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A Rare Case of Bronze Diabetes Presenting as Diabetes Ketoacidosis

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Abstract

A decrease in the beta globulin chain, or thalassemia major, can lead to splenomegaly, extramedullary haematopoiesis, and severe anaemia. Iron overload occurs as a result of the mandatory blood transfusions required by thalassemia major patients to maintain acceptable erythrocyte levels. Secondary haemochromatosis, often known as bronze diabetes, is a fatal medical disease that may arise as a result of multiple blood transfusions. A 16-year-old male presented with complaints of polyuria, polydipsia and breathlessness in EMR. His random blood sugar was 564mg/dl, serum ketones were positive, arterial blood gas analysis showed high anion gap acidosis. His mother informed the patient was known case of thalassemia major since five years of age and was undergoing blood transfusions every 21 days along with chelation therapy. The patient was diagnosed to have diabetic ketoacidosis and was stabilized. Diffuse hyperpigmentation was evident on the sun-exposed areas of skin. Diagnosed as bronze diabetes the patient is on multiple doses of insulin and on chelation therapy.

Keywords: Thalassemia Major; Ferritin; Discoloration of Skin; Diabetic Ketoacidosis

Case Presentation

A 16- year-old male student of tenth standard presented with complaints of polyuria, polydipsia since one week and breathlessness since 2 days in EMR. He also complained of weakness, fatigue and lethargy since one week. There was no history of pain in abdomen, nausea, and vomiting, altered sensorium. No other major complaints. His mother informed that he is a diagnosed case of thalassemia major from the age of five years needing blood transfusion once every 21 days. There was history of splenectomy done six years back, but no details were available. He has been on iron chelation therapy from the age of eleven years. The patient has been on T. Deferasirox 400mg 3 times in a day. On further questioning it was found that both parents have beta thalassemia trait and his younger sister too has been diagnosed to have thalassemia major and is also receiving blood transfusion. No other significant history.

Physical Examination

The patient was conscious oriented, afebrile, HR 108/min regular, RR 24breaths/min and BP 100/60mmHg. There was

no fruity odour. There was pallor, no clubbing, cyanosis, icterus, and lymphadenopathy.

He had chipmunk facies, frontal bossing, prominent zygomatic bones and depression of nasal bridge. There was malocclusion of teeth (Figure 1).

His skin showed darkening along with diffuse hyperpigmentation (bronze) in sun-exposed areas (Figure 2). There was xerosis and pruritus. There was freckling over the face.

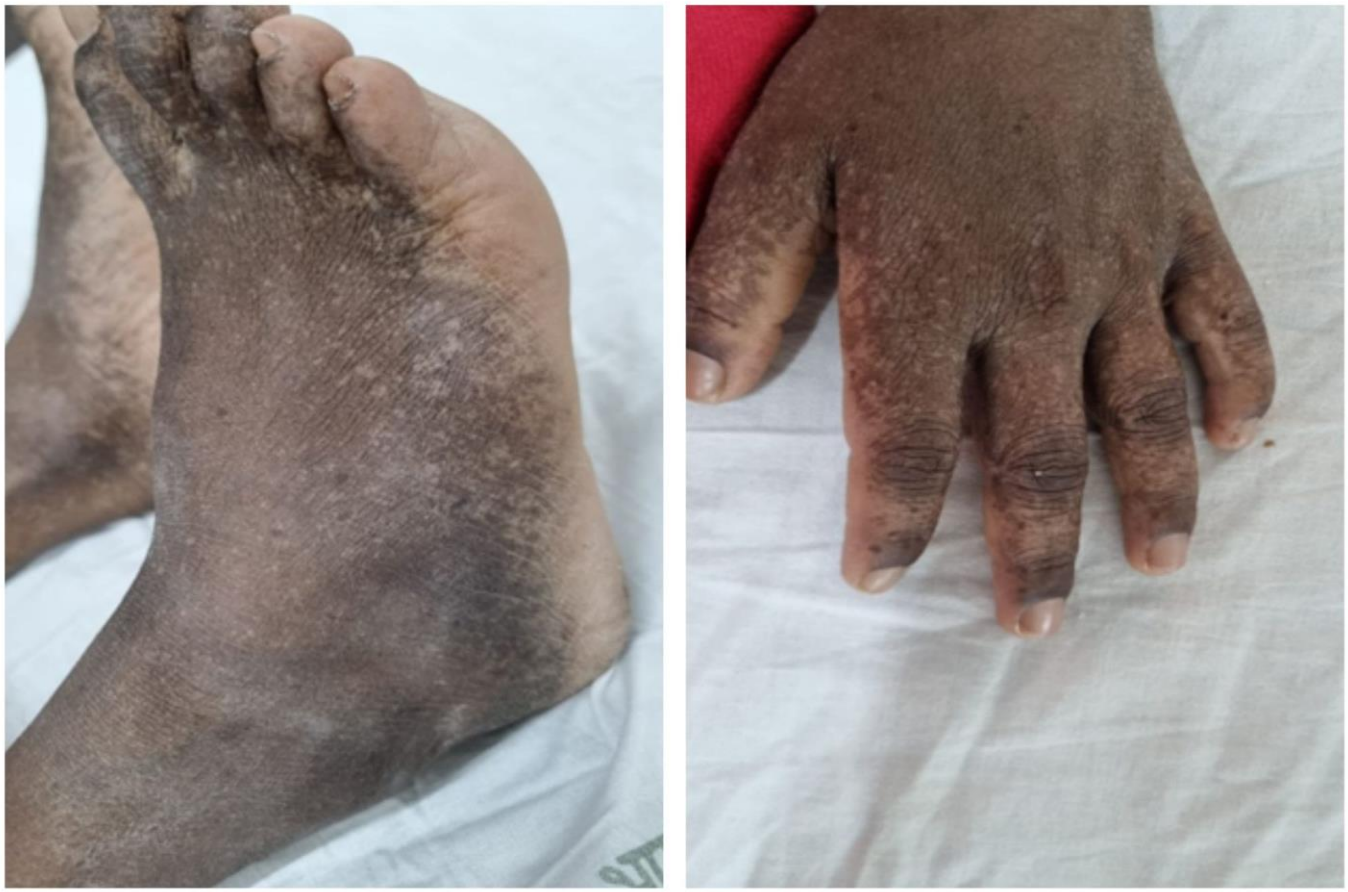


Figure 1: Hands and Feet showing hyperpigmentation with hypopigmented macules.

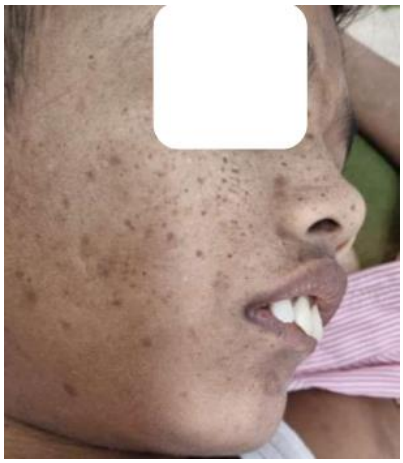


Figure 2: Freckling over the face and malocclusion of teeth.

His height was 136.2cm (- 2.89 SDS) with a weight of 27.9 kg and BMI of 15kg/m2 (Figure 3). His SMR:- bilateral testicular volume was 2cc , stretched penile length of 4cm , and P 1 (Tanner Stage in boys).

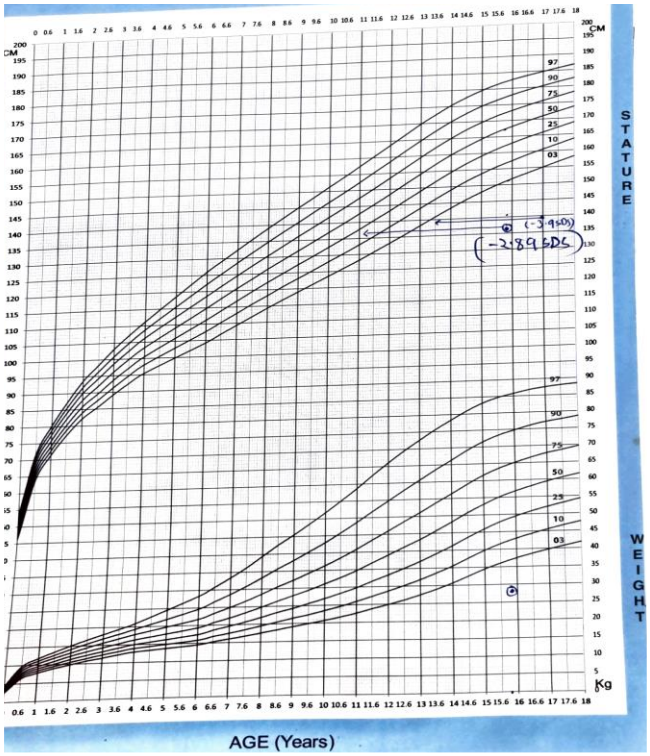


Figure 3: Stature for Age, Weight for Age.

Systemic Examination

Abdominal scar was seen in the region of spleen. The liver was just palpable, and no ascites.

The rest of systemic examination did not reveal any significant abnormality.

Investigations

On admission his random blood sugar was 564mg/dl, serum ketones were positive, arterial blood gas analysis showed high anion gap acidosis (Na 141, Cl 114, HCO3 14).

Laboratory Panel (Table)

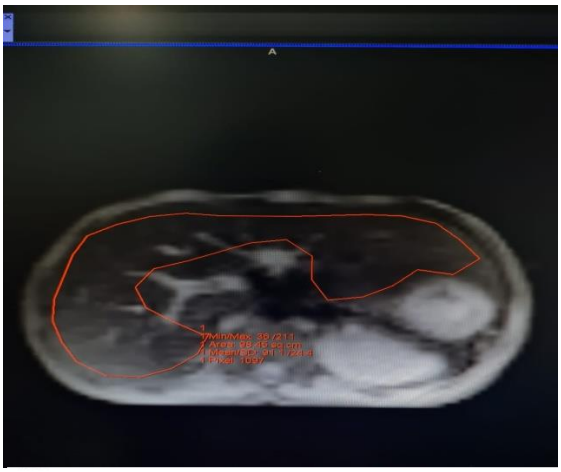
Test	Observed Values	Reference Range
Hemoglobin	7.1	12.5-18 g/dl
White Blood Cell	9100	4000-11000/mm3
Platelets	400000	150000-450000/µL
Blood Urea Nitrogen	11	10-15 mg%
Creatinine	1.2	0.7 -1.5 mg /dl
SGOT	53	4- 40 IU/L
SGPT	22	5- 40 IU/L
Total Bilirubin	1.6	0.2 – 0.8 mg/dl
Calcium	9	9.0 – 11mg/dl
Phosphorous	7.2	2.5 – 5.0 mg%
Alkaline Phosphatase	378	Up-to 117.0 U/L
Vitamin D3 Level	22	20 - 40 ng/ml
S. Proteins	6.3	6-8.3 g/dl
Albumin	3.1	3.2-5.2 g/dl
Globulin	2.5	1.5-3.8 g/dl
Serum Na	135	135-155 mEq/L
Serum K	4	3.5-5.5 mEq/L
Serum Cl	107	96-106 mEq/L
Ferritin		
28. Nov. 2022	5048	24 -336 ng/ml
01.Feb.2023	4006	24 -336 ng/ml
09.May.2023	3048	24 -336 ng/ml
FBS	331	70-99mg/dl
PPBS	490	70-140mg/dl
Glycosylated Hemoglobin	11.2	< 5.5%
Serum Ketone	Positive	Negative
Urine Ketone	Positive	Negative
GAD-65 Antibody Type 1	0.64	Negative <10 IU/mL
Islet Antigen 2 Antibody	4.2	Negative < 28 U/mL
Islet Cell Antibody	Negative	Negative
Zinc Transporter 8 Antibody	3.63	Non-reactive < 10 AU/mL
Insulin antibody	4.2	Negative <10 U/mL
C Peptide	0.5	0.78-5.19 ng/ml
Thyroid Stimulating Hormone	2.9	0.4- 4.0 uIU/ml
Free T4	1.12	0.8-1.8 ng/dl
Testosterone	0.05	3 – 11 ng/ml
Luteinizing Hormone	0.12	2.5 – 10 mIU/mL
Follicle Stimulating Hormone	0.68	2.5 – 10 mIU/mL

Insulin- Like Growth Factor 1	50.84	115 – 307 ng/mL

FBS: Fasting blood sugar, PPBS: Post prandial blood sugar, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

The X-Ray chest PA view and X-ray of the wrist joint did not reveal any bony or structural changes. Ultrasonography of the abdomen showed liver parenchymal disease with mild hepatomegaly. The spleen was not visualized (post splenectomy status). The head, body and tail of pancreas appear normal is size, shape and echogenicity. No focal lesion noted in pancreas.

MRI of heart and liver (1.5 T) iron loading assessment was done (Figure 4).



MRI Cardiac/LIVER (1.5 T) –Iron Loading Assessment

Indication:

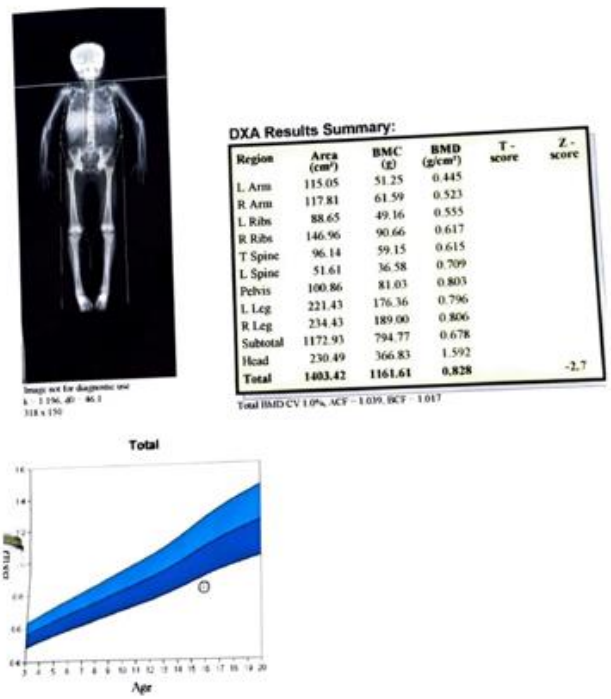
To assess iron loading.

Iron Loading:

Organ	T2*	MIC/LIC(mg/g)	Loading
Heart	10.9	2.45	MODERATE
Liver	3.7	7.1	MODERATE

Figure 4: MRI Cardiac/LIVER (1.5 T) iron loading assessment. T2* values showed moderate iron overload in both heart (T2* 10.9 ms, MIC 2.45 mg/g) and liver (T2* 3.7 ms, MIC 7.1 mg/g).

Dilated fundus examination showed normal fundus study.



DXA Results Summary:

Region	Area (cm²)	BMC (g)	BMD (g/cm²)	T-score	Z-score
L Arm	115.05	51.25	0.445		
R Arm	117.81	61.59	0.523		
L Ribs	88.65	49.16	0.555		
R Ribs	146.96	90.66	0.617		
T Spine	96.14	59.15	0.615		
L Spine	51.61	36.58	0.709		
Pelvis	100.86	81.03	0.803		
L Leg	221.43	176.36	0.796		
R Leg	234.43	189.00	0.806		
Subtotal	1172.93	794.77	0.678		
Head	230.49	366.83	1.592		
Total	1403.42	1161.61	0.828		-2.7

Total

Age

Figure 5: A DEXA scan done revealed secondary osteoporosis Z score - 2.7

The patient was treated and stabilized for diabetic ketoacidosis. Later he was shifted to multiple doses of insulin (basal bolus regime). T. Deferasirox 400mg 3 times in a day was continued. Injection Testosterone 50mg once a month was started for the first 3 months to be increased gradually. The patient was also started and calcium and vitamin D supplements. Due to board exams the patient took discharge against medical advice and has been asked to follow up on regular basis.

Discussion

Hemochromatosis is a disorder that affects many organ systems and is associated with iron overload. Because the body cannot eliminate excess iron, high pathologic amounts of iron buildup in the body resulting in hemochromatosis. Since hemochromatosis causes skin discoloration and pancreatic illness, it has been dubbed "bronze diabetes." Erythropoiesis problems and their treatment with blood transfusions can lead to secondary hemochromatosis. Thalassemia, sickle cell anaemia, X-linked sideroblastic anaemia, hereditary spherocytosis, and pyruvate kinase deficiency are the main causes of secondary

hemochromatosis (1). In transfusional hemochromatosis, retained iron is mostly deposited in the reticuloendothelial cells. The extra iron gets stored as hemosiderin within the cells. This ultimately results in the death of the cells and their replacement by a fibrous buildup, which damages or impairs the function of the organs. Patients who get repeated, typically ongoing transfusions of red blood cells experience the same mechanism (2). Hemochromatosis affects the liver, pancreas, heart, thyroid, joints, skin, gonads, and pituitary among other organs. The main symptom of pancreatic iron deposition is diabetes. In people who exhibit symptoms, the incidence of diabetes is almost 50%. The patient presented with osmotic symptoms. Apart from diabetes mellitus, the patient also had other endocrine issues like, short stature, and hypogonadism (3).

Although symptoms vary depending on the organ involved, extreme tiredness, lethargy, weakness, and arthralgias is a common complaint which was seen in the index patient. To diagnose this disorder, a complete family history and a high index of suspicion are needed. The patient's parents had thalassemia trait and the sibling had thalassemia major.

One of the initial symptoms of idiopathic hemochromatosis is skin discoloration, or diffuse hyperpigmentation, which affects over 90% of individuals. Even while it might not be severe, the skin's sun-exposed regions tend to show it more (4). This was very pronouncedly evident in the patient.

Patients with elevated ferritin levels also require an echocardiography to assess cardiomyopathy, hormone levels to assess hypogonadism, and bone densitometry to assess osteoporosis (6). The patient echocardiography did not reveal cardiomyopathy, but showed regional wall motion abnormality with an ejection fraction of 47.5%. The DEXA scan showed secondary osteoporosis.

Thought the patient had moderate iron overload in both the heart and liver, as shown in MRI (7). He neither had any symptoms of cardiac arrhythmias, congestive heart failure nor dilated cardiomyopathy nor did he have gynecomastia, ascites, or other features suggestive of cirrhosis or liver failure respectively. Although most patients have raised aminotransferase levels and elevated liver enzymes, these levels are typically not more than twice those of normal. The patient had very similar liver profile (8).

Genetic testing was not possible due to financial constraints.

To conclude, our case report, with its unique format, contributes to the body of knowledge by providing a more comprehensive understanding of the disease process and patient management.

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Contributors

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Informed Patient Consent for Publication

Signed informed consent was obtained from the patient

Disclosures

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