

Does Chronic Anemia Prevent Cardiac Remodeling and Dysfunction?

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Chronic anemia, paradoxically, was associated with both increased mortality and the protection against deleterious effect of clinical situations, such as acute kidney injury. Based on these data we hypothesized that anemia provides protection to the cardiac muscle. Accordingly, we wanted to find how the physiological changes, caused by anemia, attribute to the cardiac structure and function.

Methods:

Rats were fed with either low-iron diet or normal for 18 weeks. Weight, hemoglobin (Hb) level and blood pressure measurements were monitored weekly. Echocardiography measurements were taken every other week. Cardiac function was assessed using the Millar-Tip catheter[®].

Results:

Hb drop developed as soon as 3 weeks of diet (13 ± 2 g/dl vs. 16 ± 1 g/dl, $P < 0.05$). Few weeks later, mean capsular volume and mean capsular hemoglobin concentration (36 ± 4 fl vs. 57 ± 1 fl and 32.5 ± 1 g/dl vs. 30 ± 2 g/dl, $P < 0.05$, respectively) were lowered as well, indicating the development of hypochromic microcytic anemia. During the experiment the rats were affected by both their normal growth and the anemia. Accordingly, the anemic rats had lower body mass through the majority of the experiment duration. The heart rate of the anemic rats was higher than that of the controls, thus increasing their double product (404 ± 8 bpm vs. 360 ± 8 bpm and $60,000 \pm 1307$ mmHgXbpm vs. $54,000 \pm 1550$ mmHgXbpm, $P < 0.05$, respectively). Echocardiographic structural parameters showed consistent increase in diastolic LV area and circumference in the anemic group while in the control groups these parameters stopped increasing toward the end of the experiment (48 ± 4 mm² vs. 34 ± 4 mm² and 25 ± 1 mm vs. 22 ± 1 mm, $P < 0.05$, respectively). In both groups fractional shortening was preserved (38 ± 6 % vs. 43 ± 7 %, $P > 0.05$). Final P-V loops measurements had similar results of cardiac function measurements for both groups.

Conclusions:

Anemia in healthy young rats affects their development, induces delay in their general and cardiac development but doesn't affect their cardiac function.