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Case Report

Myelodysplastic Syndrome: Raeb-1 in A 58-Year-Old Male: A Case Report from Andhra Pradesh, India

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ABSTRACT

Myelodysplastic Syndromes (MDS) represent clonal hematopoietic disorders that are marked by ineffective hematopoiesis, peripheral cytopenias, and a fluctuating risk of progression to acute myeloid leukaemia. Research conducted in India has revealed an earlier age of onset and a greater prevalence of adverse cytogenetic profiles in comparison to Western populations; however, comprehensive case-based documentation is still lacking. We report a case of a 58-year-old male farmer from Guntur District, Andhra Pradesh, who experienced progressive dyspnea, fatigue, recurrent low-grade fever, gum bleeding, and spontaneous bruising. Examination revealed pallor, petechiae, and mild splenomegaly. Laboratory tests showed pancytopenia with macrocytic anaemia, hypogranular neutrophils, and giant platelets. Bone marrow aspiration indicated hypercellularity with multilineage dysplasia, ring sideroblasts, and 6% myeloblasts. Cytogenetic analysis revealed an isolated deletion of chromosome 5q. Based on these findings, a diagnosis of MDS - Refractory Anaemia with Excess Blasts-1 (RAEB-1) was made. The patient was treated with supportive transfusions, empirical antibiotics for febrile neutropenia, and subcutaneous azacitidine. This case exemplifies the diversity of MDS, consistent with Indian research that indicates an earlier onset and differing prognostic characteristics. It underscores the importance of documenting individual cases to connect epidemiological disparities between Western and Indian populations. This case underscores the clinical and cytogenetic characteristics of MDS in an Indian patient, drawing attention to the diagnostic difficulties and treatment considerations in settings with limited resources. Comprehensive regional case reports are crucial for enhancing the understanding of disease biology and outcomes within Indian populations.

Keywords: Myelodysplastic Syndromes, Chromosome 5q Deletion Syndrome, Bone Marrow Dysplasia, Hematologic Neoplasms, Macrocytic Anaemia, Supportive Care.

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INTRODUCTION

Myelodysplastic Syndromes (MDS) represent a diverse collection of clonal hematopoietic stem cell disorders, distinguished by ineffective hematopoiesis, dysplastic changes in one or more myeloid lineages, and cytopenias in the peripheral blood, accompanied by a fluctuating risk of progression to Acute Myeloid Leukaemia (AML) (Papaemmanuil *et al.*, 2013; Fenaux *et al.*, 2020). Clinically, individuals may exhibit symptoms associated with anemia, frequent infections, or a propensity for bleeding, whereas bone marrow examinations typically show hypercellularity accompanied by dysplastic alterations.

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Worldwide, the prevalence of MDS is approximated at 4-7 cases for every 100,000 individuals each year, with a rise in incidence correlated with advancing age. The median age at which diagnosis occurs in Western populations is approximately 70 years (Cazzola and Malcovati, 2005). Conversely, research conducted in India consistently indicates a younger median age at which patients present, generally in their early to mid-50s, with a notable percentage of these patients classified within higher-risk prognostic groups (Srivastava, *et al.*, 2024; Chaubey *et al.*, 2016). Cytogenetic abnormalities occur frequently, with monosomy 7/deletion 7q, deletion 5q, trisomy 8, and deletion 20q being the most prevalent (Narayanan, 2017; Kawankar *et al.*, 2010). Significantly, Indian cohort studies by Gupta *et al.*, (2017) and Dakshinamurthy AG *et al.*, (2005) exhibit a greater prevalence of poor-risk cytogenetic markers at younger ages than Western studies (Greenberg *et al.*, 2012; Ma, 2007), highlighting potential regional or biological disparities.

At the molecular level, alterations in genes that govern epigenetics (such as DNMT3A and TET2), splicing (for instance, SF3B1 and

SRSF2), and transcription are associated with the development of MDS; however, research involving Indian patients is still scarce. A recent study observed a high prevalence of mutations in DNMT3A, SF3B1, ASXL1, and TET2, frequently occurring together, indicating a complicated mutational environment within this demographic (Beevi, 2022). Notwithstanding these developments, the exact pathogenesis is still not fully comprehended, and elements like environmental exposures, delayed presentation, and limited access to sophisticated diagnostic tools may play a role in the variability seen among Indian patients.

Considering the diversity inherent in Myelodysplastic Syndromes (MDS), individual case reports offer significant clinical insights, particularly in less frequently reported areas. These reports assist in documenting the patterns of clinical presentation, cytogenetic characteristics, and treatment strategies observed in practical settings. In this regard, we present the case of a 58-year-old male from Guntur District, Andhra Pradesh, who has been diagnosed with Myelodysplastic Syndrome: Refractory Anemia with Excess Blasts-1 (RAEB-1). This report emphasises his clinical presentation, diagnostic assessment, and therapeutic management.

CASE DETAILS

A 58-year-old male farmer from Tenali in Guntur District visited the General Medicine outpatient department of a tertiary care teaching hospital, reporting a history of progressively worsening shortness of breath over the last two months, which had notably intensified in the past ten days. The patient indicated that he was initially able to ascend a flight of stairs without any discomfort; however, he has recently experienced breathlessness even when walking across his backyard or engaging in routine tasks such as carrying a small bucket of water. Additionally, he expressed concerns about significant fatigue, generalised weakness, and intermittent dizziness, which compelled him to cease his daily agricultural activities.

Furthermore, the patient disclosed experiencing recurrent instances of low-grade, intermittent fever (fluctuating between 99-100.2°F during his RMP visit), accompanied by chills but devoid of rigours, which have persisted for the past three weeks. He observed frequent gum bleeding during brushing and spontaneous bruising on his arms and thighs, despite having no recollection of any trauma. His family noted a gradual paleness of his skin and lips, and he reported an unintended weight loss of approximately 4 kilograms, along with a diminished appetite over the last two months. There was no reported history of cough with sputum production, hemoptysis, hematemesis, melena, or notable night sweats. He denied any history of tuberculosis, chronic alcohol use, smoking, or occupational exposure to radiation or chemicals. He had no previous hospitalisations or a history of receiving cytotoxic medications. The family history was unremarkable concerning hematological or malignant conditions.

Upon general examination, the patient presented with a chronically ill appearance, exhibiting pallor and a moderate physique, reflected in a BMI of 20.2 kg/m^2. Notable conjunctival pallor was observed, alongside petechial spots distributed across both forearms. There were no signs of icterus, cyanosis, clubbing, pedal edema, or palpable lymphadenopathy. The vital signs indicated a blood pressure of 110/70 mmHg, a pulse of 96 beats per minute (regular and low volume), a respiratory rate of 22 breaths per minute, a temperature of 99.4°F, and an SpO_2 level of 92% while on room air. The cardiovascular examination revealed a soft systolic ejection murmur at the apex, likely attributable to anemia. The respiratory assessment indicated diminished air entry at the bilateral bases, accompanied by fine inspiratory crepitations. The abdominal examination revealed mild splenomegaly, with the spleen palpable 2 cm below the left costal margin, firm and non-tender, and no evidence of hepatomegaly or ascites. The central nervous system examination was unremarkable.

Routine haematological assessments indicated a haemoglobin level of 7.8 g/dL, a hematocrit of 24%, a total leukocyte count of 2,300/µL, an absolute neutrophil count of 820/µL, and a platelet count of 65,000/µL. The red cell indices indicated macrocytosis (MCV 102 fL), with an MCHC of 33 g/dL. The reticulocyte count was notably suppressed at 0.5%. The Erythrocyte Sedimentation Rate (ESR) was measured at 62 mm/hr. The peripheral blood smear revealed macrocytic anaemia characterised by significant anisopoikilocytosis, hypogranular neutrophils exhibiting a pseudo-Pelger-Huët anomaly, and the presence of giant platelets. The coagulation profile indicated a mildly prolonged bleeding time, while both Prothrombin Time (PT) and activated Partial Thromboplastin Time (aPTT) remained within normal ranges. Serum levels of vitamin B12 and folate were normal, thereby excluding nutritional deficiencies. Additionally, liver and renal function tests yielded unremarkable results.

Bone marrow aspiration indicated a hypercellular marrow characterised by notable erythroid hyperplasia and dysplastic alterations in both the erythroid and myeloid lineages, which included the presence of binucleated erythroblasts, megaloblastoid transformations, and hypolobulated megakaryocytes. The detection of ring sideroblasts was observed through Prussian blue staining. Myeloblasts constituted roughly 6% of the nucleated marrow cells. A trephine biopsy corroborated the findings of hypercellular marrow exhibiting multilineage dysplasia. Cytogenetic evaluation via FISH revealed an isolated deletion on the long arm of chromosome 5 (del(5q)), which was associated with an intermediate-risk classification according to the Revised International Prognostic Scoring System (IPSS-R) (https://www.mdcalc.com/calc/3981/revised-international-prognostic-scoring-system-ipss-r-myelodysplastic-syndrome-mds).

Based on the clinical presentation, peripheral blood smear, bone marrow analysis, and cytogenetic findings, a diagnosis of Myelodysplastic Syndrome - Refractory Anaemia with Excess Blasts-1 (RAEB-1) was confirmed. The patient was admitted for supportive treatment. He received 2 units of packed red blood cells to alleviate symptomatic anaemia and 1 unit of platelet concentrate to address gum bleeding associated with thrombocytopenia. Empirical broad-spectrum antibiotics (piperacillin-tazobactam) were initiated due to febrile neutropenia, while awaiting culture results. Prophylactic antifungal treatment was contemplated because of ongoing neutropenia. The patient treatment was started with subcutaneous azacitidine (75 mg/m^2/day for 7 days within a 28-day cycle), and plans for erythropoiesis-stimulating agents were made for the long-term management of anaemia. The patient and his family received thorough counselling regarding the chronic nature of the condition, the potential risk of progression to acute myeloid leukaemia, and the significance of hematopoietic stem cell transplantation as the sole curative approach, albeit constrained by age and donor availability.

DISCUSSION

Our case presents a 58-year-old male who exhibited symptomatic pancytopenia, dysplastic features on peripheral smear, a hypercellular bone marrow with multilineage dysplasia, and cytogenetically confirmed del(5q), ultimately diagnosed as Myelodysplastic Syndrome (MDS), subtype RAEB-1. The clinical presentation of progressive fatigue, exertional dyspnea, mucocutaneous bleeding, recurrent febrile episodes, and splenomegaly corresponds with the classical characteristics of MDS, which indicate ineffective hematopoiesis and marrow failure (Lindsley and Ebert, 2011). The detection of 6% marrow blasts categorised this patient within the RAEB-1 group, an intermediate-to-high-risk classification according to the Revised International Prognostic Scoring System (Greenberg *et al.*, 2012; https://www.mdcalc.com/calc/3981/revised-international-prognostic-scoring-system-ipss-r-myelodysplastic-syndrome-mds), which is associated with variable survival rates and a significant risk of progression to acute myeloid leukaemia (Greenberg *et al.*, 2012). The observation of an isolated del(5q) is significant, as this cytogenetic abnormality is typically associated with a more favourable prognosis and responsiveness to lenalidomide (Lindsley and Ebert, 2011); however, in the context of excess blasts, it presents a less optimistic outlook, indicating clonal evolution beyond the classical 5q- 5q-syndrome.

From a pathophysiological standpoint, the deletion of chromosome 5q (del(5q)) is recognised to result in haploinsufficiency of critical genes, including RPS14, which plays a role in disrupted erythropoiesis and clonal proliferation. Additionally, other genes, such as CSNK1A1, are associated with irregular signalling pathways and cell viability (Ebert *et al.*, 2008). The characteristics of erythroid hyperplasia, ring sideroblasts, and megaloblastoid alterations observed in our patient's marrow underscore the intricate relationship between ineffective erythropoiesis, disrupted iron metabolism, and stromal factors. Furthermore, there is a growing acknowledgement that the pathogenesis of MDS is influenced by immune dysregulation and persistent inflammatory signalling, particularly the increased levels of TNF-α and IL-6, which could facilitate clonal dominance and hasten disease progression (Li *et al.*, 2025). The combination of these processes may elucidate the reason behind the presence of an isolated del(5q) abnormality in this patient, which was associated with an increased number of blasts, indicating a more aggressive clinical phenotype.

From a pharmacological perspective, the commencement of azacitidine treatment for this patient was justified, given that hypomethylating agents continue to be the standard treatment for RAEB-1 and higher-risk MDS. The pivotal AZA-001 trial provided evidence that azacitidine enhances survival rates, postpones the progression to AML, and lessens the need for transfusions in comparison to traditional care protocols (Fenaux *et al.*, 2020). Erythropoiesis-stimulating agents can provide symptomatic relief for patients exhibiting low serum erythropoietin levels; however, their long-term effects in RAEB-1 are still not well understood. Lenalidomide is known to be very effective in the classic 5q-syndrome characterised by isolated anaemia, but its efficacy becomes less predictable in patients with an increased number of blasts, as clonal heterogeneity may diminish the treatment's effectiveness (List *et al.*, 2006). In our case, the provision of transfusion support and the implementation of infection prophylaxis were crucial elements of management, highlighting the pharmacist's responsibility in enhancing supportive care and averting complications associated with treatment.

A plausible explanation for the progression of this patient's disease is that haploinsufficiency of genes associated with 5q led to ineffective erythropoiesis. Concurrently, additional unidentified somatic mutations, potentially involving TP53, TET2, or splicing factor genes, may have contributed to the advancement of the disease to RAEB-1. Furthermore, chronic immune activation alongside a supportive marrow microenvironment could have facilitated the expansion of blasts. This underscores the necessity of incorporating cytogenetic and molecular information into risk stratification, as the prognostic outcomes in MDS are influenced not solely by cytogenetics but also by the interplay of genetic, epigenetic, and inflammatory elements.

From a clinical perspective, this case illustrates several significant lessons. Careful monitoring of persistent cytopenias, the integration of cytogenetic information into the diagnostic process, and early referral to hematology are critical for effective management. Pharmacists play an essential role not only in supportive care but also in drug stewardship, ensuring the safe administration of hypomethylating agents and monitoring for hematological and infectious toxicities. Equally vital is the

counselling of patients and their families, communicating the chronic nature of MDS, the risk of progression to AML, and the limited but potentially curative role of stem cell transplantation, which in this patient is limited by age and donor availability. Open discussions facilitate informed decision-making for patients and families regarding treatment objectives, balancing the potential for survival with quality of life.

This report is limited by the absence of next-generation sequencing to delineate the full mutational landscape, which could refine prognostication and therapeutic choices. Longitudinal marrow assessments and follow-up data were also unavailable to document disease evolution or treatment response. Nonetheless, the detailed clinical, morphological, and cytogenetic description adds to the scarce Indian literature on RAEB-1 with del(5q), providing valuable insights into the spectrum of MDS presentation in resource-constrained settings.

CONCLUSION

This case exemplifies the clinical and cytogenetic intricacies associated with myelodysplastic syndrome, wherein an isolated del(5q) abnormality, typically correlated with a favourable prognosis, was found alongside an increase in blasts, thereby categorising the disease as RAEB-1. The report highlights the necessity of incorporating morphological, cytogenetic, and pharmacological viewpoints in both diagnosis and management, particularly in resource-constrained Indian environments where molecular profiling may not always be practical. By documenting one of the rare Indian instances of RAEB-1 with del(5q), our research contributes to the limited regional literature and underscores the importance of early detection, rational pharmacotherapy, and patient-centred counselling. Ultimately, such comprehensive case reports offer significant clinical insights into the diverse progression of MDS and underscore the role of pharmacists in enhancing both supportive and disease-modifying treatment approaches.

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ABBREVIATIONS

MDS: Myelodysplastic Syndromes; **AML:** Acute Myeloid Leukaemia; **RAEB-1:** Refractory Anaemia with Excess Blasts-1; **IPSS-R:** Revised International Prognostic Scoring System; **BMI:** Body Mass Index; **SpO_2:** Peripheral Capillary Oxygen Saturation; **MCV:** Mean Corpuscular Volume; **MCHC:** Mean Corpuscular Hemoglobin Concentration; **ESR:** Erythrocyte Sedimentation Rate; **PT:** Prothrombin Time; **aPTT:** Activated Partial Thromboplastin Time; **FISH:** Fluorescence In Situ Hybridization; **del(5q):** Deletion of the Long Arm of Chromosome 5; **TNF-α:** Tumor Necrosis Factor Alpha; **IL-6:** Interleukin-6; **RPS14:** Ribosomal Protein S14; **CSNK1A1:** Casein Kinase 1 Alpha 1; **TP53:** Tumor Protein p53; **TET2:** Ten-Eleven Translocation 2 Gene; **DNMT3A:** DNA Methyltransferase 3 Alpha; **SF3B1:** Splicing Factor 3B Subunit 1; **SRSF2:** Serine/Arginine-Rich Splicing Factor 2; **ASXL1:** Additional Sex Combs Like 1; **AZA-001:** Azacitidine Clinical Trial Study; **RMP:** Registered Medical Practitioner.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

No financial support was received for this work.

KEY CLINICAL MESSAGE

The presentation of RAEB-1 with isolated del(5q) represents a rare and diagnostically complex case of myelodysplastic syndrome in India; prompt identification, incorporation of cytogenetic results, and careful application of hypomethylating treatment can greatly affect prognosis and assist in counselling for both patients and their families.

ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was considered unnecessary for this case report, in accordance with institutional policies and international standards. The patient's legal guardian provided written informed consent to participate.

CONSENT FOR PUBLISH DECLARATION

Written informed consent was obtained for the publication of clinical details, and anonymised data was acquired from the patient. The authors affirm that all identifying information has been omitted to ensure the patient's anonymity.

AVAILABILITY OF DATA AND MATERIALS

The information collected during the current case report is not publicly accessible due to patient confidentiality; however, it can be obtained from the corresponding author upon reasonable request.

AUTHORS CONTRIBUTIONS

Renu Sri Tamanam gathered patient data, conducted a literature review, and contributed to the preparation of the manuscript. Dr. Pavan Kumar Yanamadala initiated the study, oversaw the clinical evaluation, and thoroughly revised the manuscript for its intellectual content. Abhi Chowdary Gannamaneni, Ratna

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