Association of lewis phenotype blood groups with recurrent urinary tract infection in female


The association

The study aimed to detect the relationship between histocompatibility and the Lewis blood group system and recurrent urinary tract infections (UTIs) in women. The study included 22 cases of UTIs (patients and controls) and 52 healthy women, all aged less than 30 years old. The study was conducted at the Women's Hospital and the General Hospital in the period from the second quarter of 2010 to the second quarter of 2011. The results showed a statistically significant increase in the Lewis blood group system in healthy women compared to the control group (p<0.05). However, there was no significant difference in the UTI cases compared to the control group (p>0.05). The study confirmed the absence of the Lewis blood group system in cases of pregnancy and breast cancer. The study also indicated that the Lewis blood group system is associated with the development of recurrent UTIs and the risk of developing serious infections. This study adds to the understanding of the genetic predisposition to recurrent UTIs.
Abstract

Background: Histo blood group antigens with carbohydrate molecules are found on the surface of RBC and uroepithelial cells, which influence human susceptibility to recurrent urinary tract infection (RUTI).

Objective: Of this study was to determine if a link exists between susceptibility to RUTI and host genetic phenotypes that define either an individual’s ability to respond immunologically to some antigens or the composition of host cell surface molecules. The interaction of pathogen and erythrocyte membrane may reflect antigenic similarity, adhesion through specific receptors, or modulation of antibody response. Detect the ABO blood group distribution and RUTI in accordance to lewis phenotype antigens.

Methods: The studied groups attended to Babylon Maternity and Pediatric Teaching Hospital from November, 2010 to November 2011. The study is carried out on 22 female as control groups, and 52 female as patient group. All patients had a history of RUTI, defined as a minimum of three infections in the previous year; however, most patients experienced more than three UTIs per year and had recurrent infections for several years. Results of excretory urography or renal ultrasound and cystoscopy performed on all patients were normal or showed only inflammatory changes. also using physical examination and urinalysis. Circulating immunoglobulin concentrations (IgG) was evaluated by applying Mancini single radial immunodiffusion technique for a RUTI patients and control involved in this study.

Results: - Regarding lewis phenotype blood group in RUTI women, there's association between non-secretor phenotype and predisposition to RUTIs in post menopause women and disappears in pregnant women in patient and control, while there's no association between secretor phenotype and predisposition to RUTIs in young women. The important result in this study appearance percent of weak secretor phenotype was (14%) in RUTI and control women.

WBC count shows a highly significant increase (p<0.001), significant increase (p<0.05) of granulocyte and lymphocyte count.
in RUTI women in patents comparing with control groups. and significant increase (p<0.05) in secretor than non-secretor RUTI groups of WBC ,lymphocyte and granulocyte count. Also it was found that RUTI was associated with an increase in IgG concentration more than normal IgG concentrations among secretor RUTI patients revealed an increase more than non-secretor RUTI. While, urine analysis shows all patents were suffering with pyurea, these severity was highly significant increase (p<0.001) in non-secretor than secretor RUTI group.

**Conclusion:** The results of this study support Predisposition RUTI has been associated with ABO histo Blood group and secretor status. The distribution of non-secretors was significantly higher in women with RUTI than in the control, and higher in postmenopausal and pregnant women than in young women. These results suggest that non-secretor status is associated with the genetic susceptibility to RUTI.

**Introduction**

“Histoblood group system” (HBGAs) are complex carbohydrates linked to glycoproteins or glycolipids that are present on the red blood cells and mucosal epithelial cells or as free antigens in biological fluids such as blood, saliva, intestinal contents and milk (1,2,3). These antigens are synthesized by sequential additions of monosaccharides to the active portion of the antigen precursors by several glycosyltransferases that are controlled mainly by the ABO, Lewis, and secretor gene families (4). A women who are not secretors of blood group antigens have a three- to four-fold greater risk of developing recurrent UTIs (5).

There are strong relations between individuals’ susceptibilities to some diseases and their histoblood groups, as well as their secretor status (6). The secretor gene encodes for enzymes (glycosyltransferases), which become active in mucin-secreting cells like goblet and mucous cells of mucous membranes and different glands, resulting in the secretion of the corresponding blood group antigens in the body fluids(7). These enzymes are under the control of inherited genes, which are A, B, H (FUT1)
genes and secretor (FUT2) genes (8). H (FUT1) and secretor (FUT2) genes are separate but closely linked. Lewis blood group phenotypes are important in determining the secretor status of an individual, as Lewis genes (FUT3) are closely linked to secretor (FUT2) and H (FUT1) genes(9). Subjects are either Lewis negative or Lewis positive in Lewis-negative individuals.

Recurrent urinary tract infection (UTI) refers to ≥2 infections in six months or ≥3 infections in one year. Most recurrences are thought to represent reinfection rather than relapse, although occasionally a persistent focus can produce relapsing infection(10).

Inherited factors may also play a role in predisposing postmenopausal women to recurrent UTIs. This study reported a history of family UTIs significantly more frequently than did the control patients. Nonsecretor status was also an inherited characteristic strongly associated with recurrent UTI in the postmenopausal and pregnant women. It is interesting that the previously reported association of nonsecretor status with recurrent UTI has been most evident in populations of women aged >35 years. The mean ages of the populations described by (11), in whom nonsecretor status was associated with recurrent UTI, were 38 and 55 years, respectively, and both populations included many postmenopausal women. The mechanism through which nonsecretor status predisposes to recurrent UTI is probably the presence on vaginal and uroepithelial cells of 2 unique nonsecretor-associated glycolipids that serve as binding sites for specific Escherichia coli adhesions. It is possible that expression of these nonsecretor-associated glycolipids varies with age or hormonal status. Alternatively, nonsecretor status may become more important in older women when other risk factors such as sexual activity and spermicide exposure recede (12).

**Aim of the study:** To clarify the following:

1-Association between the expression of Lewis antigens on urothelial plasma membranes and a resistance to urinary tract infection.
2- Association between non-secretor status and recurrent urinary tract infection.
3- The risk value of the histoblood group antigenic type in relation to RUTI.
4- The possibility if there is a family genetic basis of lewis antigen phenotypes and RUTI.

Materials and Methods
Patients and control
Seventy four female were conducted during this search in Maternity and Pediatrics Teaching Hospital in Hilla city through period lasted from November 2010 to November 2011. The cases were diagnosed and examined by urologist in depending of history, clinical examinations (fever, chills, flank pain, and cost vertebral angle tenderness), GUE, culture and ultrasound. Twenty healthy women as control included in this study. All patients and healthy were underwent the following laboratory investigation that included : GUE & Cultures, blood group, RH, ESR, PCV, MCV, WBC count and differentiation, neutrophil activity, total IgG, RBC count, platelet count, blood urea, blood creatinin and blood uric acid. Patients young women (unmarried) were (15), Patients pregnant women were (23) and postmenopausal women (more than 45 years old) were (14) patients. While the control group were include; (8) healthy young unmarried women, (7) healthy pregnant women and (8) healthy post-menopausal women were used as comparable group.

Methods:
Determination of lewis phenotype antigen:- The determination of lewis phenotype is done according to the procedure recommended by the company (LORNE LABORATORIES LTD). The assay principle The reagents will cause agglutination (clumping) of test red cells, that carry the corresponding Lewis antigen, after centrifugation. No agglutination generally indication the absence of the corresponding Lewis antigen.
Measurement of total serum IgG:- The method involves antigen diffusing radially from a cylindrical well through an agarose gel
containing an appropriate monospecific antibody. Antigen-antibody complex are formed which, under the right conditions, will form a precipitin ring. The ring size will increase until equilibrium is reached between the formation and breakdown of these complexes, this point being termed ‘completion’. At this stage, a linear relationship exists between the square of the ring diameter and the antigen concentration. By measuring the ring diameter produced by a number of samples of known concentration of the antigen in an unknown sample may then be determined by measuring the ring diameter produced by that sample and reading off the calibration curve according to the procedure recommended by the company (Birmingham, Germany) (13).

**Total leucocytes count:** Blood was drawn in a clean and dry WBC pipette up to the mark 0.5 and the outside of the pipette was wiped off with a gauze. Then diluting fluid (Turk's solution) was drawn up to mark 11 (dilution 1:20), the contents were mixed for three minutes; 1-2 drops were discarded then counting chamber (Neubaur chamber) was filled. It was left for three minutes to let the cells for setting down and then the chamber was examined under 40X objective lens of the microscope to count WBCs in the four corners secondary squares (14).

**Microscopic Urinalysis:** Take (10) ml of urine sample were transferred to clean centrifuge tube and centrifuged at 4000 rpm for 5 minute. 

**Urinary Cultures:** Cultures that recover more than $10^5$ bacteria per mL of voided urine have been considered evidence of UTI (21).

**Results**

**Lewis Blood Group Phenotype:** The patients and control under the study were divided into three group: young unmarried, pregnant and menopausal women and were divided according to their histo blood group antigen.

**Lewis blood group phenotype in young unmarried female:** The distribution of Lewis blood group phenotypes among the 15
patients from 52 patients with RUTI and 8 young women (unmarried) from 23 (Table 1). The difference in the proportions of Le(a-b+), Le(a+b-) , Le(a-b-) and Le(a b+).phenotypes in the control and infection groups was not statistically significant ($X^2 = 4.139$, df=3, $P=0.095$).

(Table 1) The distribution of Lewis phenotypes blood group of RUTI and control groups in young unmarried women

<table>
<thead>
<tr>
<th>phenotype</th>
<th>RUTI n=15</th>
<th>Control n=8</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Le(a-b+)</td>
<td>7/15(46%)</td>
<td>3/8(37%)</td>
<td>10</td>
</tr>
<tr>
<td>Le(a+b-)</td>
<td>3/15(20%)</td>
<td>1/8(13%)</td>
<td>4</td>
</tr>
<tr>
<td>Le(a-b-)</td>
<td>3/15(20%)</td>
<td>2/8(25%)</td>
<td>7</td>
</tr>
<tr>
<td>Le(a+ b+)</td>
<td>2/15(14%)</td>
<td>2/8(25%)</td>
<td>4</td>
</tr>
</tbody>
</table>
$X^2 = 4.139$  | df=3      | $p>0.05$    | insignificant |

Lewis blood group phenotype in pregnant women:- The distribution of Lewis blood group phenotypes among the 23 patients from 52 with RUTI and 7 pregnant from 23 as control group. (Table2). The difference in the proportions of Le (a-b+), Le (a+b-) , Le(a-b-) and Le(a+ b+).phenotypes in the control and infection groups was statistically significant ($X^2 = 8.159$, df=3, $P=0.009$).

(Table 2) The distribution of Lewis blood group phenotypes in pregnant women

<table>
<thead>
<tr>
<th>phenotype</th>
<th>RUTI n=23</th>
<th>Control n=7</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Le (a-b+)</td>
<td>1/23(4%)</td>
<td>3/7(42%)</td>
<td>4</td>
</tr>
<tr>
<td>Le (a+b-)</td>
<td>5/23(21%)</td>
<td>2/7(28%)</td>
<td>5</td>
</tr>
<tr>
<td>Le (a+b+)</td>
<td>4/23(17%)</td>
<td>1/7(14%)</td>
<td>5</td>
</tr>
<tr>
<td>Le (a-b-)</td>
<td>13/23(56%)</td>
<td>1/7(14%)</td>
<td>14</td>
</tr>
</tbody>
</table>
Significant $P<0.05$  | df=3      |

Lewis blood group phenotype in post menopause Women:- The distribution of Lewis blood group phenotypes among the 14 patients from 52 with RUTI and 8 Postmenopausal from 23 as control group. (Table3). The difference in the proportions of Le(a-b+), Le(a+b-) , Le(a-b-) and Le(a+ b+).phenotypes in the control and infection groups was statistically significant ($X^2 = 8.469$, df=3, $P=0.006$).
**Table (3) distribution of Lewis blood group phenotypes in the control and infection in Post Menopause women's**

<table>
<thead>
<tr>
<th>phenotype</th>
<th>RUTI n=14</th>
<th>Control n=8</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Le(a+b+)</td>
<td>2/14(14.3%)</td>
<td>6/8(75%)</td>
<td>8</td>
</tr>
<tr>
<td>Le(a+b-)</td>
<td>3/14(21.4%)</td>
<td>1/8(12.5%)</td>
<td>4</td>
</tr>
<tr>
<td>Le(a-b-)</td>
<td>7/14(50%)</td>
<td>1/8(12.5%)</td>
<td>8</td>
</tr>
<tr>
<td>Le(a+ b+)</td>
<td>2/14(14.3%)</td>
<td>0/8(0%)</td>
<td>2</td>
</tr>
</tbody>
</table>

\[X^2=8.469\] \[df=3\] \[P<0.05\] significant

**Lewis Blood Group weak Phenotype: -** Distribution of weak secretor show total percentage of weak secretor (14%) is shown in (Table4).

**Table (4) Distribution of Lewis weak phenotype blood group in RUTI and control group.**

<table>
<thead>
<tr>
<th>Le(a+b+) phenotype</th>
<th>RUTI n=52</th>
<th>Control n=22</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>postmenopausal</td>
<td>2/14(14%)</td>
<td>0/7(0%)</td>
<td>2</td>
</tr>
<tr>
<td>young</td>
<td>2/15(13%)</td>
<td>2/8(25%)</td>
<td>4</td>
</tr>
<tr>
<td>pregnant</td>
<td>4/23(17%)</td>
<td>1/7(14%)</td>
<td>5</td>
</tr>
<tr>
<td>total</td>
<td>8/52(15%)</td>
<td>13/22(13%)</td>
<td>11</td>
</tr>
</tbody>
</table>

**Blood cell count:-**

**Blood cell count in RUTI and control groups**
There is a highly significant increase of WBC count in RUTI group than in control groups (p<0.01), and there is significant increase of granulocyte and lymphocyte count in RUTI group than in control groups (p<0.05) as shown in Table(5).

**Table (5) Comparison (Mean ± SD) between RUTI group and control group in WBC,granulocyte and lymphocyte count.**

<table>
<thead>
<tr>
<th>P-value</th>
<th>Parameters</th>
<th>Mean ± S.Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (n = 23)</td>
<td>RUTI (n = 52)</td>
</tr>
<tr>
<td>P&lt;0.01</td>
<td>WBC count x 10^9/L</td>
<td>7.243±2.0020</td>
</tr>
<tr>
<td>P&lt; 0.05</td>
<td>Granulocytes count x 10^9/L</td>
<td>5.043±1.9690</td>
</tr>
<tr>
<td>P&lt;0.05</td>
<td>Lymphocyte count x 10^9/L</td>
<td>1.843±.4154</td>
</tr>
</tbody>
</table>
**Blood cell count in secretor and non-secretor RUTI groups**

There significant increase of WBC, granulocyte, lymphocyte count and neutrophile activity in secretor than non-secretor RUTI groups (p<0.05) as shown in Table(6).

**Table (6) Comparison (Mean ± SD) in RUTI group between and secretor and non secretor of WBC, granulocyte and lymphocyte count**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean ± S.Deviation</th>
<th>Lewis antigen RUTI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Secretor N=10</td>
<td>Non secretor (n = 42)</td>
</tr>
<tr>
<td>WBC count x 10⁹/L</td>
<td>8.00±1.633</td>
<td>6.36±1.746</td>
</tr>
<tr>
<td>Granulocyte count x 10⁹/L</td>
<td>8.450±4.1137</td>
<td>6.083±2.9571</td>
</tr>
<tr>
<td>Lymphocyte count x 10⁹/L</td>
<td>2.730±6961</td>
<td>2.170±6660</td>
</tr>
</tbody>
</table>

**Immunological Parameters:-**

**Total serum IgG patients with RUTI and control group**

In present study showed that total serum IgG concentrations of patients with RUTI were between (720-1848 mg/dl), the mean was 1133 mg/dl as shown in figure(1). While the range of total serum IgG concentrations of normal individuals (control) were between (580-1520 mg/dl), with mean (960 mg/dl). Statistical analysis of data by using (T) test was showed there is significant difference between the two groups mean, p<0.05. So that total serum concentration of IgG was higher in most cases of RUTI.

![Figure(1) The difference between the means of total serum IgG concentrations in RUTI patients and control.](image-url)
Total serum IgG patients secretor and non secretor Lewis phenotype blood group with RUTI:

Difference between the means of total serum IgG concentrations in RUTI patients secretor and non secretor Lewis phenotype blood group showed that total serum IgG concentrations of patients with RUTI secretor were between (1046-1849 mg/dl), the mean was 1259 mg/dl. as shown in figure (2). while the range of total serum IgG concentrations of patients with RUTI non secretor were between (720-1365 mg/dl), with mean 1060 mg/dl. Statistical analysis of data by using (T) test was showed there is significant difference between the two populations mean.

![Figure 2](image)

Figure (2) The difference between the means of total serum IgG concentrations in RUTI patients secretor and non secretor Lewis phenotype blood group

Result of urine test

pus cell in urine:- As shown in table (7), all urine samples which had pus cells count more than 5 cells/HPF. there was highly Significant ( P<0.001) of pyuria in non secretor RUTI group than secretor RUTI group.

Table (7): Distribution of samples according to number of pus cells / HPF, secretor and non secretor with RUTI group.

<table>
<thead>
<tr>
<th>Pus cells count/HPF</th>
<th>5-10</th>
<th>11-20</th>
<th>21-30</th>
<th>31-50</th>
<th>&gt;50</th>
<th>&gt;100</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Samples secretor</td>
<td>6(12%)</td>
<td>7(14%)</td>
<td>6(12%)</td>
<td>10(20%)</td>
<td>15(29%)</td>
<td>8(23%)</td>
<td>52(100%)</td>
</tr>
<tr>
<td>Non secretor</td>
<td>5(42%)</td>
<td>3(43%)</td>
<td>1(17%)</td>
<td>1(10%)</td>
<td>0(0%)</td>
<td>0(0%)</td>
<td>10(20%)</td>
</tr>
<tr>
<td>Significant</td>
<td>P&lt;0.001</td>
<td>df=5</td>
<td>X 2 =24.439</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Discussion

Lewis blood group phenotype in young unmarried female.

Non secretor phenotypes Le(a+b-), recessive phenotypes Le(a-b-) and weak secretor phenotypes Le(a+b-) have insignificantly incidence of recurrent urinary tract infections than secretor phenotype Le(a+b+) in young women. \((p > 0.05)\). In (table 4.1) these agreement with study of Walter et al., (1979), who found the overall risk for women to develop RUTIs does not appear to be associated with any single Lewis phenotype. (16), found Blood group and secretor phenotype were not associated with RUTI in young women.

Lewis phenotypes blood group in pregnant women

Table (4.2) there is highly significant decrease of secretor phenotype) Le(a-b+) \((p < 0.001)\). and significant increase of secretor recessive phenotypes Le(a-b-)( \(p < 0.05)\). these result agreement with study (17), who found Pregnancy was associated with a higher frequency in women with the Le(a-b-) phenotype and altered distribution of erythrocyte Lewis phenotype expression during pregnancy. In both white and black populations, there was a higher than expected incidence of Le(a-b-) women and a correspondingly lower than expected frequency of Le(a-b+) phenotypes. This apparent change in distribution is most likely due to a reduction in serum transferase and the increased adhesion of the Lewis antigen to plasma lipoproteins during pregnancy. Both mechanisms result in a significant reduction of expression on the RBC membrane.

Lewis phenotypes blood group in postmenopausal women

There have been several reports that non-secretors of blood group antigens are over-represented among women with a history of RUTI. this study indicates that non-secretor phenotypes Le(a+b-), recessive phenotypes Le(a-b-) and those who are weak secretor phenotypes Le(a+b-) have a significantly incidence \((p < 0.05)\) of recurrent urinary tract infections than secretor phenotype Le(a-b+) in postmenopausal women. (table 4.8) the concluded data of the present study go together with the results of other studies (18) who found that Women with non-secretor and recessive phenotypes had
a significantly higher incidence of recurrent UTIs than women with a secretor phenotype and (19) found that non secretor status were most strongly associated with recurrent UTI in postmenopausal women.

UPEC was responsible for 78% of the documented cases (20) while many women with no obvious genetic predisposition are susceptible to RUTI, other women with inherited blood antigen factors have enhanced susceptibility to repeat episodes. Women who are non-secretors of ABO blood group antigens are 3-4 times more likely to suffer from RUTIs (21). Biochemically this is linked to the presence of unique globoseries glycolipid receptors present in non-secretors’ epithelial cells that aid in UPEC adherence (22). Sialosyl galactosyl globoside (SGG) likely plays an important role in the pathogenesis of UTI and that its presence may account for the increased binding of E. coli to uroepithelial cells from non-secretors and for the increased susceptibility of non-secretors to RUTI.

**Lewis Blood Group weak Phenotype.**

Rare individuals (Bombay and Para-Bombay phenotypes) fail to express the H antigen in erythrocyte membranes because of lack of the FUT1-encoded enzyme activity (23), while '20% of Caucasian individuals (non secretors) fail to express the H antigen in secretary fluids because of lack of the FUT2-encoded enzyme activity. in contrast to Caucasians,. Resulted data of weak secretor prevalence in this work revealed that the distribution of weak secretor was 14% in women in RUTI and control groups (Table 4.8), these results were in good agreement with previous study done by Henry et al., (1995) how found that 20%-25% of Orientals are thought to be red cell Lewis(a+b+) or weak-secretor phenotype instead of red cell Lewis(a+b-) and non secretor phenotype.

Presence of Le^a_ and Le^b_ in Le(a+b+) samples is in marked contrast to Caucasians with normal Lewis phenotypes, who have predominantly either Le^a_ or Le^b_. These results suggest that there is a range of the secretor transferees in different individuals, possibly due to different pen trance or to several weak variants. We also
show that Lewis epitopes on longer and/or more complex core chains appear to be predominant in the Polynesian Le(a+b+) samples. The formation of these extended glycolipids is compatible with the concept that in the presence of reduced secretor fucosyltransferase activity, increased elongation of the precursor chain occurs, which supports the postulate that fucosylation of the precursor prevents or at least markedly reduces chain elongation. (15). The results further suggest that Se enzyme-deficient alleles are race specific.

**Detection of blood cell in RUTI**

The results from Table (4.14) show that there is a highly significantly increase (p<0.01) in the mean of wbc count than that of control groups and show that there was significant increase (p<0.05) in the mean of granyolocyte and lymphocyte count in RUTI group than the control group. The result of wbc granyolocyte and lymphocyte count are in agreement with study (24). WHO found uncomplicated urinary tract infections caused by *E. coli* has provided an excellent model allowing investigators to unravel the interactions of uropathogenic *E. coli* (which have acquired specific genes to facilitate infection) with the host epithelium and the innate immune response that often successfully repels such infections. Results from Table (4.15) show that there was significantly increase (p<0.05) in the mean of WBC, granyolocyte and lymphocyte count in RUTI secretor group than nonsecretor RUTI group, these result indicate the secretor antigen mobile through all the body therefore stimulate the immunodefence mechanism that indicate Predisposition to urinary tract infections has been associated with non secretor status of histo blood group antigen. (6).

**Urine studies:**

**pus cell in urine:**- Regarding the above presence of abnormal numbers of white blood cells in the urine is significant , As other researcher established that, high white blood cell count present in urine, is in fact, a major symptom of UTI, and Pyuria without bacteriuria indicates elevation for tuberculosis , stones, or cancer (26).
Conclusion
1- Association between nonsecretor phenotype and predisposition to RUTIs in post menopause women and disappears this phenotype in pregnant women in patient and control. While No association between secretor phenotype and predisposition to RUTIs in young women.

2- For the first time in our country the previous study show a present weak secretor phenotype was (14%)in RUTI and control women.

3- The WBC count shows a highly significant increase (p<0.001), significant increase (p<0.05) of granulocyte and lymphocyte count in RUTI women comparing with control groups. and significant increase (p<0.05) in secretor than non secretor RUTI groups of WBC, lymphocyte and granulocyte count.

4- Total serum IgG increases in most cases of RUTI and total serum IgG increases in secretor RUTI than non secretor RUTI.

5- Urine analysis shows all patent were suffering with pyurea, these severity was a highly significant increase (p<0.001) in non secretor RUTI group than secretor RUTI group.

Recommendations
1- Recommended for complete genetic studies to show the association between RUTI and lewis phenotypes.

2- Adopts another study to detect the presence of lewis phenotype in both women and men.

3- Special attention should be directed to complete immunological studies for RUTI and lewis phenotypes antigens.

4- Adopts another study to detect the relation between secretor histo blood group antigen and renal scar.

5- Further work is needed to focused on nonsecretor status and hormonal investigation.
References
Secretor blood group alpha(1,2)fucosyltransferase gene (FUT2). Homozygosity for an enzyme-inactivating nonsense mutation commonly correlates with the non-secretor phenotype. J Biol Chem 270:4640-4649.

