Pulmonary Arterial Hypertension Eisenmenger Syndrome Diagnosis Of Patients Serum Levels Kallistatin

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Introduction:
Kallistatin, serine proteinase inhibitor, is first discovered and identified as tissue kallikrein binding protein and a unique serine proteinase inhibitor, and has emerged as a novel inhibitor of angiogenesis and inflammation.
Pulmonary arterial hypertension/Eisenmenger Syndrome, from left to right shunt congenital heart disease is an important issue for children. Eisenmenger syndrome is progressive obliterator vasculopathy; the pathogenesis endothelial dysfunction and function of ion channels, calcium homeostasis, changes in platelet and endothelial function, intravascular thrombosis proliferation reactivity increased vascular inflammation and remodeling.
In this study, pulmonary arterial hypertension Eisenmenger syndrome diagnosis of patient's serum levels kallistatin was evaluated; by this way. Pulmonary arterial hypertension is considered a possible relationship between serum levels of kallistatin.

Methods
We enrolled the patients with pulmonary arterial hypertension diagnosis that confirmed by the angiocardiography in our center and their serum kallistatin levels were studied.
Study population: Pulmonary arterial hypertension and Eisenmenger Syndrome caused by congenital heart diseases having left to right shunt (Group I), Pulmonary arterial hypertensive patients who did not develop Eisenmenger Syndrome (Group II), primary pulmonary hypertension (Group III), Congenital heart defects having left to right shunts who did not have pulmonary arterial hypertension (Group IV), patients with Innocent murmur (Group V).
Pulmonary arterial hypertension is defined as mean pulmonary arterial pressure greater than 25mmHg.

Results:
The study included total 78 patients (44 girls, 34 boys). 16 with Eisenmenger syndrome, 15 with mild to moderate, 5 patients with severe pulmonary arterial hypertension (without Eisenmenger Syndrome).
Seven patients had primary pulmonary arterial hypertension (without left-to-right shunt), 19 patients had left to right shunt without pulmonary hypertension (13 ASD, 6 VSD). Control group consisted 16 patients with innocent murmur.

Serum levels of kallistatin were significantly lower (p<0.05) in patients with Eisenmenger Syndrome. Negative correlation was detected between mean pulmonary arterial pressure and serum kallistatin levels.

Conclusion:
Kallistatin as a novel inhibitor of angiogenesis may be an important non invasive marker in the follow-up of pulmonary hypertensive patients.