EVALUATION OF A PROSPECTIC STUDY ON THE URINARY TRACT INFECTIONS IN PREGNANCY

T. FEDE, S. VALENTE, M. BERTASI
Obstetric and Gynecologic Institute,
University of Padua (Italy)

SUMMARY

The AA. considered 58 pregnant women affected by asymptomatic bacteriuria of pregnancy and 20 pregnant women with sterile urine during the first trimester of pregnancy as control group. Despite a constant treatment suggested by antibiogram the incidence of infective relapses was high but no patients were affected by important infections as acute haemorragic cystitis and acute pyelonephritis.

In control group only 15% of patients had an asymptomatic urinary infection. These patients received the same treatment of the first group. In AA. opinion, a constant and careful screening of asymptomatic urinary infections is necessary in all pregnant women to prevent acute pyelonephritis.

The urinary tract infections are very common and, in a prognostic view, they must be considered about the consequences they conduce to.

A screening program is very hard and expensive but good results were obtained when we studied only high risk groups as children and pregnant women [1].

In pregnancy the incidence of symptomatic urinary infections occurs from 4 to 7 per cent [2, 3, 4]. Among all pregnant women who present an asymptomatic bacteriuria at the beginning of the gestation about 20 to 40 per cent will be affected by a symptomatic urinary infection at the end of pregnancy and in puerperium [5]. Despite the positive results of the antibiotic therapy which greatly reduced the incidence of a permanent renal disease, it is incorrect to neglect research and constant treatment of every urinary infection in pregnant women.

Conflicting results have been reported about fetal risks during a maternal urinary infection. Many AA. [6, 7, 8, 9, 10] agree on the risk of prematurity during an acute pyelonephritis and asymptomatic bacteriuria and others report an important fall of the prematurity rate of about 60 per cent following a urinary therapy. Other AA. [11, 12, 13] report results in opposition with the preceding AA. about asymptomatic bacteriuria and their prematurity rates are either similar to those of control group or the fall of these rates after treatment of the asymptomatic bacteriuria is denied. However, it seems that the probable renal damage [8, 14, 15] is the determinant element to cause prematurity instead of bacteriuria itself. This element connected with amniotic infection and a high number of microthrombosis in placenta [16] seems to influence the fetal growth too [4].

Among the maternal factors, the consequences of an acute maternal pyelonephritis are well known but controllable enough by a rapid diagnosis and therapy. This is why at the beginning of pregnancy

we must do an early diagnosis and opportune therapy of asymptomatic bacteriuria and then repeat it everytime we shall diagnose a relapse or a reinfection at the following controls.

MATERIAL AND METHODS

The AA. studied 58 patients with asymptomatic bacteriuria in the I trimester of pregnancy and then they were followed during all pregnancy and puerperium.

The urine with $\geqslant 10^5$ colonies/ml were considered positive.

The urinary sample was taken from the middle stream of the first urine in the morning after a diligent washing of vulva. Finally, the count of colonies gave the same results twice.

In fact a colture with a growth of more than 10^5 colonies/ml has about 80% probability of a urinary infection. Two following samples with the sale groups of colonies increase this probability to 95% ([17, 18, 19, 20, 21]).

RESULTS

All patients had normal blood pressure. They hadn't dysmethabolic diseases; they were from 22 to 37 years of age with a parity from 0 to 3 pregnancies. Among the 27 patients who had delivered once or more 18 patients had an anamnestic report of symptoms probably correlated with urinary infections (minctional burnings, pollachyuria) during the preceding pregnancies; only 6 had a urinary treatment but not adequate.

No acute pyelonephritis occurred whether in pregnancy or in puerperium, and none was reported before pregnancy.

A vaginal bacteriologic tampon was made to all patients and 21 of them showed vulvaginitis due to Candida Albicans and bacterial mixed flora.

Table 1 showed the bacteria grown in colture and Coli is the most frequent responsible in urinary infections as literature confirms ([2, 22, 23, 24]). It is followed by Klebsiella, Proteus and Pseudomonas.

Patients received a treatment suggested by antibiogram taking care that they were at the first trimester of pregnancy; therapy last 10 days and nitrofurantoin, nalidixic acid, oxolinic acid, co-trimossazolo + thrimetoprim, were employed.

Table 1. — *Patients with bacteriuria in pregnancy (58 pts).*

Uricult	1st trim. %	2nd trim. %	3rd trim. %	Puerperium %
Coli	62	37.9	43.1	31.1
Klebsiella	20.7	12	15.5	12.1
Pseudomonas	6.9	8.6	3.4	8.6
Proteus	10.4	13.7	12	8.6
Total	100	72.4	74.1	60.4
Negative	—	27.6	25.9	39.6
Total	100	100	100	100

After 10 days from interruption of therapy a uricult of control was repeated: 11 results were positive for Coli and 2 results were positive for Proteus.

Among all controls executed in the II trimester, 42 coltures were positive (72.4%) and mostly for Coli. Patients received 10 days of urinary therapy and 11 controls after therapy were significant for infection (18.9%). Further 10 days of therapy were executed and so coltures were free from infections.

Among all uricults executed in the III trimester, 43 were significant for infection (71.1%) and patients received a 15 days treatment.

When urinary control was executed, 14 patients (24.1%) were affected by urinary infections too due to E. Coli in 9 cases, to Klebsiella in 2 cases, and to Proteus in 3 cases. These patients received a fur-

Table 2. — *Patients with sterile urine during first trimester of pregnancy (20 pts).*

Uricult	2nd trim. n° pts	3rd trim. n° pts	Puerperium n° pts
Coli	2	2	2
Pseudomonas	1	1	1
Negative	17	17	17
Total	20	20	20

ther 15 days treatment and so coltures were free from infections.

In the II and III trimester all patients did same tests of renal functionality (blood nitrogen, creatinine, creatinine clearance, proteinuria) and repeated controls of blood pressure. In all patients these were normal. No case symptomatic cystitis and acute pyelonephritis were reported in the studied group.

The biochemical and echographic monitoring of pregnancies was regular. Neither premature deliveries nor small for date babies occurred. 9 patients have had a caesarian section because of contracting ring dystocia and fetal hypoxia, 49 patients had a normal term delivery.

All patients were tested by a uricult of control between the 2nd and 3rd month after delivery: 35 patients had a positive uricult (60.4%) but asymptomatic. All patients had a normal puerperium.

After 10 days of urinary treatment, 12 patients (20.7%) had urinary infection too (9 due to Coli, and 3 to Pseudomonas).

The administration of urinary drugs 10 days in a month for a period of 6 months carried out a remission of all urinary infections.

In the control group of 20 patients with sterile urine during the first trimester (table 2) only 3 patients (15%) were affected by asymptomatic bacteriuria during the II trimester of pregnancy; after urinary treatment the coltures of the patients were free from infection but in all 3 cases relapses occurred in the III trimester and in puerperium.

CONCLUSIONS

Literature reports acute pyelonephritis in pregnancy as the cause of maternal and fetal diseases, even if an opportune treatment can improve the unfavourable prognosis the question about the responsibility of significant asymptomatic bacteriuria in causing symptomatic urinary infections is different. While many AA. are at variance with these urinary infections about fetal and maternal morbility, they agree on the large responsibility of these asymptomatic bacteriuria in causing more serious infections.

Results showed how difficult the treatment of asymptomatic bacteriuria is and how frequent the relapses are despite the repeated cycles of therapy. After treatment relapses also occurred in the two or three months following the delivery in percentage similar to that of pregnant women.

In the control group (with sterile urine at the beginning of pregnancy) the patients affected by urinary infection in the II and III trimester of pregnancy obtained the same results of the I group after treatment.

The absence of acute urinary infections in all the patients considered shows the importance of a screening in urinary asymptomatic infections and their continuous and repeated treatment.

Periodic uricults are fundamental also in pregnant women with sterile urine at the beginning of pregnancy. It's unpossible to evaluate the responsibility of pregnancy in determinating urinary infections because we haven't a comparison with uricults preceding the pregnancy.

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