UROLOGICAL COMPLICATIONS DURING PREGNANCY AND LABOR

Essay

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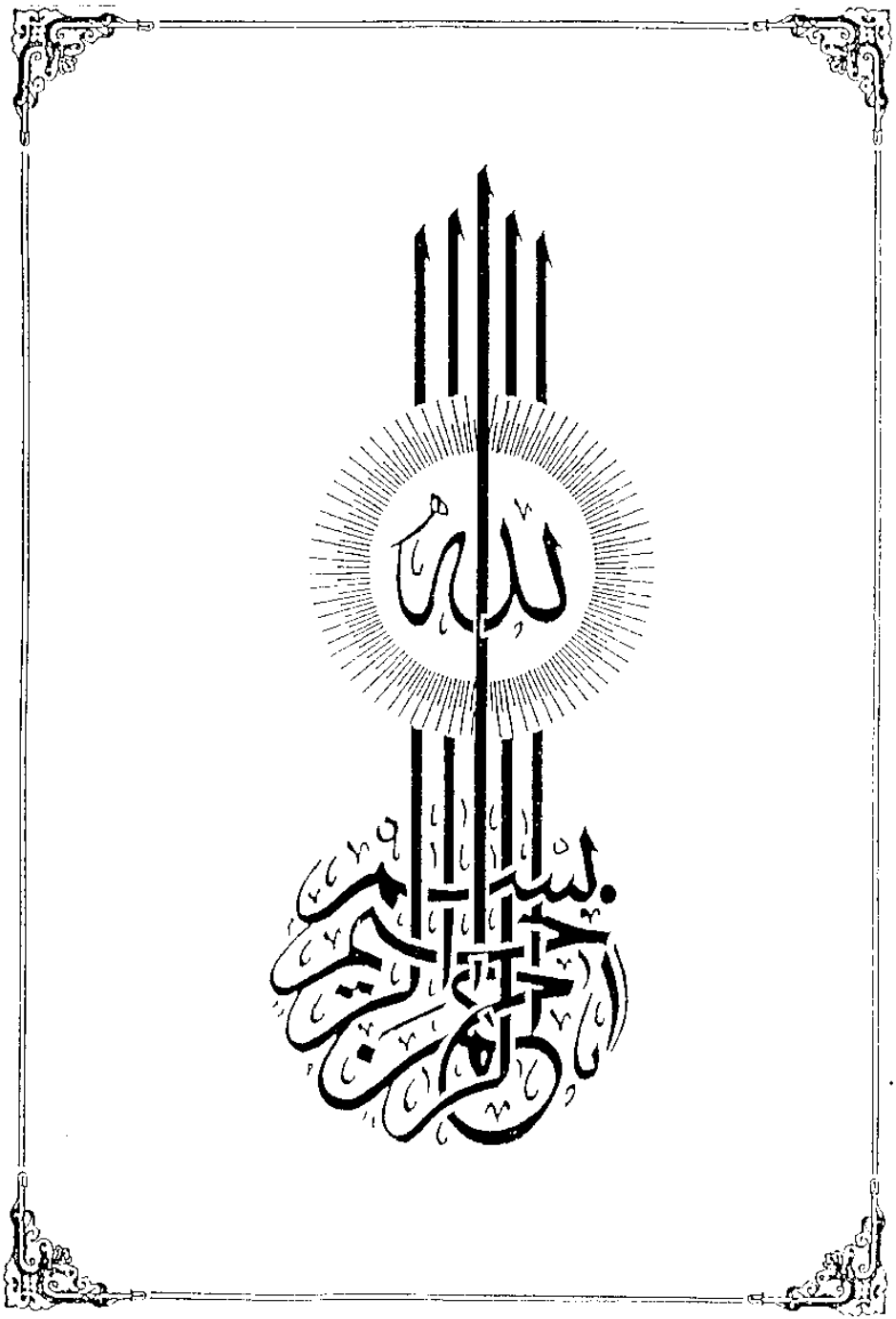

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CONTENTS

1)Introduction.....1

2) Morphological & physiological urinary changes during pregnancy.....3

3) U.T infections during pregnancy.....25

4) Pregnancy in patients with urological diseases.....44

5) Urological complications during labor.....72

6) Pregnancy & labor in patients with renal transplants.....87

7) Summary.....103

8) References.....106

9) Arabic summary.....120

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INTRODUCTION

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INTRODUCTION

The pregnancy is accompanied by considerable morphological and physiological changes to face the need of rapidly growing foetus and to prepare the mother's body to pass the stress of parturation, puerperium and lactation.

These changes are most remarkable in the urinary tract probably because of its proximity to and its common embryologic origin with the reproductive system.

These profound changes raise two major clinical problems:-

a) The increased risk of urinary tract infections.
b) The erroneous interpretation of diagnostic test results.

Recognition of these changes is essential if early signs of renal dysfunction are to be detected or if sound advice is to be given to those women with renal problems either to conceive or to continue the pregnancy.

The process of labor also carries some traumatic lesions to the urinary tract especially to the bladder and ureters.

The aim of this essay is to discuss the morphologic, the physiologic changes, the effect of renal diseases on pregnancy

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and fetal out come, the effect of pregnancy and labor on a pre-existing renal disease, and the effect of pregnancy and labor on patients with renal transplants.

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MORPHOLOGICAL & PHYSIOLOGICAL URINARY CHANGES DURING PREGNANCY

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MORPHOLOGICAL AND PHYSIOLOGICAL URINARY CHANGES DURING PREGNANCY

- The urinary system adapts itself for pregnancy by morphological and physiological changes.

MORPHOLOGICAL URINARY CHANGES DURING PREGNANCY

1) **KIDNEYS** : The kidneys almost certainly enlarge during pregnancy because both renal vascular volume increase with perhaps a degree of hypertrophy. The addition of new nephrons has never been demonstrated.(4)

- Excretory urography performed immediately (within 5 days) after delivery reveals that renal size is consistently greater than that predicted by standard hight/weight normograms and when it is repeated 6 months later it indicates a decrease in renal length by approximately 1 cm to non pregnant size. This increase also occurs in women with moderate intrinsic chronic renal disease, though the magnitude of the change may be less pronounced.(57)

The average weight of two normal kidneys during pregnancy is about 307 grams.

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Histological assessment indicated that glomerular size but not cell number was increased in pregnancy and the microscopic structure of the kidney is similar in pregnant & non pregnant women.(94)

RENAL PELVIS AND URETERS :- (FIGURE 1)

The most striking anatomical change in the urinary tract is dilatation of the calyces, renal pelvis & ureter, the so called physiological hydroureter of pregnancy. These changes are the earliest, most established morphological changes that can be seen as early as the end of the first trimester and by the third trimester they are present in 90% of women.(57)

ETIOLOGY :-

The etiology of physiologic hydroureter is disputed and several causative mechanisms have been postulated to account for :-

A) OBSTRUCTIVE THEORY :-

External compression of the ureters during pregnancy by :-

a) Direct pressure from the gravid uterus :-

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*Figure 1: shows the physiologic hydronephrosis of pregnancy.

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There is no doubt that as pregnancy progresses a supine or upright posture may cause partial ureteric obstruction as the enlarged uterus compresses the ureters at the pelvic brim, however the changes occur early at 8-12 weeks gestation.(65)

In 1956 Eastman stated that; "ureteral dilatation was more extensive in the primigravida". This may be explained by the fact that the abdominal wall of the P.G. produces more resistance than the lax abdomen of the multigravida, hence contributing to the increased pressure of the uterus on the ureters. This obstruction further supports the external compression theory.(26)

b) Pressure on the ureter by a dilated ovarian venous plexus especially on the right side.(3)

B) HORMONAL THEORY :-

Another possible mechanism for physiological hydroureter of pregnancy is hormonal.

1) ESTROGEN & PROGESTRONE :-

It was suggested that increased level of progestrone, gonadotrophins and estrogens are implicated in pyeloureteral

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dilatation. But because it is known that estrogens tend to stimulate smooth muscle contractility, the propable mechanism of hydroureter can be explained by growth promoting and interstitial fluid retaining properties of the hormone, rather than ureteral smooth muscle relaxation.(41)

2) PROSTAGLANDINS :-

The role of prostaglandin E_1, a myometrial relaxant, in the development of physiologic hydroureter is worth to be investigated.(4)

C) URETERAL THICKENING :-

Thickening of the lower part of the ureter making it rigid enough to cause mild stenosis in that area with resultant dilatation of the organ above the pelvic brim. This thickening is due to hyperplasia of the connective tissues, and hypertrophy of Waldeyer's sheath which surround the ureters as they enter the true pelvis and is more evident near the juxtavesical region of the ureter. This could also prevent hormonally induced dilatation at this level. The changes start as early as the seventh week of gestation. The cause of these changes is not well known, but it's most propably related to modification of hormonal milieu.

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All three factors discussed above play a role in the etiology of pyeloureteral dilatation, the proportional contribution of each is difficult to be determined.

The ureters tend to elongate and become more tortuous, hence probably contributing further to the development of partial obstruction. Lateral displacement of both ureters is also observed, mainly in the second half of pregnancy, and probably as a result of the growing uterus in the midline.

Ureteric dilatation terminates at the pelvic brim where the ureter crosses the iliac artery, and at this point a filling defect termed the iliac sign can be seen in an excretory urogram.(4)

The physiologic hydroureter is invariably more prominent on the right side, and right sided predominance of pyeloureteral dilatation above the pelvic brim is characteristic feature of the physiologic hydroureter of pregnancy. There are some possible etiologic factors to explain this phenomena :-

1- The iliac sign is absent on the left side because the left ureter lies only on less rigid iliac vein on that side and hence it is not exposed to compression.(25)

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2- The right ovarian venous plexus crosses the right ureter at a level between L3 and S1 in its course to join the venacava, when these venous plexus becomes remarkably dilated during pregnancy, partial ureteral compression ensues with resultant dilatation and stasis. The left ovarian vein passes parallel to the course of the left ureter to join the renal vein.(3)

3- Dextrorotation of the uterus especially towards the later part of pregnancy, further adds to the ureteral compression of the right side.

Modern urometry has demonstrated that pregnancy did not seen to modify the frequency of ureteric contractions. However, a decrease in the mean contractile pressure was observed in the lower part of the ureter very early in gestation. In the upper part of the ureter, both the contractile mean pressure and the ureteral tonus increased as pregnancy advanced, reaching maximum levels toward term. Further more, there is hypertrophy of the ureteric smooth muscles and hyperplasia of its connective tissues so that the concept of toneless, flappy ureters, with muscle paralysis by the hormonal milieu of pregnancy is erroneous.(70)

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