

SUPRARENAL TUMOURS

AN ESSAY

Submitted for partial fulfilment
of the Master Degree in **General Surgery**

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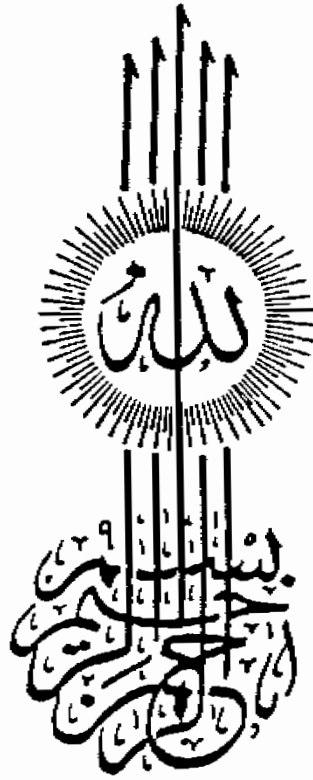
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**INTRODUCTION
AND
AIM OF THE ESSAY**

INTRODUCTION AND AIM OF THIS ESSAY

Adrenal tumours are rare and often present late when physiological derangement is well established.

These tumours present with a broad spectrum of symptoms, with three hormonally mediated syndromes being recognized: hypertensive, virilizing and Cushing's.

There is frequently a delay in recognition of the cause of these syndromes. The cause of this delay is multifactorial, including the patient's failure to seek medical assistance and a low index of suspicion by the physician.

In the past two decades, the development and refinement of computed tomography (C.T.), and magnetic resonance imaging (MRI) as well as scintigraphic techniques have markedly improved the accuracy of preoperative diagnosis and localization of adrenal neoplasm.

This essay is a review of the surgical anatomy of the adrenal glands, the pathology and pathophysiology of adrenal

tumours, the clinical presentation and methods of diagnosis including the advanced radiological and biochemical procedures. Also the essay will discuss the preoperative assesement, preparation, the techniques and complications of surgical treatment of these tumours.

EMBRYOLOGY

EMBRYOLOGY

Adrenal glands arise from the coelomic epithelium early in the 4th week of gestation in the angle between the dorsal mesentry and the head of mesonephros (*Gray's, 1989*).

The adrenal gland is composed of two endocrine systems, the medullary and cortical system. Mesodermal cells contribute to the development of adrenal cortex, the gonads and the liver; these three tissues are active in steroid metabolism in the feotus (*Nelson, 1987*).

The adrenal cortex is Mesodermal in origin. During the 4th week of embryonal life, the Mesothelial buds appear at the level of the upper third of mesonephron and project into the celom at each side of the root of the dorsal mesentry. Eventually, they coalesce to form a compact mass of cells; the adrenal cortex, lateral to the aorta (*Dluhy et al., 1979*).

These mesohelial cells proliferate and grow to differentiate into larg acidophilic cells forming the feotal cortex, latter a further mesothelial proliferation occure forming smaller cells that cover the outer surface of the feotal cortex.

As development proceeds, the outer smaller cells become arranged into zona glomerulosa and zona fasciculata of definitive cortex.

The surrounding mesenchyme forms the capsule and connective tissue of the cortex. (Fig. 1).

The cortical buds that do not join the main cellular mass disappear or form accessory adrenal tissue in the paraortic area, in the region of adrenal, kidney, celiac plexus, in the pelvis, in spermatic vessels, testis, broad ligament or ovary (*Dluhy et al., 1979*).

The adrenal medulla is ectodermal in origin being derived from cells of the neural crest. At about the 7th week of gestation the primordial cells of the adrenal cortex is invaded by sympathetic neural element (*Nelson, 1987*). (Fig. 1)

These cells ultimately give rise to sympathoblast (which developed into chromaffin cells). (*Dluhy et al., 1979*).

About one week later these cells begin to differentiate into the chromaffin cells capable of synthesizing and storing catecholamines; the methyl transferase which convert norepinephrine to epinephrine develops later (*Nelson, 1987*).

Numerous small groups of chromaffin cells are also found in association with the prevertebral sympathetic ganglia, the coeliac, mesenteric, renal, adrenal and hypogastric plexus.

A large collection above the aortic bifurcation is called the organ of Zuckerkandle. In the bladder chromaffin cells are also found within the sympathetic nerve fibers, pheochromocytoma frequently develops in these accessory sites. Accessory adrenal tissue may consist of cortical or medullary tissue alone or may contain both (*Dluhy et al., 1979*).

The adrenal glands remain close to their point of origin while the kidneys ascend from the pelvis during the 6th and 8th weeks untill they meet the adrenal glands which mold themselves to the countours of the upper poles of the kidneys at the 9th and 10th week of gastation (*Gray's, 1989*).

Table (1): Anomalies of the adrenal glands

Defect	Origin	Frequency	Remarks
Adrenal agenesis	4th week	Uncommon	Associated with ipsilateral absence of kidney
Adrenal fusion	6th week	Rare	Associated with fused kidneys
Adrenal hypoplasia	?	Rare	Associated with anencephaly (usually lethal)
Adrenal heterotopia	8th week	Uncommon	Within capsule of liver or kidney; asymptomatic
Accessory adrenal tissue	4-5th week	Common	Usually cortical tissue only, asymptomatic

Gray's (1989)

Anomalous location of the adrenal tissue is clinically important for the following reasons: (Table 1)

a. Hyperplasia in the accessory adrenal tissue may produce continued adrenal activity after adrenalectomy for Cushing's syndrome or for metastatic cancer.

b. Neoplastic transformation of heterotropic or accessory tissue may take place (*Harrison, 1979*).

In a fetus 2 months old the adrenals are larger than the kidneys, but from the 4th month, the kidneys grow rapidly,