

## Original Research Article

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# Evaluation of the pulse wave velocity in African rheumatoid arthritis subjects

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## ABSTRACT

**Background:** Rheumatoid arthritis is a systemic disease with often fatal vascular events. In addition to traditional cardiovascular risk factors, disease-specific elements contribute to this cardiovascular mortality. The aim of this study was to assess arterial stiffness in rheumatoid arthritis and to determine the factors involved.

**Methods:** We have recruited the black African patients followed in rheumatology and had rheumatoid arthritis diagnosis. Only patients between 18 and 60 years and meeting the American College of Rheumatology criteria were included. All controls were healthy. We evaluated the propagation velocity of the pulse wave finger-toe (PWVft) measured by the pOpmetre®.

**Results:** Present study shows that the PWVft was significantly elevated in over half of patients (55.10%). Besides, the mean patients PWVft was significantly higher than that of the control (respectively  $9.40 \pm 0.51$  and  $7.22 \pm 0.33$   $p=0.001$ ). In the patients, no factor was significantly involved in the arterial stiffness, but cons in the control group, the PWVft was significantly correlated with age ( $p=0.023$  and  $r=0.55$ ).

**Conclusions:** Rheumatoid arthritis patients had higher PWVft compared to controls. Due to the importance of its cardiovascular morbidity and mortality, arthritis requires a regular monitoring element as arterial stiffness, which is currently a major vascular parameter monitoring.

**Keywords:** African black, Arterial stiffness, Rheumatoid arthritis

## INTRODUCTION

Rheumatoid arthritis (RA) is a systemic disease with extra-articular manifestations that can be life threatening.<sup>1</sup> Among them, the vascular diseases are at the forefront.<sup>1</sup>

Studies have found a prominent place of cardiovascular events (CV) in the morbidity and mortality associated with RA; they would be responsible for about 50% of the excess mortality in RA.<sup>2</sup> It is considered a cardiovascular risk factor of the same weight as diabetes.<sup>3</sup> Many lesional

mechanisms have been described, including vasculopathy.<sup>4</sup> The involvement of vessels of all calibres has been demonstrated in this pathology.<sup>5</sup> It seems that in addition to conventional CV risk factors, specific elements of RA, as the existence of systemic inflammation, are participating in this cardiovascular mortality.<sup>6</sup> In fact the RA is the cause of an important vasculopathy which a loss of arterial compliance. So the purpose of this study was (1) to assess arterial stiffness (AS) in RA (2) to determine the factors associated with AS in a Senegalese hospital cohort of RA patients.

## METHODS

We have recruited patients who have been hospitalized or had to consult in the rheumatology department of the hospital Aristide LeDantec in Dakar (Senegal), who have

a diagnostic output rheumatoid arthritis. The records of all patients between 18 and 50 years were investigated and only patients meeting ACR criteria (American College of Rheumatology) have been included.<sup>7</sup> The existence of CV risk factors was investigated: treated hypertension, obesity, hypercholesterolemia treated, stopped smoking or not, types I or type II diabetes. However, subjects with RA but associated with another autoimmune disease or another affection or condition such as pregnancy, ischemia or gangrene of the extremities that can interfere with present results were excluded (Table 1). The patients were invited to come at 8:00 am and fasting at least twelve hours for the biological assessment. Pulse wave velocity was measured as a surrogate marker of arterial stiffness using a finger-toe pulse wave velocity (PWVft) device (pOpmetre®, Axelife SAS, France) as recommended by Hallab et al.<sup>8</sup>

**Table 1: The characteristics of the disease and patients.**

| Background and cardiovascular risk factors patients N=25 | N (%)      |
|--|------------|
| Family autoimmunity                                      | 7 (28%)    |
| Hypertension   | 2 (8%)     |
| Dyslipidemia   | 3 (12%)    |
| Rheumatoid arthritis characteristics (Patients N=25)     | N (%)      |
| Positive rheumatoid factor (UI/mL)                       | 17 (68%)   |
| Positif ACCP (UI/mL)                                     | 21 (84%)   |
| Means ± SD   |            |
| Duration of illness (month)                              | 54.72±9.89 |
| Duration of corticosteroid therapy (month)               | 21.16±5.99 |
| Dose corticosteroid (mg/day)                             | 9.20±0.37  |
| Duration of immunosuppressive (month)                    | 23.22±8.20 |
| Dose immunosuppressive (mg/ week)                        | 14.44±0.38 |

ACCP= antiproteines citrullinates

## Statistical analysis

The analyses were performed using Epi Info 7 and SPSS 16. Sociodemographic variables are expressed as a percentage.

Anova test is used to compare the means of quantitative variables between groups. Linear regressions were used to identify correlations between the PWVft and other variables. The significance levels were set at  $p<0.05$  and  $p<0.001$ .

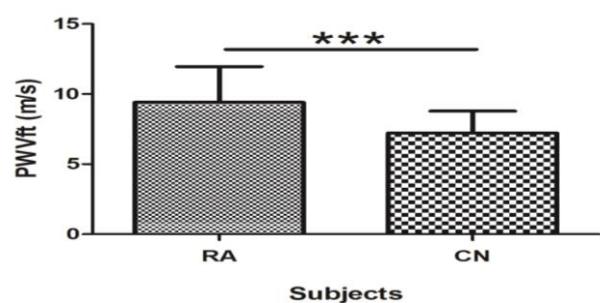
## RESULTS

### Sociodemographic, clinical and biological data

After an analysis of sociodemographic, clinical and biological variables, the two populations (patients and controls) are significantly different for SBP, DBP, total cholesterol, HDL-cholesterol and triglycerides (Table 2).

### Pulse wave velocity

The RA subjects had a significantly higher PWVft than controls, respectively  $9.40\pm0.51$  m/s and  $7.22\pm0.33$  m/s;  $p=0.001$  (Figure 1).



PWVft = pulse wave velocity finger-toe; RA = Rheumatoid arthritis; CN = controls; \*\*\*:  $p\leq0.001$ .

**Figure 1: Comparison of pulse wave velocity between groups.**

### Analytical results

After univariate analysis, any correlation was found between arterial stiffness and other variables in RA population. However, trends in the correlation were

found between PWVft and serum creatinine ( $p=0.06$  and  $r=0.38$ ) and between PWVft and immunosuppressive dose ( $p=0.09$  and  $r=0.34$ ). In controls, arterial stiffness was correlated to age ( $p=0.023$  and  $r=0.55$ ).

**Table 2: Socio-demographic, clinical and biological data.**

| Variables                | Patients (Moyen±ET) | Témoins (Moyen ± ET) | p-value |
|--------------------------|---------------------|----------------------|---------|
| Age (years)              | 35.52±1.54          | 35.27±2.16           | NS      |
| BMI (Kg/m <sup>2</sup> ) | 22.17±0.87          | 25.16±1.29           | NS      |
| HR (bpm)                 | 78.75±2.91          | 76.86±3.07           | NS      |
| SBP (mmHg)               | 116.80±3.99         | 133.14±2.64          | 0.001*  |
| DBP (mmHg)               | 81.20±2.19          | 72.86±1.39           | 0.002*  |
| MBP (mmHg)               | 93.04±2.65          | 92. 95±1.61          | NS      |
| Blood sugar (g/L)        | 0.87±0.02           | 0.89±0. 02           | NS      |
| Creatinine (mg/L)        | 7.32±0.34           | 7.91±0.54            | NS      |
| Total Cholesterol (g/L)  | 2.07±0.08           | 1.68±0.07            | 0.001*  |
| HDL-Cholesterol (g/L)    | 0.65±0.03           | 0.46±0.02            | <0.0001 |
| LDL-Cholesterol (g/L)    | 1.14±0.06           | 1.10±0.07            | NS      |
| Triglycerides (g/L)      | 0.78±0.06           | 0.53±0.06            | 0.003*  |

\* Significant difference, BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure, MBP: mean blood pressure, HR: heart rate.

### DISCUSSION

This cross-sectional and prospective study was conducted in a cohort of RA patients and control subjects to evaluate the AS to derive a screening tool for cardiovascular damage. Present study showed that the PWVft was significantly higher among more than half (55.10%) of our patients. In addition, the patients PWVft mean was significantly higher than the control (respectively  $9.40\pm0.51$  and  $7.22\pm0.33$ ,  $p=0.001$ ). High PWVft shows substantial rigidity of the arterial wall. Thus we find that the RA is a cause of arterial stiffness. The same was reported by the study of Van Doormun performed in 18 patients with RA with decreased arterial compliance of large arteries and small arms compared to control.<sup>9</sup> Klocke also found him a decreased compliance of large arteries in 14 RA.<sup>10</sup> The AS is the consequence of the structural and functional caused by chronic systemic inflammation due by the RA.<sup>11</sup>

Arterial compliance is defined as the function of damping by the arterial trunks of the arterial pulsatility generated by the intermittent cardiac ejection. The loss of the arterial compliance, that is to say the increase of the arterial rigidity, testifies to an attack of the target organ which is the artery. The arterial compliance mainly depends on the large arteries and depends essentially on the structure of their wall. The compliance of the smaller arteries is influenced by the muscle tone of the artery.<sup>12</sup> Declining compliance of both large and smaller arteries are predictive factors for cardiovascular events.<sup>12</sup> In fact, RA patients have an increased risk of cardiovascular

(CV) disease, including myocardial infarction, heart failure, and an increased risk of CV death.<sup>13</sup> Chronic inflammation is held responsible for this increase in CV risk.<sup>14</sup>

Besides in RA, macroscopic vessels are the place to accelerated atherosclerosis promoted by chronic inflammation, which is already considered an independent CV risk factor in patients with RA.<sup>15,16</sup> According to other authors, the inflammation control would reduce cardiovascular mortality in RA.<sup>17</sup> In patients, any factor was significantly involved in this AS. However in the control group, the PWVft is strongly and significantly correlated with age ( $p=0.023$  and  $r=0.55$ ). In fact, it is established that the physiological arterial stiffness increases with age.<sup>18</sup>

### CONCLUSION

In this study the pOpmetre allowed us to confirm the loss of compliance described in a chronic systemic inflammation such as rheumatoid arthritis. In present hospital environments, it would be a simple and practical tool for large-scale monitoring of chronic inflammatory diseases comprising a structural change of the blood vessels. A study on a larger cohort would better establish our results.

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