# DIABETES IN E.N.T.

## Essay

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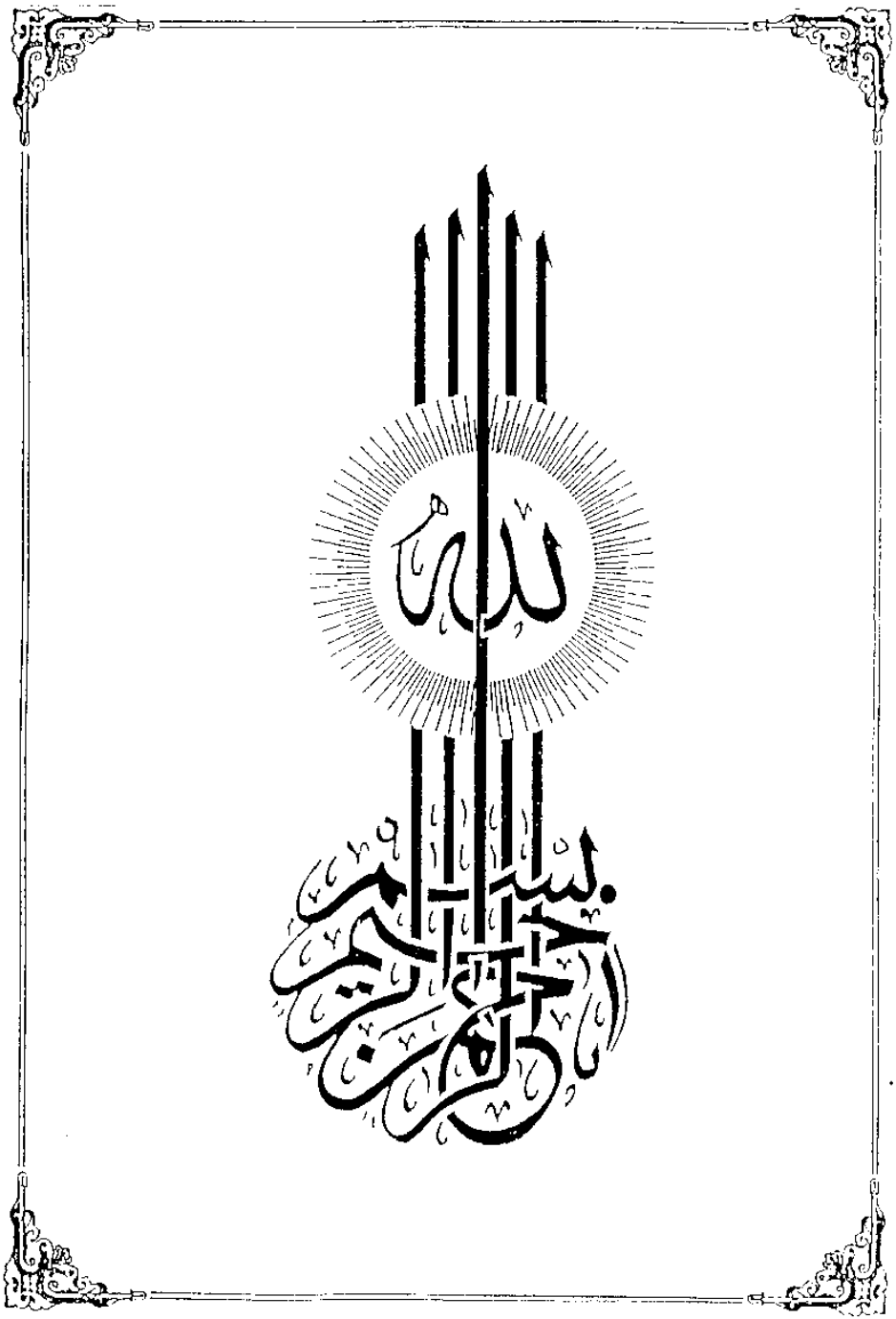

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# INTRODUCTION

# AIM OF THE WORK

The aim of the present study is to revise the reviews giving the relationship between diabetes and ear, nose and throat.

# DIABETES INSIPIDUS

Definition:

Diabetes insipidus is an uncommon disease. it is a temporary or chronic disorder of the neurohypophyseal system, due to deficiency of vasopressin (ADH) and characterized by excretion of excessive quantities of very dilute (but otherwise normal) urine and by excessive thirst. (Robert et al., 1977).

Classification:

This disorder is termed vasopressin - sensitive diabetes insipidus (DI) to distinguish it from nephrogenic diabetes insipidus (NDI), in which the kidney is vasopressin resistant. The disturbance in water metabolism is similar in both and is characterized by the daily production of a very large volume of dilute urine (Sp. Gravity usually $< 1.005$ or osmolality $< 200$ m Osm/L). DI may be complete partial permanent, or temporary. All of the pathologic lesions associated with DI involve the hypothalamic nuclei (supraoptic and paraventricular) or a major portion of the pituitary stalk. Simple destruction of the posterior lobe, although associated with temporary DI, does not produce the sustained disorder. The posterior lobe is the major site for ADH storage and release but is not involved in its synthesis. In its absence newly synthesized hormone can still be released into the circulation as long as the hypothalamic nuclei and part of the neurohypophyseal tract are intact. (Robert et al., 1977).

Ranson and associates (1938), utilized cats to demonstrate that animals with hypothalamic lesions sufficient to cause diabetes insipidus underwent a triphasic response to the surgical damage. Immediately following the lesion a polyuria and polydipsia commenced which losted usually 4 to 5 days. After this a period of intense antidiuresis set in for some 6 days. This was followed by the permanent polyuria and polydipsia of established diabetes insipidus. The same pattern of response has been observed in humans following high pituitary stalk section. It is generally agreed that the initial diuretic phase results from acute damage to hypothalamic function so that stored hormone is not released. The antidiuretic stage results from degeneration of hormone-laden tissue with release of the contained hormone into the circulation.

Administration of a water load during the antidiuretic phase will not induce the usual diuretic response. Removal of the posterior pituitary at the time of hypothalamic damage will prevent the antidiuretic phase (Hallinshead, 1964). With lesser damage to the hypothalamus, the diabetes insipidus may never recur after the initial polyuria and polydipsia. This is seen after head trauma or neurosurgery. In early diabetes insipidus of traumatic origin, the attending physician is wise to avoid hasty prognostications regarding the permanence of the condition.

Causes:

David et al., 1977, studied 51 patients (34 males and 17 females) who had had diabetes insipidus for at least 3 months, findings were as follows;

(1) Twenty were shown to have resulted from various types of neoplastic or infiltrative lesions of the hypothalamoneurohypophseal system. In addition to the lesions encountered in this series, other types of malignant metastases (such as carcinoma of the lung, breast cancer and lymphoma), sarcoid, hemorrhage, vascular lesions, abscess, meningitis, histocytosis, tuberculosis and syphilis have been described as occosional causes of diabetes insipidus.

(2) An increasingly frequent causes of diabetes insipidus (9 patients) is surgical procedures in the region of the hypothalamoneurohypophyseal system. These include hypophysectomy by surgical excision, cryosurgerys implantation of 90 yttrium and other radioactive isotopes, and heavy particle irradiation used for known tumors or for other lesions of the system or as a palliative procedure for diabetic retinopathy and metastatic carcinoma of the breast. Experience has confirmed the experimental finding in animals that surgically induced diabetes insipidus usually appears between 1 and 6 days after the operation. It often disappears

after being present for a few days, and may remain absent or may recur and become chronic after an "interphas" of 1 to 5 days.

(3) A third major group of patients (8 of the 51) developed diabetes insipidus after severe head injuries usually, if not always associated with fractures of the skull. This type of diabetes insipidus may last indefinitely but frequently lasts only a few days and sometimes (in 2 of our 8 patients) disappears spontaneously after several months or years presumably because of regeneration of disrupted supraoptic axons within the pituitary stalk.

(4) In a fourth group of patients (14 of 51 ) none of the currently available diagnostic procedures has been successful in revealing the cause. Some of these patients with idiopathic diabetes insipidus have other features of hypothalamic and/or pituitary disease such as subnormal stature, hypopituitarism, galactorrhea, and narcolepsy, and they presumably have functional or anatomic lesions of other systems in the hypothalamus or the pituitary, in addition to their dysfunction in the supraoptico neurohypophyseal system. Though no evidence of tumor can be found in these patients, the continued awareness of this possibility is sometimes rewarded by the finding of sellar enlargement after long-term follow-up. Occasional patients with such apparently isolated idiopathic deficiency of vasopressin have come to autopsy where a striking

decrease in the number or virtual absence of ganglion cells has been found is the supraoptic and paraventricular nuclei, associated with loss of Nissl substance in the remaining neurons and obvious gliosis. The neurohypophysis was reduced in size in these patients. It seems likely, therefore that the reduced number of supraoptic neurons is the cause of the vasopressin deficiency in these patients.

(5) Familial diabetes insipidus is very uncommon and must constitute less than 1 per cent of cases of diabetes insipidus It may occur in infancy or childhood and may affect either males or females. The condition has been described in seven generations of one family ( Blotner, 1942). Familial diabetes insipidus is to be distinguished from nephrogenic diabetes insipidus, the latter is a renal tubular defect inherited largely by males where in the affected tubules are unresponsive to antidiuretic hormone.

Pathophysiology:

Deficiency of vasopressin release in response to the appropriate stimuli may result from lesions at a variety of functional sites in the physiologic chain of events which culminates in discharge of the hormone into the blood stream. There may be a defect in the osmoreceptors where the stimulus to ADH release is normally sensed. Such patients have no antidiuretic response to hypertonic

saline, yet develop a good antidiuresis in response to nicotine or acetyl -B-methylcholine (Mecholyl). Occassionally there is an abnormally elevated osmotic threshold for vasopressin release. These patients excrete hypotonic urine in spite of water deprivation until serum sodium rises to chronically elevated levels corresponding to their raised osmotic thresholds for ADH release, above 300 milliosmoles per kg. In other, rare instances elevation of the osmotic threshold has presented with largely asymptomatic hypernatremia associated with loss of thirst and mild or absent evidence of diabetes insipidus. Occasional patients have been reported to show normal antidiuretic responses to osmotic stimuli but impaired responses to such presumably neurogenic influences as nicotine or the smoking of cigarettes. Reduction in the number of supraoptic neurons may be the cause of deficient ADH production in some patients who present clinically with "idiopathic" diabetes insipidus. - In diabetes insipidus there is defective reabsorption of water from the collecting tubules of the kidney. - The loss of water without a corresponding depletion of electrolytes will increase osmolarity of the extracellular fluid. This leads to a transfer of intracellular water to the extracellular compartment and a consequent cellular dehydration. As the water leaves the cells, it is accompanied by potassium ions. Sodium excretion is unchanged at first, but later on when there is a shrinkage of the