INTRODUCTION

Urinary tract infections are the most common medical complication of pregnancy. Asymptomatic bacteriuria is the most prevalent of these infections and it is defined as, the finding of greater than (100,000) colony forming units per mL of urine of a single pathogen in two consecutive, midstream, clean catch urine specimens or one catheterization specimen. Lower colony counts in asymptomatic women usually represent contamination ***(Hooton, 2002).***

The physiologic changes of pregnancy predispose patients to bacteriuria. These physiological changes include urinary retention from the weight of the enlarging uterus and urinary stasis due to ureteral smooth muscle relaxation (caused by increases in progesterone). Although progesterone influence causes a relative dilation of the ureters, ureteral tone progressively increases above the pelvic brim during pregnancy. However, controversy exists as to whether bladder pressure increase or decrease during pregnancy. In addition, glucosuria and aminoaciduria during pregnancy provide an excellent culture medium for bacteria in areas of urine stasis. These changes, along short urethra (approximately three to four centimeters in females and difficulty with hygiene due a distended, pregnant belly, cause urinary tract infections (UTIs) to become a common occurrence for pregnant women ***(Harris et al., 1981).***

Urinary tract infections can involve mucosal tissue (cystitis) or soft tissue (pyelonephritis). Anatomically, the

infection can be limited to the lower urinary tract (cystitis involving the bladder and urethra) or the upper tract (pynephritis). Complicated urinary tract infections can occur in either the upper or lower urinary tract but are accompanied by an underlying condition that increase the risk for failing therapy, such as obstruction, urologic dysfunction, or resistant pathogens. Most urinary tract infections occur via an ascending route ***(Hill et al., 1986).***

The incidence of pyelonephritis during pregnancy is consistently higher in women with asymptomatic bacteriuria than in women with sterile urine at the time of the first prenatal visit. Fifteen to fifty percent of pregnant women with untreated asymptomatic bacteriuria will develop pyelonephritis during pregnancy, compared with one to two percent of those without asymtpoamtic bacteriuria ***(Stamm et al., 2001)***.

A study was done in ***Philippines (1997)*** about the prevalence of asymptomatic bacteriuria among pregnant females. It denoted that the prevalence there was 2-12% with higher rate in low socioeconomic status, anaemic women, increased maternal age and high parity ***(Maranchie et al., 1997).***

Escherichia coli is the most common cause of urinary tract infections, accounting for eighty to ninty percent of cases. It originates from fecal flora that colonize the periurethral area (ascending infection). Klebsiella, Enterobacter and Proteus species cause most of the remaining cases. Gram-positive organisms, particularly Enterococcus faecalis and group B streptococcus (GBS), are also clinically important pathogens.

Infection with staphylococcus saprophyticus, an aggressive, community acquired organism, can present with upper urinary tract disease, and the infection is more likely to be persistent or recurrent ***(Villar et al., 2000).***

Clinical trials demonstrated important reduction in many of adverse effects by proper screening and treatment by antibiotics as amoxillin (cephoperazone nitrofurodantoin) ***(Curzik et al., 2001).***

AIM OF THE WORK

The aim of this work is to assess the prevalence of asymptomatic bacteriuria in primi gravid patient during mid trimester in those attending Kasr El Aini Hospital obstetric outpatient clinic from July to December 2008.

THE MORPHOLOGICAL CHANGES OF THE RENAL TRACT IN PREGNANCY

The morphology of the urinary tract is altered considerably during gestation. Changes occur as early as the first trimester and increase as pregnancy progresses. The edema and hyperemia that occur in the bladder along with its decreased tone predispose the patient to asymptomatic bacteriuria. The bacteria can then be transmitted to the ureters via the vesicoureteral reflux. The increase in urinary volume in the ureteropelvic system enhances the growth of the organisms and hence is thought to be the major cause of the increased incidence of pyelonephritis in pregnancy ***(Beydoun, 1985).***

I. Anatomical Changes

Pregnancy-related hormonal and mechanical factors induce changes in the renal collecting system that lead to urinary stasis, which is crucial to the pathophysiology of urinary tract infection in pregnancy. These changes can be seen as early as six weeks of gestation and may not resolve until six to twelve weeks postpartum**.** The pathogenesis is not completely defined and is probably related to more than one factor, with differing contributions from each factor at each stage of gestation (hormonal and/or mechanical) ***(Edmund et al., 2008)***.

The urinary tract undergoes a variety of anatomic changes during normal pregnancy. An appreciation of the maternal physiologic adaptations that occur in the renal system during pregnancy is fundamental to the understanding and proper clinical management of normal pregnancy, renal disorders in the gravid patient, and pregnancy specific conditions such as pre-eclampsia ***(Jeyabalan and Lain, 2007).***

a) Kidneys:

Both kidneys increase in size by 1 to 1.5 cm during pregnancy. This is primarily due to an increase in renal vascular and interstitial volume. There are no histological changes or change in number of nephrons. The renal pelves and caliceal systems are dilated as a result of progesterone effects and compression ***(Edmund et al., 2008).***

Renal pelvis and ureters:

Ureters — Ureteral dilatation during pregnancy results from hormonal effects, external compression, and intrinsic changes in the ureteral wall ***(Edmund et al., 2008)***:

- ***High concentrations of progesterone*** reduce ureteral tone, peristalsis, and contraction pressure leading to dilatation of the calyceal pelves and upper ureters (ie, physiological hydroureter of pregnancy).

- Hydronephrosis typically occurs and is more common on the right than left side (90 versus 10 percent). ***External compression of the right ureter*** may be due to dextrorotation of the uterus by the sigmoid colon, kinking of the ureter as it crosses the right iliac artery, or proximity to the right ovarian vein.

- ***The vessels in the suspensory ligament of the ovary enlarge*** and may compress the ureter at the brim of the bony pelvis, thus causing dilatation above that level.

- ***Uterine enlargement*** may cause the ureters to become elongated, tortuous, and displaced laterally as pregnancy advances. In rare cases, compression of the ureters may cause pain and lead to renal failure, which resolves with placing the mother on her side, insertion of stents, and/or delivery.

- ***Hypertrophy of Waldeyer's sheath*** (ie, the longitudinal muscle bundles in the lower ureter) causes mild stenosis in the juxtavesical region, thereby contributing to dilatation of the ureter above the pelvic brim.

The earliest recognized and most established morphologic changes in the urinary tract during gestation are dilatations of the renal pelvis and ureters, the so-called

physiologic hyroureter of pregnancy. These findings were first described in autopsy materials as early as the mid nineteenth century and clinically observed by retrograde pyelography in 1925 ***(Fainstat, 1963)***. They become evident in the first trimester and persist to term and for a good part of the puerperium ***(Schulman and Herlinger, 1975; Marchang, 1978).***

The volume of the ureters in pregnancy may increase 25- fold and contain as much as 300 ml of urine. Furthermore, the urine flow progressively decrease as pregnancy advances. in ten term pregnant patients with predominantly right sided hydroureter, ***Bergstrom (1975)*** demonstrated by radioisotope renography a five fold delay in post renal excretion on the affected side.

Eastman in (1956), stated that ureteral dilatation was more extensive in the primigravida. This may be explained by the fact that the abdominal wall of the woman pregnant for the first time produces more resistance than the lax abdomen of the multigravida, hence contributing to the increased pressure of the uterus on the ureters. This observation further supports the external compression theory.

No theory alone, however, adequately explains all the characteristic changes observed. The bulk of the available information suggests that while all three factors discussed above play a role in the etiology of pyeloureteral dilatation,

the proportional contribution of each is difficult to determine. The sequence of events is probably the following: initially, the increased levels of sex hormones produce thickening of the lower part of the ureter and softening of its upper part. Those changes lead to mild dilatation of the upper portion of the organ. As pregnancy progresses, partial ureteral compression occurs at the pelvic brim by any or all of the various structures discussed previously (i.e., uterus, fetus, iliac arteries, ovarian veins), promoting further dilatation and urinary stasis ***(Beydoun, 1985).***

Right sided preponderance:

Right sided preponderance of pyeloureteral dilatation above the pelvic brim is a characteristic feature of the physiologic hydroureter of pregnancy. By performing IVPs on 220 pregnant patients with presumably normal urinary tracts, ***Schulman and Herlinger (1975)*** demonstrated that in 86% of their subjects the right ureter was more filled than the left. In addition, the authors observed that the extent of the dilatation and the percentage of severely dilated ureters (on an arbitrary scale) were also higher on the right side.

The cause of this unequal degree of dilatation is not very well understood. Early authors stated that the right sided preponderance was due to a greater kinking of the right ureter as it crossed the iliac vessels at the pelvic brim.

More recently, ***Dure-Smith (1970)***, demonstrated a radiologic filling defect of the right ureter as it crossed the iliac artery at this level. This filling defect was termed the iliac sign and was absent on the left, because, as pointed out by the author, the ureter lies only on the less rigid iliac vein on that side and hence is not exposed to compression ***(Dure-Smith, 1970).***

Another possible etiologic factor in the preponderance of right ureteral dilatation is the relationship of the right ovarian vein to the right ureter. The vein on the right side is described as composed of multiple channels that join into several multiple branches and crosses the right ureter at a level between L3 and S1 ***(Clark, 1997)***. On the other hand, the left ovarian vein parallels the course of the ureter, to join the renal vein. Very rarely, however, it or one of its very few branches may cross the ureter on that side. Because the right ovarian vein complex becomes remarkably dilated during pregnancy, partial ureteral compression ensues, with resultant dilatation and stasis ***(Bellina et al., 1970 and Clark, 1997).***

b) Ureteral tone and pressure:

Early investigations regarding the changes in tone and motility of the ureters during pregnancy produced conflicting results. These discrepancies were attributed to the different methods employed ***(Beydoun, 1985).***

They concluded that the increase in tone during pregnancy was due to compression and obstruction of the ureter at the pelvic inlet by the pregnant uterus ***(Rubi and Sala, 1968).***

Concomitant to the dilatation and alterations in contraction pressure and tonus described above, the ureters undergo more subtle and probably less clinically significant changes during pregnancy ***(Schulman and Herlinger, 1970)***. They 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.

c) Bladder:

As a probable reflection of the atonic effects of progesterone on smooth muscles, the tone of the bladder during pregnancy is progressively decreased, and its capacity increase to reach more than a liter (double its volume) toward term**.** The trigone area, presumably as a result of estrogen stimulation, undergoes moderate

hyperplasia and muscular hypertrophy, similar to the changes that take place in the lower part of the ureter**.** Mainly during the latter part of the second trimester and during the third, because of the enlarging uterus, together with the hyperemia of the pelvic organs, the bladder is displaced anteriorly and superiorly. This process advances throughout pregnancy; and as term approaches, especially with the descent of the presenting part, the trigone area changes from a concave to a convex surface, rendering diagnostic and therapeutic procedures through the cystoscope more difficult ***(Cunningham et al., 2005).***

The bladder mucosa undergoes no major changes during pregnancy other than congestion and increase in size and tortuosity of the blood vessels, a phenomenon compatible with the generalized hyperemia of the pelvic organs. Toward term, and when the presenting part becomes engaged, however, the bladder mucosa becomes more edematous and easily traumatized and therefore more susceptible to infection ***(FitzGerald and Grazian, 2007).***

Vesicoureteral reflex:

The bladder in pregnancy becomes progressively more flaccid, and its tone decrease. These changes have been shown to cause incompetence of the vesicoureteral valve, which creates a reflux from the bladder to the ureters. Moreover, the anatomic alterations of the bladder base produced mainly toward the end of gestation by the

enlarging uterus cause stretching of the trigone with lateral displacement of the intravesical portion of the ureters. This shortens the terminal ureter and results in a decrease in intraureteral pressure. When the intravesical pressure increases during micturition, regurgitation of urine from the bladder to the ureters occurs ***(Mattingly and Borkowf, 1978).***

Bladder flaccidity may cause incompetence of the vesicoureteral valve. This change, combined with increased intervesical and decreased intraureteral pressure, appears to result in intermittent vesicoureteral reflux ***(Nel et al., 2001).***

II. Renal functional changes during pregnancy:

Pregnancy is associated with significant functional changes in the kidney and its collecting system. These changes begin to occur shortly after conception and may persist for several months postpartum ***(Citiak and Newton, 1985).***

Hemodynamic changes and renal physiology in pregnancy:

Normal pregnancy is characterized by increased renal perfusion and several, usually minor changes in extracellular composition, including chronic respiratory alkalosis and mild hyponatremia (Table 1). Increased renal perfusion is attributable to the increased cardiac output that occurs during pregnancy.

Table (1): Hemodynamic and renal changes during normal pregnancy

Systemic hemodynamics

- Increase in cardiac output
- Fall in vascular resistance and blood pressure
- Blood volume expansion

Renal function and electrolyte balance

- Increase in glomerular filtration rate
- Chronic respiratory alkalosis
- Hyponatremia due to resetting of osmostat
- Increased ADH metabolism and polyuria in selected women.

(DeSweit, 2002)

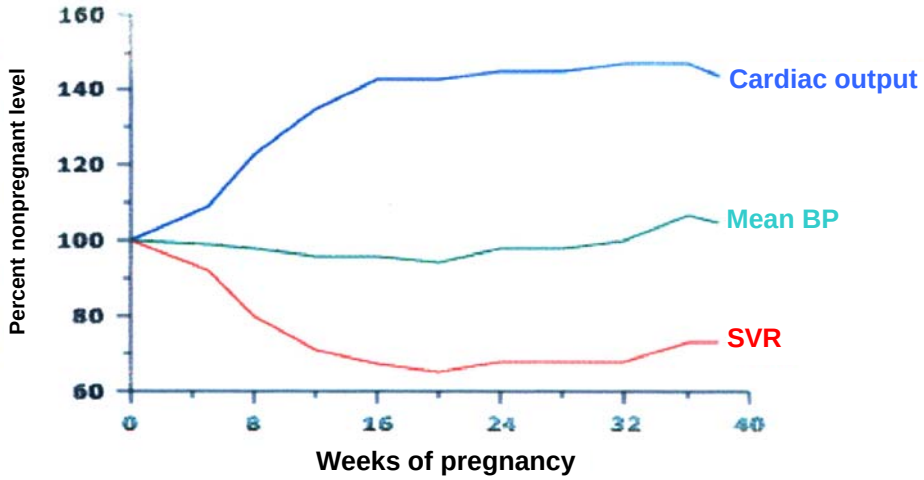

Figure (1): Hemodynamic changes in normal pregnancy ***(DeSweit, 2002).***

One of the most striking features of pregnancy is that blood pressure and peripheral vascular resistance fall soon after conception, when pregnant baboons were studied serially with Swan-Ganz catheters from the onset of

pregnancy, decreases in right atrial, systemic, and pulmonary vascular resistance were found by the fourth week of gestation. Cardiac output increases in the first trimester of pregnancy, reaching a maximum of 30% to 40% above the nonpregnant level by the 24th week of gestation ***(DeSweit, 2002).***

Blood volume increases approximately 50% in pregnancy beginning in the first trimester with a rise in both plasma volume and red blood cell volume. A greater increase in plasma than in red blood cell volume causes the physiologic anemia of pregnancy. Expansion fo maternal extracellualr volume continues throughout pregnancy with a cumulative Na+ retention o fbetween 500 and 900 mEq ***(Hytten and Leitch, 1971).***

Na+ retention precedes at the rate of approximately 20 to 30 mEq/wk, which results in a mean weight gain of 12.5 kg. the major stimulus for the kidney to retain Na+ is the decrease in peripheral vascular resistance, a recognized stimulus for Na+ retention ***(Phippard et al., 1986).***

Plasma volume was not observed to be expanded until 8 weeks after Na+ retention. The expanded extracellular volume may cause edema, which is present in 35% to 83% of healthy pregnant women, the frequency depending on the effort made to detect it ***(Robertson, 1971).***