Letter to the Editor

Massive pulmonary thrombo-embolism in a case of multiple myeloma and concurrent anti-phospholipid syndrome

Sir,

Patients with multiple myeloma (MM) are susceptible to venous thrombo-embolism (VTE) because of enhanced blood viscosity, procoagulant property of monoclonal protein, inflammatory cytokines,[1] immobilization, acquired activated protein C resistancy,[2] increased level of factor VIII and Von Willebrand Factor, decreased level of protein S,[3] and also thalidomide-induced thrombogenesity.[4] Secondary anti-phospholipid syndrome (APS) was reported in patients with malignancies.[5] We present development of massive pulmonary thrombo-embolism (PTE) in a case of MM and APS after starting thalidomide.

A 56 y/o female admitted with bone pain. She had history of two spontaneous abortions around 12th week of gestation about 10 years ago. Assessment revealed a normocytic anemia, an elevated ESR, and monoclonal gammopathy. Serum IgM, IgA, and IgG all were diminished. [Table 1] IgD and IgE tests are not available in our local laboratories. Urine bence jones was negative.

Table 1: Laboratory tests of patient

Test	Value	Reference range	Unit
Calcium	8.47	8.5-10.5	mg/dl
Phosphor	2.6	2.8-4.5	mg/dl
Creatinine	1	0.5-1.3	mg/dl
LDH	550	Up to 480	U/L
ESR	145	mm/hour	
CRP	+3	-ve	
Albumin	3	3.5-5.2	gr/dl
Protein	8.6	6.6-8.8	gr/dl
IgG	4.25	6.56-13.51	gr/dl
IgA	0.364	0.86-3.2	gr/dl
IgM	0.311	0.349-2.55	gr/dl
PT	15	11-13	Second
PTT	32	28-38	Second
Anti B2-Glycoprotein1	17	<12	U/ml
Anti phospholipid (IgM)	6	<12	U/ml
Lupus Anticoagulant	85	31-44	Second

IgG=Immunoglobulin G; IgA=Immunoglobulin A; IgM=Immunoglobulin M; PT=Prothrombin time; PTT=Partial thromboplastin time; LDH=Lactate dehydrogenase; ESR=Erythrocyte sedimentation rate; CRP=C-reactive protein

Skeletal survey was normal. Serum calcium and creatinine were normal. After bone marrow aspiration and biopsy, which showed more than 40% plasma cells, the diagnosis of MM was made and treatment included: Thalidomide 200 mg daily and dexametasone (40 mg days 1-4, 9-12, 17-20) was initiated. We administered aspirin 80 mg daily for deep vein thrombosis prophylaxis. After 1st cycle of treatment with thalidomide and dexamethasone, she came back with sudden onset dyspnea, pleuretic chest pain, tachycardia, and fever. Physical examination showed: Tem = 38.5°C, BP = 90/60 mmHg, tachypnea, and decreased breathing sounds in lower part of right lung. Chest X-ray showed right-sided minimal pleural effusion and also a wedge-shaped pleural-based opacity. Echocardiography revealed right ventricle dilatation with increased pulmonary artery pressure. Chest CT angiography showed blood clots in proximal part of ascending and descending branches of left pulmonary artery and right descending pulmonary artery. Diagnosis of PTE was made. Since her hemodynamic status was stable, we did not administer thrombolytic therapy. Enoxaparin 1 mg/kg twice-a-day was initiated and would continue for period of 6-12 months for VTE treatment. Because of positive history of two abortions, we thought for APS and performed further investigations.

Lupus anti-coagulant was high. Titer of anti-B2-glycoprotein-1 antibody was in borderline range. Anti-phospholipid Antibody (IgM) was normal [Table 1].

Since she had history of two abortions, in the presence of massive PTE and high lupus anti-coagulant, the diagnosis of APS was made accompanied with MM.

Although patients with multiple myeloma who are taking thalidomide are predisposed to thrombo-embolic events, we should think and investigate further to other causes of hypercoagulable states such as APS in myeloma patients, especially in those with major thrombotic events.

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