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Case Report

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Spontaneous Non-Traumatic Massive Chest Wall Hematoma in A Case of Chronic Myeloid Leukemia at Diagnosis

Dr Vishnu Sharma¹, Dr Vansh Bagrodia^{2*}, Dr Jahanvi Grover³

¹Associate Professor, Department of Clinical Hematology, SMS Medical College, Jaipur. Email: vanshbagrodia@gmail.com; Address - 510, Midas Touch Apartments, Malhotra Nagar, Jaipur, Rajasthan, India

²Intern, SMS Medical College, Jaipur

³Intern, PGIMS, Rohtak

*Corresponding author: Dr Vansh Bagrodia, Intern, SMS Medical College, Jaipur

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A female in her 50s presented with complaints of weakness and mild fever for the past 15 days, accompanied by an unintentional weight loss of 10% of her initial body weight. Physical examination revealed significant splenomegaly, extending 15 cm below the costal margin. Chronic Myeloid Leukaemia

(CML) was suspected, prompting routine blood investigations and a bone marrow biopsy, the results of which are presented in table 1. Given that these investigations supported our initial suspicion of CML, a PCR for BCR-ABL was ordered to confirm the diagnosis.

S.no	Test/ Investigation	Results	Normal values
1.	Hemoglobin (in g/dL)	6.3	12-16
2.	 WBC count (in 10³/mm³) Neutrophils Lymphocytes Monocytes Eosinophils Basophils 	400.2 89.2% 3.2% 1.4% 3.2% 3.0%	4-11 40-80% 20-40% 2-10% 1-6% 1-2%
3.	Platelet count (in 10 ³ /mm ³)	136	150-400
4.	Peripheral blood smearRBCWBCPlatelet	Normocytic normochromic to macrocytic with presence of polychromatophilic cells. Notable of leucocytosis with prominent left shift, basophilia and eosinophilia. Adequate, no hemoparasite	
5.	Bone marrow biopsy	Biopsy comprises of hypercellular bone marrow with increased myeloid precursor with metamyelocytes and myelocytes and dwarf megakaryocytes.	

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The patient returned 20 days later with BCR-ABL assay results indicating a high BCR-ABL1/ABL1 ratio (77.098% IS) with the major transcript (P210). She also reported new complaints of swelling over the right side of the chest in the subscapular region with sharp, localized pain that worsened with movement. The patient denied recent trauma or injury, and there was no history suggestive of an inherited bleeding disorder in the patient and her family. Physical examination revealed a palpable, tender

mass on the right posterior chest wall, accompanied by ecchymosis. A hematoma was suspected, leading to an HRCT scan, which (Figure 1) revealed an ill-defined heterogeneous density [13.8*5.2*19.9 cm] lesion with multiple hyperdense foci along the right lateral chest wall muscle. Few air foci were observed within the lesion, and no underlying rib fractures or vascular abnormalities were identified.



Figure 1: CT Scan showing hematoma in right lateral chest wall muscle.

The patient was initiated on imatinib therapy for CML and received analysesics for the hematoma. She achieved complete hematological resolution in one month, and the chest wall hematoma resolved spontaneously.

The initial manifestation of spontaneous nontraumatic bleeding in CML has been rarely documented, with fewer than 40 cases reported in the available literature, most commonly involving intracranial bleeds. Regarding spontaneous chest wall hematoma, only four earlier reports were found in the available literature [2,3,5,6], with the remaining cases primarily involving intraabdominal and musculoskeletal bleeds [1].

The exact mechanisms responsible for these bleeding episodes in CML are not thoroughly understood, likely due to their infrequent occurrence. Several factors have been proposed as potential causes, including coagulation abnormalities, platelet dysfunction, excessive granulocyte infiltration, uremia, and low red blood cell count [1]. Kartthik et al. documented a case in which a CML patient experienced bleeding caused by acquired Glanzmann's thrombasthenia [4].

Platelet dysfunction in chronic myeloproliferative disorders is a complex phenomenon. These patients may exhibit various platelet abnormalities, including irregular platelet morphology, defects in storage pool function, abnormalities in the platelet membrane, and disruptions in arachidonic acid metabolism. The most commonly observed abnormality is a reduced platelet response to epinephrine, while responses to collagen and ADP are less frequent. This reduction is believed to stem from a decrease in α -adrenergic receptors on the platelet membrane. Notably, patients with CML tend to experience hemorrhagic events less frequently compared to individuals with other myeloproliferative disorders [3]. We believe the cause of

bleeding in our patient to be due to an acquired platelet function disorder.

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