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Detection of ACCPA Levels in Patients with *Proteus Mirabilis* Urinary Tract Infection: Possible Implications for Rheumatoid Arthritis

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Abstract

Objective: To investigate the anti-citrullinated protein antibodies (ACCPA) value for patients who have urinary tract infection (UTI) with *Proteus mirabilis (P. mirabilis)* and rheumatoid arthritis (RA) patients.

Methods: Eighty serum samples were analyzed for antibodies to cyclic citrullinated peptides (Anti-CCP) by ELISA. The samples included 40 from patients with RA diagnosed according to the 2010 American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) criteria, 20 from patients with UTI caused by *P. mirabilis*, and 20 from healthy controls.

Results: ACCPA was significantly higher in RA patients ($P < 0.001$) than in the UTI and control groups; however, ACCPA also showed a significant elevation in the mentioned bacterial UTI group compared to the control group ($P < 0.001$).

Conclusion: *P. mirabilis* infection is associated with raised ACCPA levels, and early screening and treatment could help to mitigate progression to RA.

Keywords: Rheumatoid arthritis, urinary tract infection, *Proteus mirabilis*, anti-citrullinated protein antibodies, molecular mimicry

Introduction

Rheumatoid arthritis (RA) is an autoimmune disorder characterized by deformed and malfunctioning joints arising from prolonged inflammation of the articular synovium and the presence of highly specific autoantibodies, most notably anti-citrullinated protein/peptide antibodies (ACPA) and rheumatoid factor (RF), that cause cartilage and subchondral bone degeneration over time, autoantibodies frequently appear many years before to the onset of symptoms.[1,2] RA is a global disorder that affects females more commonly than males, with a worldwide prevalence of 0.5 to 1%.[3] Its precise origin is not entirely recognized yet, various factors, including infections, genetic factors, and mutations, are believed to contribute to disease emergence.[4]

Urinary tract infections (UTIs) are the most common bacterial infections worldwide.[5,6] They represent a major public health problem, with millions of cases reported each year. The burden of disease is rising due to the rise of multidrug-resistant organisms. Women of all ages are most likely to get UTIs. Approximately 70 % of women will experience a UTI at some point in their lives.[5,7] Numerous studies indicate a correlation between RA and UTIs, particularly *Proteus mirabilis* (*P. mirabilis*) infections.[8–10] *P. mirabilis* can trigger an autoimmune response by mimicking a human antigen in a genetically susceptible individual at risk of developing RA.[11]

P. mirabilis is a gram-negative that pathogen belongs to the Enterobacteriaceae family. It is found in nature and as part of the normal gut microbiota of both humans and animals.[12] It is the third most frequently isolated bacterium in UTIs.[6] It is medically significant because it secretes numerous extracellular enzymes, notably urease and hemolysin, which facilitate tissue damage and the progression of infection.[13]

Previous research showed that patients with RA had a higher incidence of UTI than patients with other arthritis diseases and healthy individuals, and the most frequently isolated bacteria were *P. mirabilis*.[14,15] Furthermore, studies have found that RA patients have higher serum antibody levels against *Proteus* than healthy individuals.[9,10] The current study aims to investigate whether the infection with *P. mirabilis* UTI may has an influence in ACCPA level production through cross-reactivity and compare it with patients have an established RA disease and healthy individuals.

Material and Methods

Sample Collection and Isolation

This is a case-control study conducted in Babil province, Iraq, with a total of 80 participants: 40 RA patients, 20 UTI patients, and 20 healthy controls. The sample size was determined primarily based on feasibility and participant availability. Urine culture revealed that 20 of the isolates showed characteristic growth of *P. mirabilis* on blood and MacConkey agar obtained from total 150 urine samples; all bacterial isolates showed B-hemolysis on blood agar and were urease-positive on the urease test. *P. mirabilis* bacteria were confirmed by using the Vitek 2 compact system. Serum samples were collected from patients with a positive culture of *P. mirabilis* for this study. Forty serum samples were obtained from RA patients attending Rheumatology and Medical Rehabilitation Unit in Merjan Teaching Hospital who were diagnosed according to the 2010 American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) criteria, all of patients were currently receiving disease-modifying antirheumatic drugs (DMARDs) for several years. Disease activity was monitored using erythrocyte sedimentation rate (ESR) and RF titers. Twenty serum samples were taken from healthy controls who had no history of RA and other arthritis diseases, as well as no history of UTI in recent months.

Measurements of ACCPA

Three milliliters of venous blood were taken from the participant in a serum tube, and the tubes were centrifuged for

2 minutes at 3000 rpm. Serum-containing tubes were kept in a deep freeze at −20°C until sample processing. Serum quantified using ELISA (Cat: ELK9202, ELK Biotechnology) with detection range: 1.57–100 ng/mL. The results were analyzed according to the manufacturer's reference manual, which defines the clinical threshold for seropositivity as >10 ng/mL.

Ethical Approval

The study protocol was approved by the Institutional Review Board of the College of Medicine at the University of Babylon. Official permission was granted by Merjan Teaching Hospital. All participants were asked for their willingness to participate in the study and provided informed verbal consent prior to enrollment.

Exclusion Criteria

UTI patients with positive bacterial cultures with microorganisms other than *P. mirabilis*, patients with other autoimmune diseases or arthritis conditions, and patients under 18 years old.

Statistical Analysis

Data were analyzed using IBM SPSS version 26, continuous variables were expressed as mean ± standard error (SE). Comparisons among the three groups were performed using one-way analysis of variance (ANOVA), followed by post-hoc multiple comparison tests when appropriate. Comparisons between two groups were conducted using the independent samples *t*-test. Categorical variables were analyzed using the chi-square test. A significance level of $P < 0.05$ was considered statistically significant.

Results

The measurement of ACCPA values shows a significant difference among the three groups ($P < 0.001$). RA patients had the highest mean ACCPA value (26.273 ± 1.967 ng/mL; 95% CI: 22.419–30.127). The UTI group demonstrated a significantly elevated ACCPA level (16.187 ± 1.075 ng/mL; 95% CI: 14.080–18.294) compared to healthy controls (2.795 ± 0.101 ng/mL; 95% CI: 2.597–2.993), as shown in Fig. 1 and Table 1. Furthermore, pairwise comparisons demonstrated very large Cohen's d values (RA vs control: 2.30; UTI vs control: 3.92; RA vs UTI: 0.95), reinforcing that the differences are not only statistically significant but also clinically meaningful.

Table 2 shows there is no statistically significant differences in ACCPA mean across RA age groups ($P = 0.949$). Although RF and ESR were higher in the age group 21–40, the *P* values show no statistically significant differences, 0.75 and 0.18, respectively.

Serum obtained from patients with *P. mirabilis* associated UTI showed no significant variation across age groups ($P = 0.887$). However, a positive family history of RA was reported by 28.5% and 20% in the 41–60 and 61–80 age groups, respectively as illustrated in Table 3.

In control patients results demonstrated in Table 4, it shows significant variation by age group ($P = 0.012$). Family history of RA (33.3%) observed in 21–40 age group.

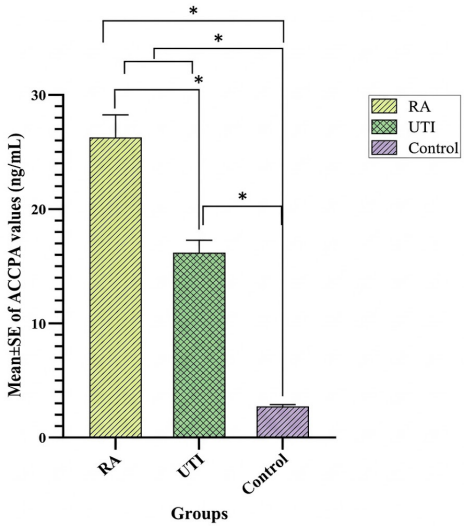

Fig. 1 **Comparison of ACCPA values among RA, UTI, and control group.**

Table 1. **Comparison of ACCPA values among RA, UTI, and control group. Data are expressed as Mean ± SE with 95% Confidence Intervals (CI)**

Groups	Number	ACCPA Mean ± SE (ng/mL)	95% CI (ng/mL)	Cohen's d (vs Control)	*P* value
RA	40	26.273 ± 1.967	22.419–30.127	2.30	
UTI	20	16.187 ± 1.075	14.080–18.294	3.92	<0.001*
Control	20	2.795 ± 0.101	2.597–2.993	–	

P value significant at $P < 0.05$.

Table 2. **Comparison of ACCPA mean values among RA group according to age groups**

Age range (Years)	Number	RF (IU/mL)	ESR (mm/hr)	ACCPA (ng/mL)
21–40	11	173 ± 41.97	48.34 ± 4.73	25.280 ± 4.01
41–60	26	151 ± 35.71	42,65 ± 2.87	26.679 ± 2.456
61–80	3	134.67 ± 45.43	39.13 ± 4.62	26.817 ± 6.865
***P* value**		**0.75**	**0.18**	**0.949**

Table 3. **Comparison of ACCPA mean values among UTI group with age groups**

Age range (Years)	Number	Family history of RA	ACCPA (ng/mL)
21–40	8	0%	16.718 ± 1.939
41–60	7	28.5%	15.457 ± 1.808
61–80	5	20%	16.360 ± 2.060
	***P* value**		**0.887**

Table 4. **Comparison of ACCPA mean values among control group**

Age range (Years)	Number	Family history of RA	ACCPA (ng/mL)
21–40	12	33.3%	2.650 ± 0.085
41–60	6	0%	2.811 ± 0.174
61–80	2	0%	3.610 ± 0.101
	***P* value**		**0.012***

Further, Fig. 2 displays the comparison of ACCPA values among the RA, UTI, and control groups according to age.

The comparison of results according to sex groups is shown in Table 5, ACCPA levels are higher in RA of both sexes in comparison to UTI and control groups there is a significant difference in $P < 0.001$. In addition, ACCPA comparison between female and male within each groups showed no statistically significant difference.

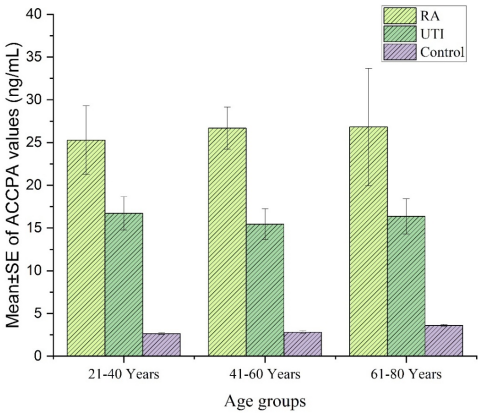

Fig. 2 **Comparison of ACCPA values among RA, UTI, and control groups according to age.**

Table 5. **Baseline characteristics of ACCPA values among RA, UTI, and control groups according to sex. Represent a significant difference at $P < 0.05$**

Sex groups	Number	RA (ng/mL)	UTI (ng/mL)	Control (ng/mL)	*P* value
Female	52	25.207 ± 2.247	17.594 ± 2.096	2.745 ± 0.129	<0.001*
Male	28	28.487 ± 3.920	15.036 ± 0.907	2.910 ± 0.157	<0.001*
***P* value**		**0.476**	**0.287**	**0.433**	

Discussion

RA is marked by the presence of autoantibodies RF and ACPA according to the 2010 ACR/EULAR criteria for diagnosis. ACPA is present in 55–91% of individuals with RA and RF in 50–60%. However, RF is not limited to RA and may be seen in multiple rheumatic and infectious conditions, as well as in up to 10% of healthy persons. ACPA exhibits 90% disease specificity, making it the most significant biomarker for the condition, and it is rarely reported in other rheumatic disorders. Nevertheless, some reports indicate that it can be increased in some infectious illnesses.[16–19] In addition, DAS28-ESR is commonly used by clinicians to measure disease activity in RA patients.[3]

Research data found that 37.03% of patients with RA developed UTIs in the months preceding symptom onset. In addition, women have a higher incidence of UTIs than men, which may explain why the incidence of RA is 3–4 times higher in women.[7,19,20] Furthermore, RA patients who previously experienced urinary infections faced a higher risk of increased disease activity.[20] Many studies have reported a high isolation rate of *P. mirabilis* in RA patients with UTI.[14,21]

Ebringer and Rashid proposed that UTIs caused by *P. mirabilis* may contribute to RA development. The pathogenic pathway comprises six phases initiated by cross-reactive autoantibodies generated by *Proteus* infection. The amino acid sequences of *P. mirabilis*, notably ESRRAL and IRRET, which are present in hemolysin (HpmB) and urease (UreC), respectively, contain arginine doublets and can be altered by peptidyl arginine deiminase (PAD); this pathway could potentially lead to early emergence of ACPA.[22,23]

A study by Newkirk et al. measured *anti-Proteus* IgG, IgM, and IgA in RF-seropositive and RF-seronegative individuals.[24] The study revealed a marked elevation in IgM and IgA levels in RF-seropositive compared with RF-seronegative individuals. In the United Kingdom, researchers measured RF IgG levels in the RA and control groups, and the data revealed that RF was positive in 27 of 35 RA patients and negative in the control group. In addition, bacteriological examination of the urine in the study uncovered that 13 samples (52%) had positive growth of *P. mirabilis*, which was two-fold higher than that of *Escherichia coli*.[25]

Motivated by the literature, the current study focuses on the measurement of ACCPA levels in patients with UTI caused by *P. mirabilis*, investigated whether the infection could produce ACCPA, and compared the results with those of RA and healthy groups.

Table 1 and Fig. 1 indicate a significant elevation in ACCPA levels in RA compared with other groups. The mean ACCPA level in RA was (26.273 ± 1.967 ng/mL) and it is consistent with the established role of ACCPA as a highly specific serological marker for RA. However, UTI complicated by *P. mirabilis* showed a significant increment of ACCPA compared to the control, with a mean of (16.187 ± 1.075 ng/mL), while the control group showed a mean of (2.795 ± 0.101 ng/mL).

Although the ACCPA is highly specific for RA, a previous report showed that ACCPA was detected in other bacterial infections. A study on 47 patients with active pulmonary tuberculosis found that 15 patients (32%) were positive for ACCPA.[18] Another study by Sangha et al. reported strong positive ACCPA results in patients who developed arthritis two weeks after an *Escherichia coli* infection.[26] UreC and HpmB are key *P. mirabilis* proteins that contribute significantly to pathogen virulence. UreC and HpmB exhibit cross-reactivity with human tissue. This means that antibodies directed against UreC and HpmB in *P. mirabilis* might cross-react with self-antigens in RA patients, thereby promoting the production of ACCPA.[11,21]

Table 2 shows that there is no statistically significant difference in the means of RF, ESR, and ACPA between age groups in RA patients. These findings may be explained by the fact that patients in this study had established disease and a history of using DMARDs for several years, resulting in their test titers not being significantly elevated.[27] The prevalence of RA is higher in the 41–60-year age group. This aligns with epidemiological data indicating a peak incidence of the disease in middle age due to repeated environmental exposures, age-related immune dysregulation, and hormonal alterations that facilitate autoimmune inflammation.[28]

In Table 3, the ACCPA mean in UTI across age groups indicates no statistical differences. Yet in groups 41–60 and 61–80, patients reported a family history of RA with 28.5% and 20%, respectively.

Genetic factors account for roughly 60% of the risk of developing RA. The primary genetic risk factor, the shared epitope (SE), is located at the HLA-DR1 and HLA-DR4 locus.[4] A study by Nielen et al. found that half of blood donor from RA family have positive ACCPA in their blood before the onset of clinical symptoms, this finding when coupled with environmental factors, such as interactions with triggering microbes, it can increase the likelihood of RA. *P. mirabilis* is a common microorganism linked to RA.[29]

The IRRET sequence in UreC mimics the LRREI sequence in type XI collagen, a component of hyaline cartilage, abundant in the small joints of the hands and feet. The cross-reactivity can occur when antibodies targeting *Proteus* urease bind to tissues containing type XI collagen, namely hyaline cartilage, and, in the presence of complement, can cause cytopathic damage that may leads to synovial inflammation.[25,30] Also, the SE has the EQRRAA sequence, which is similar to the ESRRAL sequence found in HpmB. This similarity allows cross-reactivity between the SE in the HLA-DR1 and HLA-DR4 molecules and HpmB. Antibodies may bind to cells containing such HLA antigens and, in the presence of complement, could cause cytopathic damage that leads to inflammation.[31,32]

Interleukin-8 (IL-8) that elevated in both UTI and RA, may have a role, a higher level of IL-8 was observed in response to *Proteus* infection compared with other bacteria implicated in UTIs. IL-8 is a powerful chemoattractant and neutrophil stimulator, leading to significant neutrophil recruitment to the infection site.[33,34] Additionally IL-8 produced by synovial fibroblast contributes significantly to neutrophil recruitment. Recurrent *Proteus* infections may lead to systemic surges of IL-8, which in turn activate neutrophils to produce neutrophil extracellular traps (NETs). Substantially augmented NETs were detected upon lipopolysaccharide (LPS) stimulation in peripheral blood and RA synovial fluid.[35]

Autoimmune disease development has been shown to be significantly influenced by NETs formation. Improper activation of NETs results in damage to tissues and the recruitment of important inflammatory mediators, such as IL-6 and IL-8, which facilitate the spread of inflammation, thereby exacerbating joint and synovial inflammation, leading to cartilage injury in RA.[36,37]

ESRRAL and IRRET in *P. mirabilis* both contain an arginine doublet that can be modified by PAD in granulocytes during infection, resulting in citrullinated protein. In the event of exposure of citrullinated protein to NETs which is an important trigger of autoimmunity, and in the presence of SE in genetically susceptible individuals, these may lead to cross-reactivity by antigens evoked by *P. mirabilis,* leading to loss of tolerance and production of autoantibodies, ACCPA can be detected even in the absence of RA manifestation.[23,32,34]

This study demonstrates a significant association between *P. mirabilis* UTI and elevated ACCPA levels but it cannot definitively predict future RA development or clinical onset due to a relatively small sample size, particularly among elderly subgroup patients aged 61–80 years. Additionally, the study was carried out at a single hospital within one geographic region, where patients had registered at the Rheumatology Unit and were receiving long-term DMARDs. These factors may restrict the generalization of the results, which helps to clarify the role of *P. mirabilis* infection in the early immunopathogenesis of RA.

Conclusion

This study showed that *P. mirabilis* UTIs are associated with an elevation in ACCPA level. While this rise does not necessarily lead to the development of RA, it reinforces the hypothesis that bacterial infection may play a role in autoimmune response, especially among genetically susceptible patients. Further studies with larger cohorts and genotyping are recommended to determine if the early production of autoantibodies associated with *P. mirabilis* infection can predict the clinical onset of RA.

Conflicts of Interest

The authors declare no conflicts of interest for this study.

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