Case Report

DOI: 10.7860/JCDR/2026/86063.23960

Internal Medicine Section

Glycogen Storage Disease Type IIIa Presenting as Progressive Hepatomegaly and Abdominal Distension in a Three-year-old Child: A Case Report

UDAY KUMAR VANGALA[1], SAMRUDDHI PATIL[2], VIJENDRA KIRNAKE[3], AMEYA VAIDYA[4], BHAGYESH SAPKALE[5]

ABSTRACT

Glycogen Storage Disease type IIIa (GSD IIIa) is known to be a rare autosomal recessive metabolic disorder which results due to deficiency of the Glycogen Debranching Enzyme (GDE), that further leads to abnormal glycogen accumulation in liver and muscles. The current case report describes a three-year-old female child who presented with progressive abdominal distension, hepatomegaly since last one year. Clinical evaluation of patient showed short stature for age, overweight build also markedly enlarged liver. Laboratory investigations of patient reported persistently elevated levels of transaminases, dyslipidaemia while imaging showed hepatomegaly along with presence of a characteristic beaver's tail appearance. Liver biopsy revealed intra-hepatocytic accumulation of glycogen having plant cell-like morphology. Whole exome sequencing was done which helped to identify a homozygous frameshift mutation in the amylo-alpha-1,6-glucosidase and 4-alpha-glucanotransferase (AGL) gene (c.53_54insTCCT), thus confirming the diagnosis of GSD IIIa. The patient was then managed conservatively, who also remained under close follow-up. The present case highlights about importance of early recognition, histopathological correlation as well as molecular confirmation of GSD IIIa to facilitate timely management and long-term surveillance for potential progressive complications.

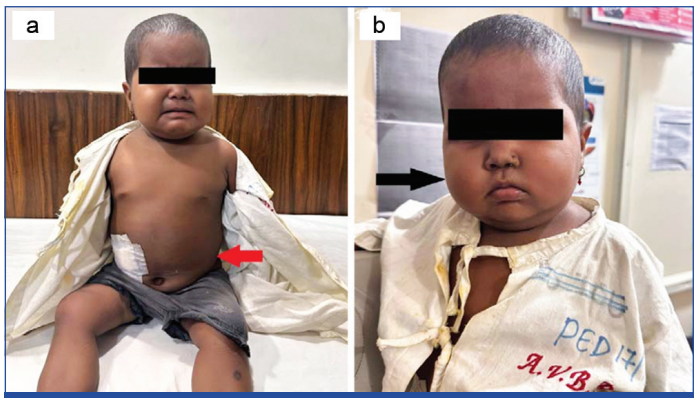
Keywords: Amylo-alpha-1,6-glucosidase and 4-alpha-glucanotransferase gene, Beaver's tail appearance, Debranching enzyme deficiency, Errors of metabolism, Paediatric liver disease

CASE REPORT

A three-year-old female child was brought by her parents with the chief complaint of progressive abdominal distension for the past one year. According to the parents, the child was apparently well one year prior to presentation, when they first noticed gradual enlargement of the abdomen. The distension was insidious in onset, progressively increasing in nature, which was not associated with abdominal pain, fever, vomiting, jaundice, altered bowel-bladder functionality. The parents also reported that the child had been overweight since birth; however, the abdominal distension became more prominent over the last year. The patient was born at term via normal vaginal delivery having a birth weight of 2.8 kg. There was no history of perinatal asphyxia. She also did not experience any recurrent neonatal hypoglycaemic episodes within first 48 hours of life, nor did she require intravenous dextrose infusion. No seizures were reported in patient during her neonatal period.

The child had a history of accidental thermal burn injury six months prior to the current admission inclusive of approximately 6% Total Body Surface Area (TBSA) affecting anterior chest and upper limbs. The burns were managed conservatively at local hospital without any further associated documented long-term complications. There was also no history of prolonged fasting as well as metabolic decompensation during that admission. The burn injury was unrelated to the current presentation. There was no significant past medical history, no history of similar illness in the family. Initially, child was evaluated at a local hospital, where blood investigations showed deranged liver enzymes (Serum Glutamic-Oxaloacetic Transaminase (SGOT) 161 IU/L, Serum Glutamate Pyruvate Transaminase (SGPT) 116 IU/L), and ultrasonography of the abdomen showed gross hepatomegaly then she was subsequently referred to present tertiary care hospital for further evaluation and management.

On admission, the child's vital signs were stable with a pulse rate of 90/min and a respiratory rate of 22/min. Anthropometric measurements of patient showed a height of 79 cm and weight of 14 kg, with a body mass index of 22.43 kg/m^2 and body surface area of 0.55 m^2. General physical examination was unremarkable except having short stature for age, puffy cheeks, overweight build, and a visibly distended abdomen as shown in [Table/Fig-1].



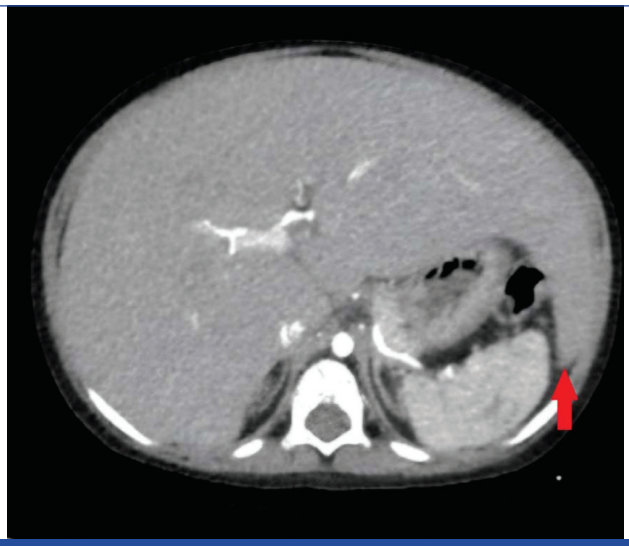
[Table/Fig-1]: Clinical photographs showing: (a) Short stature for age, overweight build, and abdominal distension highlighted with red arrow; (b) Puffy cheeks pointed with black arrow.

Systemic examination of patient revealed normal findings in the respiratory, cardiovascular, and central nervous systems. During hospital stay, child did not experience any episodes of hypoglycaemia, also serial blood glucose monitoring was within normal limits. Cardiac evaluation which was inclusive of two-dimensional echocardiography, showed normal cardiac structure and function, and absence of cardiomyopathy. The child was clinically active and playful, without any signs of muscle weakness, hypotonia, exercise intolerance, as well as there was no evidence of musculoskeletal involvement on examination.

Abdominal examination showed a distended abdomen along with full flanks and a horizontal scar located just above the

umbilicus because of a previous burn injury. On palpation, liver was enlarged with a span of approximately 14 cm, lower margin palpable 11 cm below the costal margin; it was firm, non-tender, with rounded margins. Percussion of patient's abdomen demonstrated dullness over right hypochondriac and lumbar regions, with no evidence of free fluid.

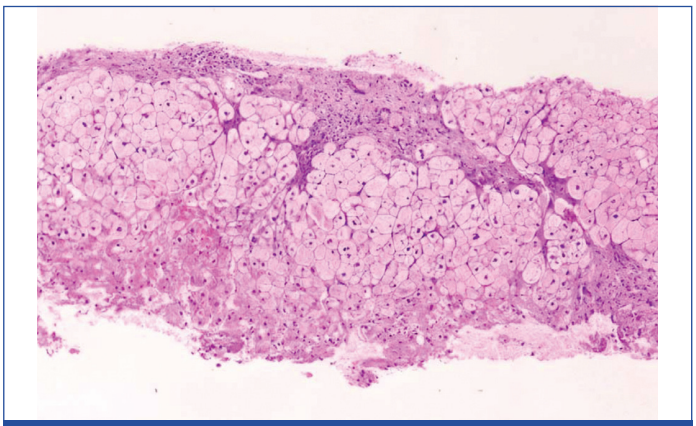
Laboratory investigations of the child also showed deranged liver function tests such as SGOT 530 IU/L and SGPT 279 IU/L on admission. Bilirubin levels were normal. Lipid profile of patient also showed hypertriglyceridaemia (271 mg/dL) with low High-Density Lipoprotein (HDL) (28 mg/dL). Random blood sugar and HbA1c were within normal limits. Renal function tests, thyroid profile, cortisol levels, coagulation parameters were within the normal limits. Viral markers which are specific for hepatitis B and C were non reactive. Contrast-Enhanced CT (CECT) scan of the abdomen revealed having hepatomegaly along with a characteristic "beaver's tail" appearance as shown in [Table/Fig-2].



[Table/Fig-2]: Contrast-Enhanced CT (CECT) scan of the abdomen showing hepatomegaly along with a characteristic "beaver's tail" appearance pointed with red arrow.

Based on clinical and radiological findings, a provisional diagnosis of a glycogen storage disorder was considered in child. In view of progressive hepatomegaly as well as elevated transaminases, differential diagnoses inclusive of non-alcoholic fatty liver disease, other glycogen storage disorders (Types I, VI, IX), lysosomal storage disorders (e.g., Pompe disease), chronic viral hepatitis, autoimmune hepatitis and Wilson disease were considered. Non alcoholic fatty liver disease was excluded because of absence of significant steatosis on biopsy. GSD Type I was ruled out because of absence of persistent hypoglycaemia, lactic acidosis, and hyperuricaemia. Lysosomal storage disorders were considered unlikely due to normal cardiac evaluation as well as absence of myopathy. Viral hepatitis markers were also non reactive. Autoimmune hepatitis, Wilson disease were excluded based upon normal relevant laboratory parameters as well as lack of supportive histological findings. Definitive confirmation was done through identification of a pathogenic homozygous variant in the AGL gene on molecular testing, consistent with GSD Type IIIa.

The patient was started on supportive management, including vitamin supplementation, ursodeoxycholic acid, and iron therapy. Ursodeoxycholic acid was administered at a dose of 15 mg/kg/day in two divided doses and was continued for six months with periodic liver function monitoring. Oral iron supplementation was given as elemental iron at a dose of 3 mg/kg/day and was continued for four months until normalisation of haemoglobin levels. Gastroenterology consultation was obtained, and liver biopsy along with genetic evaluation was advised. The liver biopsy of patient was performed under sedation and the procedure was uneventful. The histopathological examination of patient showed altered liver morphology where hepatocytes showed intra-hepatocytic accumulation displaying classical plant cell-like morphology, minimal steatotic changes, and mild chronic non specific inflammation in the portal triads, consistent with a glycogen storage disorder as shown in [Table/Fig-3]. Additional samples were sent for special staining as well as external evaluation, and whole exome sequencing was initiated; final reports were received during follow-up.



[Table/Fig-3]: Histopathological features of liver biopsy section showing intra-hepatocytic glycogen accumulation with classical plant cell-like morphology, minimal steatosis, and mild chronic nonspecific portal inflammation, suggestive of a glycogen storage disorder (H&E stain, 100x magnification).
H&E: Haematoxylin and eosin

Whole exome sequencing of the patient showed a homozygous likely pathogenic frameshift variant in the AGL gene (NM_000642.3) involving exon 2 (c.53_54insTCCT), resulting in a premature termination codon (p.Glu19ProfsTer9). This variant is known to be associated with GSD type IIIa that follows an autosomal recessive inheritance pattern. The molecular findings were thus consistent with patient's clinical presentation, biochemical abnormalities, imaging features, and histopathological findings, thereby it helped confirming molecular diagnosis of GSD type IIIa.

During her hospital stay, abdominal girth monitoring showed no further increase in abdominal size. Repeat liver function tests further revealed having persistently increased levels of transaminases (SGOT 422 IU/L, SGPT 298 IU/L). In view of biopsy, molecular findings, final diagnosis of GSD type IIIa was confirmed.

Following confirmation of the diagnosis, dietary counseling was initiated. The child was then advised a high-protein diet with controlled complex carbohydrates, along with suggested to take frequent small meals to prevent fasting-related metabolic derangements. Uncooked Cornstarch (UCS) supplementation was recommended as a slow-release source of glucose which helped to prevent potential fasting hypoglycaemia. Simple sugars, refined carbohydrates were advised to be avoided for minimising postprandial glycaemic fluctuations and excessive glycogen accumulation.

The gastroenterology team thereby further advised long-term follow-up and evaluation for liver transplantation only if progressive dysfunction of liver develops. The parents were counselled in detail regarding the diagnosis, prognosis, and need for further follow-up. The child was then discharged in stable condition with supportive medications, also advised regular follow-up. The child was followed up regularly in the paediatric gastroenterology outpatient department. At 6-month follow-up, she was clinically stable with no episodes of hypoglycaemia and no further increase in abdominal girth. Liver enzymes also showed mild persistent elevation along with partial improvement as compared to baseline values.

DISCUSSION

The GSD IIIa is known to be a rare autosomal recessive inborn error of carbohydrate metabolism caused by deficiency of the GDE, encoded by the AGL gene [1]. This enzyme plays a very important role in

glycogenolysis by enabling the complete degradation of glycogen into glucose; its deficiency leads to accumulation of structurally abnormal glycogen (limit dextrin) within affected tissues [1,2]. GSD IIIa is most common subtype of GSD III which is characterised by involvement of both hepatic and skeletal/cardiac muscle, thus distinguishing it from subtype IIIb that affects only the liver [2]. Clinically, patients having GSD IIIa usually present in infancy, early childhood with complaints like hepatomegaly, fasting hypoglycaemia, hyperlipidaemia, and growth retardation [3]. The skeletal muscle and cardiac involvement can become progressively more apparent in adolescence, adult age [3]. Long-term complications which are inclusive of progressive hepatic disease (fibrosis or cirrhosis), cardiomyopathy, and skeletal myopathy with varying severity [3,4]. Genetic testing which helps to identify biallelic pathogenic variants in AGL further establishes the diagnosis of GSD IIIa for which dietary management remains the mainstay of therapy [4].

Although glycogen storage disorders were identified already in early 20th century, adequate biochemical basis of GSD III evolved gradually with advancements into metabolic research [5]. Early clinical descriptions of patients having enlarged livers as well as fasting intolerance were reported in late 1920s and 1930s, but it was not until the mid-20th century that underlying defect in glycogen structure was characterised properly [6]. In early period of 1950s, Illingworth B and Cori GT described abnormal glycogen with short outer chains in affected individuals, as well as subsequent investigations suggested that this abnormality was due to defects in glycogen debranching activity [7]. These observations laid down foundation for diagnosis of GSD III as a distinct biochemical disorder with affected metabolism of glycogen in patients [5,7].

GSD III including the IIIa subtype, is known as a very rare autosomal recessive metabolic disorder having a generally low incidence in the global population [5]. GSD III occurs at a frequency of one case per 100,000 live births as reported in few broad population studies. Although an accurate and precise global birth prevalence remains unclear because of under-reporting as well as variable diagnostic practices [5,8]. GSD IIIa subtype which affects both liver and muscle, accounts usually around 80 to 85% of all GSD III patients [8]. Although general population incidence of GSD IIIa is very low, higher prevalences are also previously documented in certain genetic isolates due to founder effects, which is inclusive of Faroe Islands where GSD IIIa prevalence has been calculated approximately one in 3,600 live births, thus far exceeding global values [9].

In a case series reported by Zhang Y et al., four Chinese patients having genetically confirmed GSD IIIa from three unrelated families inclusive of two siblings were evaluated longitudinally [10]. The cohort comprised three males, one female having symptom onset between 1-2 years of age most usually presenting with progressive abdominal distension in early childhood [10]. At first presentation (ages 14-32 years), predominant complaint was physical weakness which was accompanied by markedly elevated Creatine Kinase (CK) levels reflecting significant skeletal muscle involvement [10]. Hepatomegaly with raised transaminases was common during childhood while asymptomatic Left Ventricular Hypertrophy (LVH) with preserved ejection fraction was observed in three patients [10]. Ketotic hypoglycaemia was documented in only one patient, whereas hyperlipidaemia was noted in the others [10]. Electromyography showed myopathic motor unit potentials in all cases while nerve conduction studies and brain imaging were normal [10]. Muscle Magnetic Resonance Imaging (MRI) in one patient further showed mild fatty infiltration, selective involvement of posterior as well as lateral lower limb muscles [10].

Genetic analysis of AGL gene identified three different mutations among four patients including a novel homozygous frameshift mutation (c.206dupA, p.N69Kfs*8), a homozygous splice-site mutation (c.1735+1G>T) and compound heterozygous mutations {c.1735+1G>T and c.2590C>T (p.R864X)} in sibling pair [10]. Following initiation of high-protein diet (2 g/kg/day) as well as UCS therapy (1.5 g/kg per dose, 1-3 times daily) significant improvement in muscle weakness, normalisation of preprandial blood glucose and lipid levels were observed over two years [10]. Cardiac hypertrophy remained stable, transaminase levels declined also no hepatic adenoma, cirrhosis, or hepatocellular carcinoma was detected during follow-up [10]. However, CK levels persisted at 5-10 times above upper limit of normal thereby suggesting ongoing myopathic involvement despite metabolic stabilisation [10]. This series demonstrates about multisystemic nature of GSD IIIa as well as points toward importance of early diagnosis, molecular confirmation, dietary therapy along with long-term follow-up of patients [10].

In a case reported by Kumru Akin B et al., a nine-year-old male child from Turkey having GSD-IIIa highlighted an important role of dietary composition in the progression and reversibility of cardiac involvement [11]. The patient harbouring a homozygous truncating AGL mutation (c.1783C>T; p.Arg595*), presented early having ketotic hypoglycaemia, hepatomegaly, myopathy, as well as further developed hypertrophic cardiomyopathy with left ventricular outflow tract obstruction in context of poor adherence to diet [11]. Introduction of a high-fat, high-protein, and low-carbohydrate regimen to the child supplemented with UCS led to marked clinical, biochemical, and echocardiographic improvement, including resolution of hypoglycaemia and regression of cardiomyopathy [11]. However, discontinuation of dietary therapy resulted further into clinical relapse, recurrence of cardiac hypertrophy, which again improved after re-initiation of the prescribed diet [11]. The present case illustrates about dynamic and diet-responsive nature of cardiomyopathy in GSD IIIa also it emphasises the importance of sustained adherence to diet along with long-term follow-up for preventing progression of disease, recurrence [11].

In a case reported by Senbanjo IO et al., a 14-month-old female child presented having progressive abdominal distension, marked hepatomegaly [12]. The child had associated hypoglycaemia, hyperuricaemia, hypercholesterolaemia and elevated transaminases thereby raising suspicion of GSD [12]. Liver biopsy showed classical swollen hepatocytes along with plant-like morphology and fibrous septa formation which was consistent with GSD [12]. The patient was commenced on frequent UCS feeds along with dietary modification resulting in significant clinical and biochemical improvement at 36-month follow-up including regression of hepatomegaly as well as normalisation of serum uric acid levels [12]. However, persistent elevation of AST, CK further suggested ongoing metabolic involvement in the patient [12]. This case emphasises about importance of early recognition, dietary intervention, long-term follow-up in children with suspected cases of GSD as timely management can improve growth parameters and hepatic outcomes in patients [12]. Comparative summary of reported GSD type IIIa cases and the present case is depicted in [Table/Fig-4] [10-12].

CONCLUSION(S)

The present case highlights GSD type IIIa which is an important also often under-recognised cause of progressive hepatomegaly and abdominal distension in early childhood. The current case report

Feature	Zhang Y et al., case series (China) [10]	Kumru Akin B et al., nine-year-old male (Turkey) [11]	Senbanjo IO et al., 14-month-old female [12]	Present case, three-year-old female (India)
Age at presentation	Onset at 1-2 years; evaluated at 14-32 years	Early childhood	14 months	3 years (symptom onset at 2 years; progressive abdominal distension for 1 year)

Sex	Three males, one female	Male	Female	Female
Initial Symptoms	Progressive abdominal distension in childhood; later weakness	Ketotic hypoglycaemia, hepatomegaly, myopathy	Progressive abdominal distension, hepatomegaly	Progressive abdominal distension, overweight, short stature
Metabolic abnormalities	Elevated CK in all; ketotic hypoglycaemia in one; hyperlipidaemia in others	Ketotic hypoglycaemia	Hypoglycaemia, hyperuricaemia, hypercholesterolaemia, elevated transaminases	Elevated transaminases, dyslipidaemia
Liver involvement	Hepatomegaly with raised transaminases in childhood; no adenoma/cirrhosis on follow-up	Hepatomegaly	Marked hepatomegaly; biopsy showed swollen hepatocytes with plant-like morphology and fibrous septa	Hepatomegaly, raised transaminases, "beaver's tail" appearance on CECT
Cardiac involvement	Asymptomatic LVH in three patients (stable on follow-up)	Hypertrophic cardiomyopathy (diet-responsive)	Not reported	No cardiac involvement detected
Muscle involvement	Prominent myopathy; markedly elevated CK; EMG abnormal in all	Myopathy	Persistent AST and CK elevation suggesting metabolic involvement	Not clinically significant at presentation
Neurophysiology /imaging	EMG: myopathic MUP; NCS & brain MRI normal; muscle MRI showed mild fatty infiltration (posterior/lateral lower limb muscles)	Not detailed	Not detailed	Not significant
Genetic variant	c.206dupA (p.N69Kfs*8, novel); c.1735+1G>T; c.2590C>T (p.R864X)	Homozygous c.1783C>T (p.Arg595*)	Not reported	Homozygous c.53_54insTCCT (p.Glu19ProfsTer9)
Management/ therapy	High-protein diet (2 g/kg/day) + UCS (1.5 g/kg/dose, 1-3 times/day)	High-fat/protein, low-carbohydrate + cornstarch diet	Frequent UCS feeds + dietary modification	Supportive therapy; dietary counselling initiated (high-protein diet, complex carbohydrates, frequent meals, UCS); long-term follow-up planned
Outcome	Improvement in weakness; normalisation of glucose and lipids; cardiac stability; CK persistently elevated	Regression of cardiomyopathy and hypoglycaemia; relapse on discontinuation	Significant improvement at 36 months; regression of hepatomegaly; normalisation of uric acid; persistent AST and CK elevation	Stable during hospital stay; liver dysfunction persists; monitoring for progression
Key learning points	Multisystemic nature; molecular heterogeneity; dietary response but persistent myopathy	Diet-responsive cardiomyopathy; importance of adherence	Early recognition, biopsy correlation, benefit of cornstarch therapy, long-term monitoring	Early diagnosis, molecular confirmation, need for long-term surveillance for hepatic progression

[Table/Fig-4]: Comparative summary of reported Glycogen Storage Disease (GSD) type IIIa cases and the present case [10-12].
EMG-Electromyography

reflects the value of a systematic diagnostic approach integrating clinical features, imaging, histopathology along with molecular genetic testing for definitive diagnosis. Early identification of the condition allows timely counselling, initiation of proper dietary and supportive management including high-protein intake, avoidance of prolonged fasting, and use of complex carbohydrates as well as facilitates structured long-term surveillance. Considering the potential of GSD IIIa for progressive hepatic, cardiac, and muscular complications over time, lifelong follow-up is very essential to optimise outcomes while preventing advanced-type involvement of organs in patients.

REFERENCES

[1] Sentner CP, Hoogeveen IJ, Weinstein DA, Santer R, Murphy E, McKiernan PJ, et al. Glycogen storage disease type III: Diagnosis, genotype, management, clinical course and outcome. J Inherit Metab Dis. 2016;39(5):697-704. Doi: 10.1007/s10545-016-9932-2.

[2] Wang W, We M, Song HM, Qiu ZQ, Zhang WM, Wu XY, Lu CX, Qi JM, Jing H, Li F. [Diagnosis of glycogen storage disease type IIIA by detecting glycogen debranching enzyme activity, glycogen content and structure in muscle]. Zhonghua Er Ke Za Zhi. 2009;47(8):608-12. Available from: https://pubmed.ncbi.nlm.nih.gov/19951495/.

[3] John TA, Anastasopoulou C. Glycogen storage disease. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Dec 21]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK459277/.

[4] Du C, Wei H, Zhang M, Hu M, Li Z, Zhang C, et al. Genetic analysis and long-term treatment monitoring of 11 children with glycogen storage disease type IIIa. J Pediatr Endocrinol Metab JPEM. 2020;33(7):923-30.

[5] Gümüş E, Özen H. Glycogen storage diseases: An update. World J Gastroenterol. 2023;29(25):3932-63. Doi: 10.3748/wjg.v29.i25.3932.

[6] Szymańska E, Jóźwiak-Dzięcielewska DA, Gronek J, Niewczas M, Czarny W, Rokicki D, et al. Hepatic glycogen storage diseases: Pathogenesis, clinical symptoms and therapeutic management. Arch Med Sci. 2019;17(2):304-13. Doi: 10.5114/aoms.2019.83063.

[7] Illingworth B, Cori GT. Structure of glycogens and amylopectins. III. Normal and abnormal human glycogen. J Biol Chem. 1952;199(2):653-60.

[8] Kumar TV, Bhat M, Narayanachar SG, Narayan V, Srikanth AK, Anikar S, et al. Molecular and clinical profiling in a large cohort of Asian Indians with glycogen storage disorders. PLoS ONE. 2022;17(7):e0270373. Doi: 10.1371/journal.pone.0270373.

[9] Santer R, Kinner M, Steuerwald U, Kjaergaard S, Skovby F, Simonsen H, et al. Molecular genetic basis and prevalence of glycogen storage disease type IIIA in the Faroe Islands. Eur J Hum Genet EJHG. 2001;9(5):388-91. Doi: 10.1038/sj.ejhg.5200632.

[10] Zhang Y, Xu M, Chen X, Yan A, Zhang G, Liu Z, et al. Genetic analysis and clinical assessment of four patients with Glycogen Storage Disease Type IIIa in China. BMC Med Genet. 2018;19(1):54. Doi: 10.1186/s12881-018-0560-6.

[11] Kumru Akin B, Ozturk Hismi B, Daly A. Improvement in hypertrophic cardiomyopathy after using a high-fat, high-protein and low-carbohydrate diet in a non-adherent child with glycogen storage disease type IIIa. Mol Genet Metab Rep. 2022;32:100904. Doi: 10.1016/j.ymgmr.2022.100904

[12] Senbanjo IO, Lamina MO, Kumolu-Johnson T, El-Said H, El-Guindi MAS. First report of glycogen storage disease Type IIIa in a Nigerian child. Niger Postgrad Med J. 2019;26(2):138-141. Doi: 10.4103/npmj.npmj_17_19.

PARTICULARS OF CONTRIBUTORS:
1. Senior Resident, Department of Gastroenterology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
2. Junior Resident, Department of Paediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
3. Professor, Department of Gastroenterology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
4. Senior Resident, Department of Gastroenterology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
5. Undergraduate Student, Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Uday Kumar Vangala,
Senior Resident, Department of Gastroenterology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha-442001, Maharashtra, India.
E-mail: udai.reddii@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Dec 26, 2025
- Manual Googling: Apr 04, 2026
- iThenticate Software: Apr 06, 2026 (3%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Dec 24, 2025**
Date of Peer Review: **Feb 19, 2026**
Date of Acceptance: **Apr 08, 2026**
Date of Publishing: **Aug 01, 2026**