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Research Article

Evaluation of anthropometric parameters, white blood cell count, and morphological changes of red blood cells in a pristane-induced rheumatoid arthritis rat model

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Abstract

Introduction: Rheumatoid arthritis (RA) is an autoimmune disease characterized by a complex pathophysiological mechanism. The adipose tissue, blood and its cellular components are less-studied extra-articular manifestations of RA.

Aim: In this study, we used a pristane-induced RA rat model to assess the impact of RA-induced inflammation on anthropometric parameters, obesity indices, white blood cell differentiation, and morphological changes in red blood cells.

Materials and methods: The randomized controlled, prospective, experimental study involved 20 adult female Wistar rats, aged 12-13 weeks, with body weights ranging from 180 to 220 grams. The rats were randomly assigned to two groups: an experimental RA-induced group (PIA group; n=10) and a control group of healthy animals (n=10). Rheumatoid arthritis was induced by intradermal injection of 150 μL of pristane at the dorsal base of the tail.

Results: Rats in the PIA group exhibited significantly lower values of body weight (p=0.002), abdominal circumference (p=0.011), and BMI (p=0.028) compared to those in the control group. The number of neutrophils (p<0.001) and eosinophils (p<0.001) in the PIA group was significantly higher than in the control group, while the number of lymphocytes (p=0.001) was lower. Peripheral blood smear analysis showed a significant increase in the number of dacryocytes (p=0.003), anulocytes (p=0.002), spherocytes (p<0.001), and reticulocytes (p<0.001) in the PIA group compared to the control group.

Conclusion: This study demonstrates that the pathological processes in rheumatoid arthritis are reflected in the anthropometric parameters, the distribution of specific leukocyte types, and the morphological characteristics of erythrocytes. These factors collectively contribute to the complexity of disease progression in RA.

Keywords

anthropometric parameters, leukocyte cells, red blood cells, rheumatoid arthritis, rat model

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PENSOFT.

Introduction

Rheumatoid arthritis (RA) is a chronic, inflammatory autoimmune disease of complex pathogenesis that leads to joint deformities and loss of their function.[1] There is still no cure for RA. Detailed insight into the pathogenesis would facilitate the discovery of new specific treatments.[2] Although the RA pathogenesis has not yet been elucidated, it has been shown that the complex interplay between multiple inflammatory mediators is pivotal for this process.[2,3] The inflammatory process has been linked to oxidative stress, but the mechanisms by which its increase affects the inflammatory process in RA remain to be determined.[3]

In addition to its articular manifestations, RA influences other organ systems, such as the cardiovascular or pulmonary systems.[1,4] Recently, adipose tissue dysfunction has been associated with the autoimmune process in RA[5,6], mainly through reactive oxygen species (ROS) and adipokine production.[7] RA-induced inflammation causes lipid and glucose metabolism disturbances.[6] There are contradictory data regarding the relationship between RA and changes in body composition. A number of studies have shown that patients with RA display considerable alterations in body composition, loss of lean mass, and increase in adipose tissue[8], while others, on the other hand, have not shown a significant difference in the observed parameters.[9]

Blood tissue is a less well-studied extraarticular target of RA[10], and its cellular components have been linked to disease development.[11] Despite the lack of mitochondria in red blood cells, they produce a large amount of reactive oxygen species due to a high arterial oxygen tension and heme content in their cytoplasm.[12] Morphological changes of red blood cells associated with increased oxidative stress have been reported in various conditions/diseases, such as diabetes mellitus, hypertension, smoking, obesity, etc.[13,14] ROS can affect the red blood cell membrane, in which they cause lipid peroxidation, alter membrane plasticity, and reduce their ability to deform themselves when passing through narrow blood vessels. These altered red blood cells display a disturbed function.[15] Changes in red blood cell appearance have been found in patients with RA.[10,16]

Animal models are invaluable tools in RA research. One of the main challenges in the experimental RA research is the development of animal models that approximate the human situation.[17] All animal models were developed under certain conditions reflecting some, but not all, features of the human disease process. Therefore, in order to appropriately use available RA animal models to address specific research questions, we must know the characteristics of a particular model.[17,18] The T-cell-driven pristane-induced arthritis (PIA) model has a high reproducibility and an incidence of RA induction of almost 100%.[19] Its further characterization would yield valuable insights and optimize its use in the context of research in the various aspects of the human RA syndrome.

Materials and methods

The study design and animals

The research was designed as a prospective, observational, and experimental study using the pristane-induced arthritis (PIA) model in Wistar rats.

The study involved 20 adult female Wistar rats, aged 12-13 weeks, with body masses ranging from 180 to 220 grams. The animals were bred and maintained under standard conditions until the initiation of the experiment. They were housed in a temperature (23°C±3°C) and humidity (50%±10%) controlled room and were kept in an environment free of pathogenic microorganisms. A natural light-dark cycle (12 hours of light and 12 hours of darkness) was maintained.

The animals were housed in standard cages and had access to water and commercial rodent food (supplied by Agromix-Madi) ad libitum. Animal monitoring and supervision were conducted daily, 2-3 times per day, at approximately the same times, by a team of competent and qualified personnel. The laboratory animals were randomly assigned to two groups: an experimental RA-induced group (PIA group; n=10) and a control group of healthy animals (n=10).

The procedure of arthritis induction

During the administration of pristane, the experimental animals were positioned in a supine position, with their limbs immobilized. The dorsal area of the base of the tail was meticulously cleansed with 70% ethanol, and 150 μL of pristane (Acros Organics) was injected intradermally using a fine needle (27G).[20] The control group of animals received 150 μL of physiological saline solution (0.95% NaCl) following the same procedure and at the same site. Subsequent to the completion of the procedure, each animal was returned to its cage for continued observation.

Anthropometric measurements and calculation of obesity indices

Body weight, body length, neck circumference (NC), thoracic circumference (TC), and abdominal circumference (AC) were measured in all animals on the 30th day of the experiment. Using the obtained values, the body mass index (BMI) and Lee index were calculated. Abdominal circumference was measured by placing a measuring tape just in front of the forelimbs, while chest circumference was measured just behind the forelimbs.

Body length (nose-to-anus length) was defined as the distance from the nose to the anus.

The body mass index (BMI) was calculated using the formula:

$$\text{BMI (g/cm}^2\text{)} = \text{body mass/body length}^2.$$[21]

The Lee index was calculated by dividing the cube root of body weight (in grams) by the naso-anal length (in centimeters).[21]

Body weight was measured using an electronic scale (ACCULAB VI-I 200) with decimal precision, while other measurements were made using a flexible centimeter tape. All measurements were performed in triplicate, and the average of the three values was recorded as the final measurement.

Blood sample collection and microscopic examination of peripheral blood cells

On the thirtieth day of the experiment, peripheral blood samples were collected via caudal vein puncture into 3 mL tubes containing ethylenediaminetetraacetic acid (EDTA) and gel. The injection site was disinfected before sampling using a standard disinfectant (0.2% chlorhexidine spray). A drop of blood from each rat of the control and experimental groups was transferred to the glass slides, and blood smears were made with a second slide held at a 45-degree angle. After air-drying, the smears were stained using the Giemsa method, following standard laboratory procedures.[22] Stained blood smears were evaluated by two independent investigators based on standard morphological criteria.

Counting was conducted on representative monolayer fields with non-overlapping blood cells. A total of 1000 erythrocytes from each smear were analyzed using a Motic Type 102M light microscope at 1000× magnification to assess the presence of poikilocytic red blood cells. The number and type of poikilocytes were recorded as percentages of each poikilocyte type concerning the total red blood cell count.[23]

The white blood cell differential was performed for each animal by classifying 1000 leukocytes as neutrophils (NEU), lymphocytes (LYM), monocytes (MON), basophils (BAS), and eosinophils (EOS). The results are presented as relative percentages of each type of white blood cell in relationship to the total white blood cell count.[24]

Statistical analysis

The data distribution was assessed using the Shapiro-Wilk test. The Student's *t*-test was applied to compare differences between groups for continuous variables with a normal distribution, with results presented as the mean and standard deviation. The Mann-Whitney U test was used to compare differences between continuous numerical variables with a non-normal distribution, with results presented as the median and interquartile range. A p-value <0.05 was considered statistically significant. Statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS, version 19.0; Chicago, IL, USA).

Results

The anthropometric parameters (body weight, body length, neck circumference, abdominal circumference, thoracic circumference) and obesity indices (body mass index, AC/TC index, Lee index) are shown in **Table 1**. The body weight of the rats in the PIA group was significantly lower (p=0.002) compared to that of the control group. Rats in the PIA group exhibited significantly lower values of abdominal circumference (p=0.011) and BMI (p=0.028) than those in the control group. Body length, neck circumference, thoracic circumference, AC/TC index, and Lee index did not show statistically significant differences between observed groups.

Table 2 demonstrates statistically significant differences between the PIA group and the control group in the number of neutrophils (p<0.001), lymphocytes (p<0.001), and eosinophils (p=0.001). No statistically significant differences were noted in the number of monocytes (p=0.117) and basophils between observed groups.

Analysis of peripheral blood smears revealed that the morphology of red blood cells in the control group of animals was normal **(Fig. 1A)**. In the PIA group of rats, an increased number of spherocytes (red arrows), reticulocytes (black arrows), dacryocytes (white arrows), and anulocytes (purple arrows) was observed **(Fig. 1B, C)**

Table 1. Anthropometric parameters and obesity indices in PIA group and control group of rats

	Control group (n=10)	PIA group (n=10)	*p*
Body weight, g	200.2±9.8	183.8±10.1	0.002
Body length, cm	18.0 (18.0–18.25)	19.0 (17.75–19.0)	0.353
Neck circumference, cm	11.5 (11.0–12.0)	11.0 (10.0–11.125)	0.089
Abdominal circumference, cm	13.5 (13.0–14.0)	13.0 (12.5–13.0)	0.011
Thoracic circumference, cm	12.5 (12.0–13.0)	12.0 (12.0–12.125)	0.063
Body mass index, g/cm^2	0.61±0.04	0.54±0.08	0.028
AC/TC index	1.08 (1.077–1.092)	1.08 (1.04–1.08)	0.684
Lee index, g/cm	0.323 (0.316–0.327)	0.301 (0.292–0.325)	0.075

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Table 2. Leukocyte cells in PIA group and control group of rats in 1000 cells

Types of white blood cells (%)	Control group (n=10)	PIA group (n=10)	*p*
Neutrophils	16.7±8.4	40.5±7.2	<0.001
Lymphocytes	81.1±8.4	49.9±6.6	<0.001
Monocytes	0.8±1.55	2.9±3.73	0.117
Basophils	0.0	0.0	-
Eosinophils	2.4±2.3	6.7±2.7	0.001

In the PIA group of rats, there was a significant increase in the percentage of spherocytes ($p<0.001$), reticulocytes ($p<0.001$), dacryocytes ($p=0.003$), and anulocytes ($p=0.002$) compared to the control group. There were no significant differences in the percentage of ovalocytes ($p=0.573$), echinocytes ($p=0.668$), stomatocytes ($p=0.345$), schizocytes ($p=0.372$), acanthocytes ($p=0.422$), and target cells ($p=0.286$) between the observed groups **(Table 3)**.

Table 3. Forms of red blood cells in PIA group and control group of rats in 1000 cells

Types of poikilocytosis (%)	Control group (n=10)	PIA group (n=10)	*p*
Ovalocytes	5.2±3.4	4.3±3.6	0.573
Spherocytes	5.3±3.95	39.6±16.8	<0.001
Reticulocytes	1.0±1.89	42.7±12.4	<0.001
Dacryocytes	28.2±10.6	52.8±19.5	0.003
Anulocytes	4.6±6.6	45.0±34.3	0.002
Echinocytes	20.3±14.9	16.0±27.4	0.668
Stomatocytes	5.1±5.2	7.7±6.7	0.345
Schizocytes	2.7±1.8	3.4±1.6	0.372
Acanthocytes	0.2±0.63	1.2±3.8	0.422
Target cells	0.20±0.63	1.0±2.21	0.286

Discussion

Inflammatory manifestations of rheumatoid disease are often associated with changes in anthropometric and hematological parameters in humans. In our experimental model of adjuvant-induced arthritis, we noted marked falls in body weight and BMI of arthritic rats versus healthy controls. This finding is in line with earlier reports that systemic inflammation could be accountable for lesser weight accrual or even weight loss, chiefly through mechanisms like reduced food intake, diminished mobility, and muscle catabolism. The decrease in BMI in the PIA group may indicate early manifestations of rheumatoid cachexia linked to chronic inflammation.[25] On the other hand, higher BMI values have also been found to be associated with lower levels of joint damage in the early stages of RA.[26] According to Novelli et al.[27], BMI is a simple and easy-to-use index for assessing obesity in rats. These authors stated that BMI can reliably estimate body fat in rats, although it is not sensitive enough to detect changes in body composition caused by diets with different macronutrient compositions.[27]

In our study, BMI values in both groups of animals throughout the entire experimental period were below 0.68 g/cm^2, which is the obesity threshold for adult male Wistar rats.[27] The results of the present experimental study show that BMI and all other anthropometric parameters and obesity indices had lower values in the PIA group than in the non-arthritic group. However, only the differences in body weight, abdominal circumference, and BMI were statistically significant.

The observed decrease in body weight in the RA group may be due to increased production of pro-inflammatory cytokines or reduced food intake resulting from decreased animal mobility. Similar to our results, Lopez-Menduina et al. observed reduced body weight gain in adjuvant-induced arthritic rats, suggesting that inflammation inhibits body weight gain and leads to a lower relative fat mass in diseased animals compared to healthy rats.[28]

Even though our results showed significant reductions in body weight, abdominal circumference, and BMI in the PIA group, the Lee index and AC/TC ratio did not significantly differ between arthritic and control animals. This finding aligns with what Novelli et al.[27] observed; though

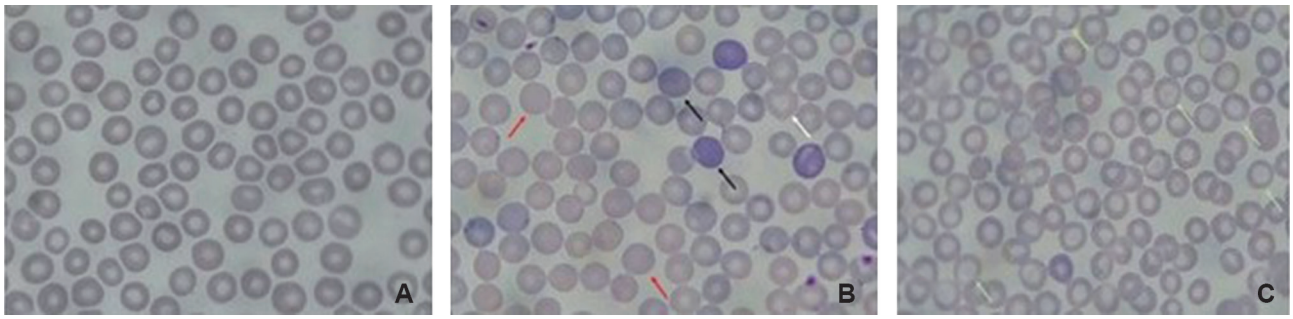

Figure 1. Representative photomicrographs of red blood cells (×100). **A**. Control group; **B** and **C**. PIA group. Spherocyte (red arrow); reticulocyte (black arrow); dacryocyte (white arrow); anulocyte (purple arrow).

BMI increased proportionally with fat accumulation in Wistar rats, the other two parameters—the Lee index and AC/TC ratio—did not change much under different conditions of diet-induced obesity except when there was extreme adiposity. These indices seem to have lower sensitivity regarding moderate changes that occur in fat distribution or changes in lean body mass, as would be typified by inflammatory conditions such as adjuvant-induced arthritis.

This finding may be explained by the fact that skeletal muscle mass is primarily lost in inflammatory cachexia in RA patients, whereas fat is retained or redistributed, particularly in non-abdominal areas, reducing changes in indices that rely on proportional measurements or body geometry.[29]

In addition, inflammatory edema may distort abdominal or chest measurements in a non-uniform way and therefore further decrease the sensitivity of the AC/TC index. Whereas BMI readily reflected the overall decrease in body mass in this study, the apparently non-significant changes of AC/TC and Lee index may more truly reflect their limitations to detect body composition changes related to inflammation rather than an actual absence of effect.

This finding supports the Novelli et al.[27] interpretation that, compared to AC/TC and Lee indices, BMI provides a more responsive and reliable marker of adiposity and metabolic disruption in rodent models, particularly in non-dietary experimental settings with systemic inflammation.

According to Franch et al.[30], there are two periods in the arthritic process of adjuvant-induced arthritis: the first, from days 14 to 28 after induction, which is characterized by systemic inflammation, and the second, from the 42nd to the 70th day, which corresponds to the chronic phase, during which there is a tendency for the arthritic parameters to return to reference values. According to these authors, the period of emergence and establishment of systemic inflammation is specifically associated with weight loss in experimental animals.

In contrast, in a study examining the protective effect of consuming different nut oils on morphological characteristics and markers of inflammation in rats with adjuvant-induced arthritis (AIA), a significant increase in the rats' final body weight and arthritis was observed. The authors concluded that inflammation and swelling explained the increased body weight of the rats.[31] Likewise, Shamlan et al.[32] investigated the anti-arthritic and anti-inflammatory effects of Moringa peregrine seed oil and leaves in Freund's complete adjuvant-induced arthritis in rats and also found an increase in body weight in a group of arthritic animals compared to healthy control animals.

A comparison of the WBC differential parameters, neutrophils, lymphocytes, monocytes, basophils, and eosinophils showed a statistically significant increase in the number of neutrophils and eosinophils and a statistically significant decrease in the number of lymphocytes in the PIA group compared to healthy rats. The increase in the number of monocytes in the PIA group was not statistically significant. We did not observe basophils in the blood smear of rats from either the control or the PIA group. In line with our results, Franch et al. reported a significantly higher percentage of leukocytes and a decrease in the lymphocyte count in adjuvant-induced arthritis (AA). These authors stated that the arthritic process did not significantly affect the percentage of eosinophils and monocytes, while basophils were virtually absent. In contrast, from the seventh day after the induction of arthritis, there was a significant increase in the percentage of neutrophils (approximately 300% compared to the control from day 14 to day 28) and a decrease in the percentage of lymphocytes (approximately 30% compared to the control from day 14 to day 28). The authors concluded that the differential changes in the leukocyte profile are probably a secondary consequence of the inflammatory process present in AA.[30] A limitation of our hematological analysis is that the total white blood cell count was not directly measured; we rather relied on differential counts from blood smears. This limits the interpretation of absolute inflammatory cell burden and should be acknowledged as a methodological constraint. Although research has confirmed the presence of various hematological abnormalities in RA, little is known about the red blood cell morphologic alterations caused by the pathophysiologic process in RA. Poikilocytosis refers to variations in the shape of erythrocytes and can be caused by different internal or external agents, stress, or genetic factors. A recent study confirmed the presence of poikilocytosis and anisocytosis in RA patients. The most common forms observed in a peripheral blood smear of patients in this RA cohort were knizocytes, stomatocytes, acanthocytes, microcytes, and irregularly contracted cells.[10] Peripheral blood smear analysis in our study showed the presence of ovalocytes, dacryocytes, anulocytes, echinocytes, stomatocytes, schizocytes, spherocytes, acanthocytes, reticulocytes, and target cells. Analysis of morphologically altered erythrocytes showed significantly higher percentages of dacryocytes, anulocytes, spherocytes, and reticulocytes in the PIA group compared to the control group. There were no significant differences in the percentage of other forms of poikilocytic erythrocytes between the observed groups. According to Staroń et al.[16], the reduced protective role of erythrocyte membrane thiol groups and peripheral antioxidant systems may cause dysfunction in RA erythrocytes and shorten the lifespan. Prior studies have shown that systemic inflammation in RA causes oxidative damage to erythrocytes, affects their shape, and significantly reduces their ability to move through smaller-diameter blood vessels, such as capillaries. Factors that disrupt the biophysical and physiological properties of red blood cells (RBCs) alter the structure of hemoglobin and its capacity to bind oxygen (O_2), which may contribute to the complexity of the disease status in RA.[10]

Conclusion

Pristane-induced arthritis results in a decrease of body weight, abdominal circumference, and BMI. It also caus-

es significant changes in the red blood cells, leading to an increase in the number of dacryocytes, anulocytes, spherocytes, and reticulocytes. In addition, it affects the white blood cell differential by decreasing the number of lymphocytes and increasing the number of neutrophils and eosinophils.

While our findings indicate that RA may influence both body composition markers and hematological profiles, we see these results as an early step rather than a final conclusion. Since this study was carried out in a pristane-induced rat model, further experimental and translational work is needed to better understand how—or if—these observations might apply in a clinical setting.

Conflicts of interest

The authors have declared that no competing interests exist.

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Ethical statement

This study was approved by the Ethics Committee of the Medical Faculty of the University of Sarajevo with document registration No. 07-03-523-2/23, Bosnia and Herzegovina.

Author contribution

Conceptualization: Samir Mehmedagić, Muhamed Katica, and Amela Dervišević; methodology: Amela Dervišević and Samir Mehmedagić; data collection: Dina Kapić, Aida Bešić, and Nadža Kapo-Dolan; data analysis: Dina Kapić, Muhamed Katica, and Nermina Klapuh-Bukvić; writing - review and editing: Almir Fajkić and Asija Začiragić; supervision: Amela Dervišević.

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