이 비교적 안정된 일상생활을 영위하고 있다.

Excess secretion of antidiuretic hormone (ADH) from deranged hypothalamic control causes water intoxication encephalopathy with hyponatremia and renal salt loss. In some patients with adrenal insufficiency or with hypothyoidism, elevated plasma ADH levels have been found.[1] Neurological signs occur regularly when the serum sodium falls below 110 mEq./ltr., and coma ensues when the level falls below 90–105 mEq./ltr [2]

Our patient is a 44 year old male who was first admitted briefly in May 1984 complaining of abdominal pain, nausea and vomiting for three days but admitting loss of appetite for years and loss of libido for about 8 months. Physical examination revealed a blood pressure of 100/60, 110/70 and some diffuse epigastric tenderness Mental changes were noted He was found to be quite hyponatremic with wide fluctuations of urine specific gravity. He was treated with 306 mEq. NaCl daily for 3 days. The serum sodium rose to 118 with improvement and mental clearing. He refused more fluids and was discharged after 9 days, improved but weak.

He was readmitted September 7, 1984 complaining of loss of appetite, nausea and vomiting, much worse in the preceding 20 days. He appeared dehydrated. Blood pressure ranged from 100/70 to 110/70. He had serum sodium 109 mEq./ltr. with potassium 3.9 mEq./ltr. See Table I. Two days later the sodium was 100 mEq./ltr. He had lapsed into stupor and suffered a grand mal seizure. This resulted in a brain CT scan(fig. 1) showing a moderate sized pituitary tumor with slight suprasellar extension towards the left, whereupon he was transferred to our department. Using 3% NaCl the serum sodium was slowly brought

up while dexamethasone was continued and dilantin was started A skull film showed an enlarged eroded sella turcica with doubly contoured floor indicating more tumor growth on one side (fig 2) Carotid angiograms showed opening of the left carotid siphon (fig. 3). The patient soon became lucid, thirsty and then hungry He could calculate and talk with mild dysarthria. Visual acuity was 0.7 O.D and 0.6 O S Perimetry showed only concentric constricituon worse on the right (fig 4).

A left pterional craniotomy was performed and the chiasmal area explored using the microscope A large soft grayish-pink fairly firm turmor was found between the optic nerves arising from the sella turcica. There was more tumor bulk under the left optic nerve than the right About 4 cc of tumor was removed while lysing the adhesions to the optic nerves and chiasm The cavity was filled with pieces of temporal muscle to gain hemostasis. He was awake and talking on leaving the operatng room.

The patient has convalesced nicely. The tumor is a typical pituitary adenoma shown in fig. 5, while preoperative and postoperative perimetry is shown in fig. 4

Postoperatively, a couple of urinary specific gravities were down to 1.009 and 1.007, but he was stabilized in the range of 1.013–1.030. Serum sodium has stabilized at about 130mEq/ltr. Peripheral fields show minimal increase of the left temporal constriction but of questionable significance. Following radiotherapy he was able to resume work, taking no medication, and eating well.

Table. I. Summarized Laboratory Data

Test	Normal Values	May 1984	Sept 1984
Serum Na mEq.		110, 109	109, 100, 105
Serum K 〃		4.0, 3.6	3.9, 4.0
Serum Cl 〃		72, 71	
Urine Na 〃		116*	25
Urine K 〃		29*	9
F. B. S.		105, 93	105
BUN		12, 12	10, 11
Creatinine		1.0	
Plasma Cortisol μg/dl.	8.2–8.6		1.1#
TSH μn/ml.	0–10		1.0
T_3 ng/dl.	70–190		30.0
T_4 μg/dl.	5–15		10.0
Prolactin ng/ml.	2.4–12.7		4.0
Hb		14.5	13.8
Hct		42	40
Urine S. G.		1.032, 1.006	1.019, 1010
		1.003, 1.040	1.004

*Patient had received 306 mEq NaCl in preceding 24 hrs

#Patient had received 20mg dexmethasone in the preceding 24 hrs

DISCUSSION

This patient shows some of the features of SIADH syndrome, but as Streeten, et al.[3] warn, it is essential to exclude adrenal insufficiency which this man may have (plasma cortisol of 1.1) along with a slight degree of hypothyroidism. He had an excessive ADH secretion. Possibly this large tumor inhibits some unknown inhibitor of ADH secretion, allowing the excessive secretion At the same time by direct pressure on the anterior pituitary cells the pituitary tumor probably suppresses ACTH secretion with the resultant low plasma cortisol.

We attempted to rule out disease in other organs which might cause increased ADH secretion as follows.

1 The normal chest x-ray rules out bronchogenic carcinoma.

2 Thyroid tumor is ruled out by normal physical examination, low normal T_3 and normal T_4 Both were normal postoperatively.

3. Meningitis is ruled out by absence of fever, lack of meningeal irritation, and normal white cell count.

4. Chronic liver disease is ruled out by lack of physical findings, normal liver function tests on two

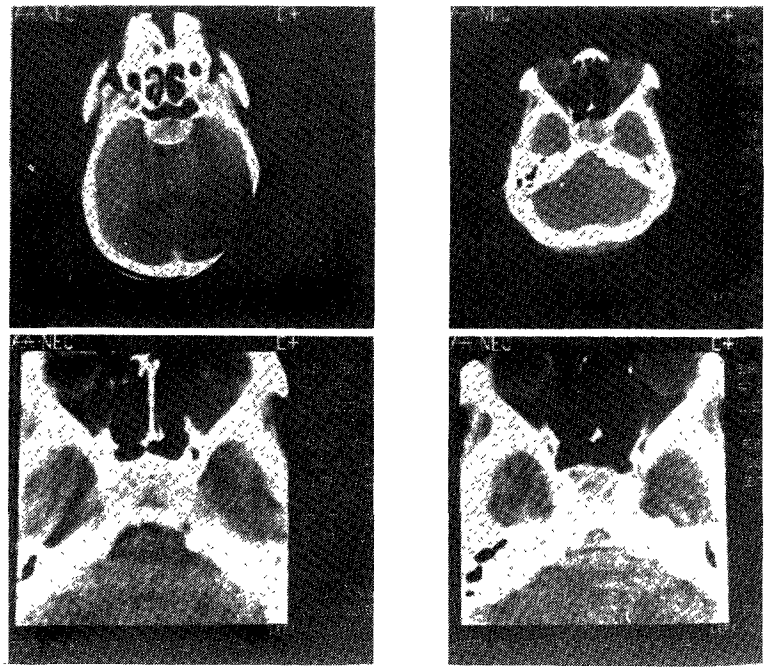

Fig. 1. CT shows a moderate sized pituitary tumor with suprasellar extension.

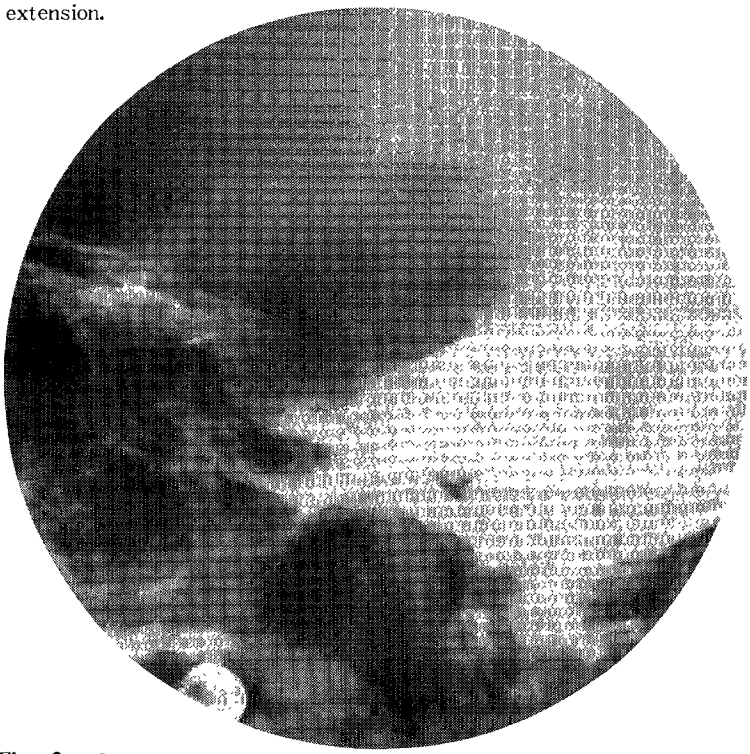

Fig. 2. Sella view shows enlarged, eroded sella turcica with doubly contour floor indicating more tumor growth on one side.

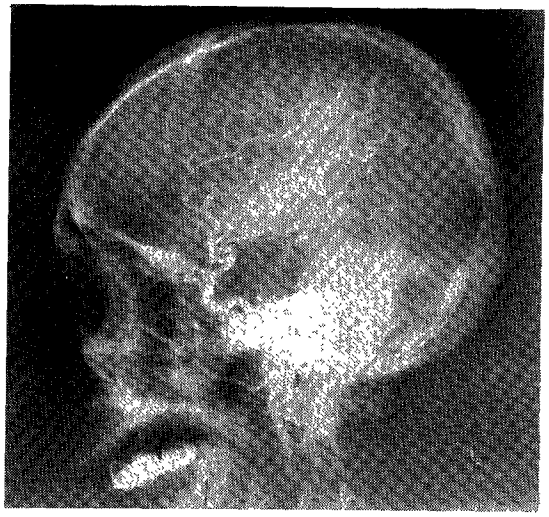

Fig. 3. Carotid angiogram shows opening of the left carotid siphon.

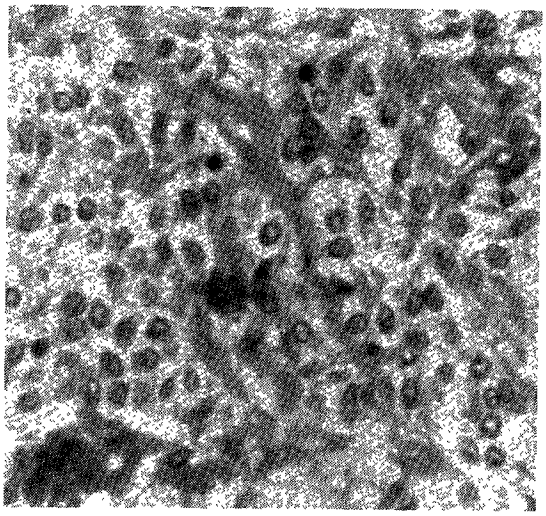

Fig. 5. Microscopic findings of pituitary adenoma.

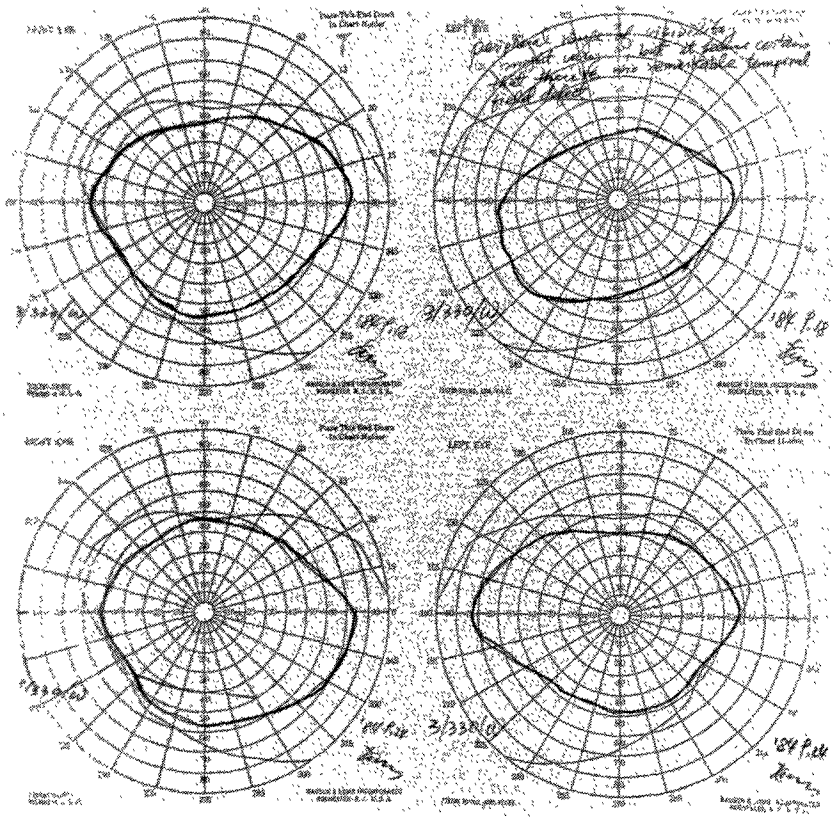

Fig. 4. The post-op perimetry shows slight improvement as compared with the pre-op one in the left temporal visual field.

occasions, and a normal liver scan

5. Renal disease is ruled out by normal BUN and creatinine on several occasions. Urine volumes approximately balanced the intake before and after surgery.

6 Volume overload is ruled out by the absence of heart disease with two normal ECGs and by the absence of liver disease.

The normal electrical activity of the brain is dependant on normal ionic membrane potential in all the brain cells. When the serum Na^+ falls from 140 to 100 mEq./1tr. there must be a corresponding disturbance in the ionic membrane potenital of many brain cells which triggers the grand mal seizure.

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