Granulocytic Sarcoma as Initial Presentation of Acute Myeloid Leukaemia

Sir,

Acute myeloid leukaemia is a neoplastic proliferation of immature cells of haemopoietic system characterized by infiltration by these cells in blood, bone marrow and other tissues and is diagnosed by finding of blasts more than 30%, either in bone marrow or peripheral blood film (PBF). Myeloblastoma is a rare tumour of early myeloid precursor cells. Dock1 was the first to note the association of it with acute leukaemia, and this tumour is regarded as an ominous harbinger of acute myelocytic leukaemia (AML), or the onset of blast crisis in chronic myelogenous leukaemia. Here, we are reporting a case who presented with subacute paraplegia due to a mass lesion (granulocytic sarcoma) without typical AML symptoms like fever, bleeding, weakness, fatigue.

A 23 year old male patient presented to orthopaedic surgeon with complaints of backache and numbness of bilateral legs. Pain was in the midback region associated with band like feeling in lower chest. It was dull aching and persistent. After 3 days patient developed numbness of right leg which progressed over 2-3 days to involve left leg. In next 2-3 days, it progressed to involve both lower limbs and lower part of abdomen. Patient also had the difficulty in standing steadily and felt the weakness in bilateral lower limbs. On clinical examination, there was mild anaemia and upper motor weakness in bilateral lower limb (power III/V) with sensory loss upto mid thoracic region. Peripheral blood film revealed occasional immature cell with normal total and differential count. MRI of spine revealed a moderately large posterior epidural mass extending from D4 to D6 level with resultant cord compression and cord edema at D4 to D6 level with narrow signal alteration involving D1 and D5 to D7 vertebrae. The patient was operated and the mass was excised and sent for histopathological examination. The material was fleshy grayish white tissue. Microscopically, it showed diffuse proliferation of anaplastic round cells appearing small to large in size. These cells had scanty cytoplasm and nuclei containing dense stained chromatin. Prominent nucleoli were seen in large cells. There was no differentiation seen and patient was diagnosed as poorly differentiated malignant tumour. Bone marrow biopsy from iliac crest showed deposits of malignant round cells suggestive of AML. Bone marrow aspiration showed hypercellular marrow, M:E ratio increased, normo to macroblastic erythropoiesis, myeloid cell showed maturation arrest. There were numerous immature myeloid cells (around 40% blasts), occasional cell showed Auer rods. megakaryocytes, plasma and reticular cells were normal. Findings were consistent with acute myeloid leukemia. T cell lymphoma marker (CD45-RO) was negative.

The patient was diagnosed as a case of acute myeloid leukaemia presenting with paraplegia due to spinal cord compression, due to cord myeloblastoma / granulocytic sarcoma. Patient was treated with standard chemotherapy and improved and he is still on the follow up of clinical haematology clinic of PGIMS, Rohtak.

Usually an AML patient presents due to consequences of anaemia, leucocytosis, leucopenia, leucocyte dysfunction, thrombocytopenia (i.e. fatigue, weakness, effort intolerance, fever or bleeding). Rarely patient may present with the symptoms of mass lesion located in soft tissues of breast, uterus, ovary, cranium, spinal dura, gut, lung, mediastinum, prostate, bone and other organs. This mass lesion, a collection of leukaemic cells is called granulocytic sarcoma or chloroma or myeloblastoma.1 The typical AML may occurs simultaneously, later, or not at all in these patients. In our patient there were no complaint of fever, bleeding, fatigue, weakness and the first manifestation was predominantly paraplegia due to mass lesion. However the bone marrow was full of blasts, more than 40%. Granulocytic sarcoma / chloroma / myeloblastoma are rare manifestations of AML and can occur in any part of human body. There has been similar report in the past by Muss Moloney et al which stated that 50% of such tumours were asymptomatic.2 Rooku et al reported a case presented with the compression paraplegia due to chloroma or granulocytic sarcoma,3 as was also seen in our patient.

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Spontaneous Tumor Lysis Syndrome in Acute Lymphoblastic Leukemia

Sir,

We describe a case of acute lymphoblastic leukaemia who presented with acute oliguric renal failure (ARF) following spontaneous tumor lysis (STLS), a rare
A 20-year-young gentleman was admitted with two week history of low grade fever, headache, gum bleeding and hematuria. Two days before admission to our hospital, he had been examined at local hospital and blood investigations done there revealed total leucocyte count 1,15,600/mm³ with 86% blasts in peripheral blood film. He denied history of taking any medicines for his illness. General physical examination was unremarkable except single mobile right axillary lymph node, and fundal hemorrhages. Rest of his systemic examination was unremarkable. Laboratory tests revealed hemoglobin 10.4 gm/dl, total leucocyte count (TLC) of 6400/cumm, differential count revealed 41% neutrophils, 55% lymphocytes, 1% monocytes, 1% eosinophils and 2% myelocytes and no blasts in the peripheral blood film. Platelet count was 22,000/cumm. Blood biochemistry revealed urea 192 mg/dl; creatinine 5.9 mg/dl; uric acid 34.7 mg/dl; phosphate 14mg/dl; potassium 7.8 mmol/l; and calcium of 7.5 mg/dl. Abdominal ultrasound revealed increased renal echogenecity with loss of cortico-medullary differentiation. Arterial blood gases revealed uncompensated metabolic acidosis. Fine needle aspiration cytology from the lymph node was consistent with infiltration by acute leukemia. Bone marrow examination revealed 84% blasts which were negative for myeloperoxidase but showed granular positivity for periodic acid schiff (PAS). The morphology of blasts was consistent with acute lymphoblastic leukemia, FAB L1. Immuno-phenotyping could not be done because of non-availability. The patient was managed with intravenous fluids, platelet concentrates, allopurinol and heparin-free hemodialysis on alternate days till renal functions recovered. Subsequently he was given chemotherapy with weekly vincristine for 6 weeks and daily prednisolone for 6 weeks because of his non-affordability for intensive chemotherapy. His bone marrow after 6 weeks was in remission. He was started on prophylactic intracranial radiation. However, within two weeks of achieving complete remission, he again had a relapse and died a little later of sepsis.

TLS occurs when necrosis of large amount of malignant cells leads to overwhelming of normal renal excretory functions, leading to acute renal failure. STLS is usually related to higher tumour load and malignancies that are rapidly proliferating. It is an oncological emergency which requires intensive and coordinated care. The laboratory incidence of tumour lysis syndrome is much higher than symptomatic clinical syndrome.

The incidence of TLS is higher following chemotherapy in tumors with high tumour burden such as ALL and Burkett’s lymphoma. In one of the largest study about TLS, out of 1791 children with ALL and non-Hodgkin Lymphoma, 78 children (4.4%) developed TLS and of these 26.4% patients were with ALL (B-cell) and 14.9% Burkett’s lymphoma. On the other hand, STLS has not been reported very frequently. Crittenden and Ackerman described the first case of STLS with ARF in 1977. Until 2002, STLS leading to oliguric acute renal failure has been reported in only four patients in English literature. In a retrospective analysis of 926 patients of acute renal failure in 2004, 10 patients (1.08%) had STLS. All 10 patients became oliguric and required dialysis. Seven patients (70%) developed diuresis following dialysis. However, only three patients survived, with two having residual renal impairment.

Our patient presented with deranged renal functions and within 24 hours went on to develop oliguric renal failure before chemotherapy or even diagnosis of basic disease could be made. He developed hyperuricaemia, hyperphosphataemia, hypocalcaemia and hyperkalaemia necessitating hemodialysis.

The common causes of tumour lysis syndrome are related to type, tumour burden, aggressiveness of malignancy and response to chemotherapy besides preexisting renal insufficiency and poor response to hydration. STLS is related to rapid turnover of the cells and can be precipitated solely by fever. In addition high viscosity and or perivascular infiltration by leukemic cells may give similar end organ damage. Our patient had a classic picture of spontaneous tumor lysis. At admission there was no history of decreased urine output and it was only on investigations that he was found to have renal failure, hyperuricaemia, hyperkalaemia, hypocalcaemia and hyperphosphataemia, the cardinal features of laboratory tumor lysis syndrome but within 24 hr developed oliguric renal failure. Though hyperphosphataemia is not the presenting feature of STLS, advanced renal failure and normal TLC at presentation with no blast cells may be responsible for hyperphosphataemia in the present case. The aggressive nature and poor prognosis of the leukemia in the present case is depicted by the fact that patient relapsed within two week of going into remission.

TLS should be suspected in all patients of hyperuricemic ARF. STLS is reversible when recognized early and treated appropriately as in the present patient.

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