Low Blood Pressure and Amyloidosis

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Dear Sir,

Amyloidosis is a complex disease entity caused by deposition of amyloid substance in various organs or systems with resultant derangement in bodily functions. The symptomatology of the disease depends on the organ involved. Kidneys are known to be primarily affected and proteinuria due to glomerular permeability increase is usually the leading symptom in the patient with systemic amyloidosis. As the disease progresses renal functions further deteriorate and death is usually caused by renal complications [1-4]. Cardiovascular involvement is another frequent feature and cause of death in amyloidosis patients [1,5,6].

Hypotension is reported to be a common feature of the disease though the exact mechanism is not established. Autonomic neuropathy, suppression of the renin-angiotensin-aldosterone system due to juxtaglomerular apparatus involvement, adrenal insufficiency, low-cardiac output and secondary reasons like dehydration and malnutrition are some factors that are held responsible [1, 7-9]. For elucidating the incidence and etiology of low blood pressure in systemic amyloidosis, we conducted a study on 30 systemic amyloidosis patients diagnosed by renal biopsies. Two of the patients had primary, 16 patients had reactive and 12 patients had FMF-related hereditary familial amyloidosis. The mean age of the patients was 35.6 ± 15.0 years. Sixteen of the patients were newly diagnosed while 14 had had the disease for an average duration of 42 ± 33 months. The presenting symptoms were edema in 26, uremic complications in 2, gastrointestinal bleeding in 1 and restrictive cardiac failure in 1 of the patients.

After a complete physical examination, blood pressure and heart rate was taken. Laboratory tests like a hemogram, biochemical tests and urinalysis were performed. Electrocardiographic, echocardiographic, ultrasonographic examinations were done. Blood pressure while standing was measured and autonomic tests like Valsalva maneuver, sub-maximal handgrip test were performed for evaluating the autonomic system involvement. The results of cardiac and autonomic tests were compared to those of a control group of 15 subjects.

Average mean arterial blood pressure was 80.6 ± 13.7 mm Hg in the patients while it was 89.2 ± 12.7 mm Hg in the control group, the difference being statistically significant ($p < 0.02$).

Postural hypotension was detected in 6 (4 pathologic and 2 borderline values according to a criterion of ≥ 30 mm Hg fall in systolic pressure) patients and 2 subjects in the control group. Valsalva maneuver was abnormal in 8 (2 borderline) patients and 3 subjects in the control group. Hand grip test was pathologic in 17 patients (6 borderline) and 5 control subjects. In the neurological evaluation 21 patients had polyneuropathy (9 sensory, 12 mixed). These results were not parallel with the duration, type or extent of the disease nor could they be related to the incidence of low blood pressure. The electrocardiographic and echocardiographic measurements revealed normal systolic functions while interventricular septal thickness and left ventricular posterior wall thicknesses were found to be increased in 10 patients. Diastolic functions as reflected by the ratio of early to late diastolic peak being less than 1, were found to be abnormal in 6 patients. Left ventricular mass was greater than normal in 2 patients. Eight patients had pericardial effusion which

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seemed to be related to the severe proteinuria these patients had.

Among the significant laboratory values were hypoalbuminemia (2.6 ± 1.0 g/dl) with severe proteinuria (4.7 ± 3.6 g/day) and high PRA values in patients with low blood pressure.

Among the striking findings was the incidence of low blood pressure being increased in the patients with mild to moderate renal disease of shorter duration. Blood pressure increased parallel to the degree of renal disease. The laboratory tests, cardiac evaluations and autonomic tests did not yield any specific parameter correlating with low blood pressure though the incidence of severe nephrotic syndrome and hypoalbuminemia was increased in the patients with low blood pressure. The decrease in intravascular volume due to hypoalbuminemia might have an impact on low blood pressure in these patients.

Catecholamines, adrenal function, anti-diuretic hormone values which could have important effects on blood pressure could not be measured because of technical reasons.

In conclusion, we think that the issue of low blood pressure in amyloidosis patients is still obscure and needs to be further elucidated with quite sophisticated biochemical and clinical analysis.

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