NIGERIAN JOURNAL OF PAEDIATRICS

VOLUME 53 | NUMBER 3 | JULY - SEPTEMBER 2026

https://www.njpaediatrics.com

PRINT: ISSN 0302-4660

ONLINE: ISSN 2814-2985

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CASE REPORT



Nigerian Journal of Paediatrics 2026 (September); Volume 53(3):479-490.
https://dx.doi.org/10.63270/njp.v53i3.2000087.

Neonatal Diabetes Mellitus Masquerading as Sepsis in a Nigerian Infant: A Case Report

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Abstract

Neonatal diabetes mellitus (NDM) is a rare monogenic disorder presenting within the first six months of life. It is frequently under-recognised in low-resource settings due to its clinical overlap with severe sepsis. We report an 11-week-old Nigerian infant who presented with fever, respiratory distress, convulsions, severe dehydration, and altered consciousness, and was initially managed as severe bronchopneumonia with sepsis. Subsequent evaluation revealed marked hyperglycaemia (random blood glucose >33.5 mmol/L), metabolic acidosis (serum bicarbonate 11 mmol/L), and ketonuria, confirming diabetic ketoacidosis (DKA). The persistence and severity of hyperglycaemia beyond expected stress responses raised suspicion of an underlying metabolic disorder. The patient was managed with intravenous fluids, insulin therapy, and antibiotics, followed by careful metabolic stabilisation, after which a structured transition from insulin to oral glibenclamide was undertaken, resulting in sustained glycaemic control. In the absence of genetic testing, the favourable clinical and biochemical response to sulfonylurea therapy provided functional support for the diagnosis of NDM. The child has remained stable on follow-up care for two years. This case highlights the diagnostic challenge posed by the overlap between sepsis and metabolic disorders in early infancy and underscores the importance of routine glucose assessment in critically ill infants.

Keywords: Altered sensorium, Diabetic ketoacidosis, Glibenclamide, Hyperglycaemia.

Introduction

Neonatal Diabetes Mellitus (NDM) is a rare disease characterised by persistent hyperglycaemia presenting within the first six months of life. Unlike autoimmune Type 1 diabetes, it is a monogenic disorder affecting pancreatic β-cell development and insulin secretion. The estimated global incidence ranges from 1 in 100,000 to 1 in 500,000 live births, making it an uncommon but important cause of neonatal hyperglycaemia.[1-4] In high-income settings where genetic testing is readily available, researchers have identified and comprehensively characterised a growing number of NDM cases, with well-documented therapeutic success after switching from insulin to oral sulfonylureas in appropriate subtypes.[5-11]

In contrast, in many low-resource settings, including Nigeria, the true burden remains uncertain, likely due to under-recognition, frequent misdiagnosis, and limited access to definitive genetic testing, resulting in very few documented cases across Africa.[12-15] In Nigeria, only a few cases of neonatal diabetes mellitus have been reported. The report by Okolo *et al*.,[12] published over four decades ago, described neonatal diabetes mellitus occurring in association with infantile hypertrophic pyloric stenosis, without the use of sulfonylurea therapy. More recently, Obasa *et al*.[13] documented a case of neonatal hyperglycaemia in which distinguishing transient neonatal

diabetes from other causes posed a diagnostic challenge, and sulfonylurea therapy was not utilised. These reports highlight existing gaps in diagnosis and evolving management of neonatal diabetes mellitus within the local context.

NDM is genetically heterogeneous and results from mutations in several genes involved in pancreatic β-cell development, function, and insulin secretion. The most common defects involve activating mutations in KCNJ11 and ABCC8, which encode subunits of the ATP-sensitive potassium (KATP) channel, as well as mutations in the insulin gene (INS). Less frequently, abnormalities in genes responsible for pancreatic development may be implicated. NDM is broadly classified into transient neonatal diabetes mellitus (TNDM), which may remit but can relapse later in life, and permanent neonatal diabetes mellitus (PNDM), which requires lifelong treatment.[4, 16-18] While genetic testing is essential for definitive diagnosis and treatment guidance, it remains largely unavailable in many resource-limited settings.[12-14] Consequently, in these settings, diagnosis often relies on clinical features, biochemical findings, and response to therapy.[8, 19 20]

Clinically, affected infants may present with poor feeding, dehydration, failure to thrive, and lethargy, while diabetic ketoacidosis (DKA), though uncommon in early infancy, may occur and closely mimic severe sepsis. This overlap frequently leads to delayed recognition of the underlying metabolic disorder, particularly in environments where infectious conditions predominate. Early consideration of NDM is therefore essential, with simple bedside glucose testing serving as a critical tool for detection. Prompt diagnosis and timely initiation of appropriate therapy, including insulin and, where feasible, transition to sulfonylureas, are crucial to improving outcomes and reducing morbidity and mortality.[21-25] Evidence indicates that timely diagnosis and appropriate treatment can achieve good glycaemic control and favourable developmental outcomes in affected infants.[7, 26, 27]

The case reported represents one of the few reported cases from Nigeria and the wider African region demonstrating successful drug management of neonatal diabetes mellitus.

Case Presentation

An 11-week-old male infant was referred to the Emergency Paediatric Unit of the Federal Medical Centre, Bida, Niger State, Nigeria, on account of a four-day history of high-grade fever, followed by cough and progressive difficulty in breathing. Three days into the illness, he developed worsening respiratory distress, and approximately four hours before presentation, he had multiple episodes of generalised tonic convulsions with subsequent loss of consciousness. He had last breastfed about two hours before the onset of seizures. Based on the presenting features, he was referred with a provisional diagnosis of severe bronchopneumonia complicated by sepsis. Pregnancy, delivery, and early neonatal period were uneventful. He was delivered at term via spontaneous vaginal delivery with a birth weight of 2.8 kg and cried immediately after birth. Breastfeeding was initiated within the first hour of life and was exclusive. There was no history suggestive of perinatal asphyxia or prior neonatal illness, and immunisations were appropriate for age. A family history of diabetes mellitus was present in the maternal grandfather.

At presentation, the infant weighed 3.6 kg and appeared acutely ill, unconscious, and severely dehydrated, with a Blantyre coma score of 3/5. His axillary temperature was 40.1°C, pulse rate 194 beats per minute, and respiratory rate 80 breaths per minute, with oxygen saturation ranging between 88% and 90% on room air, improving to 98% with intranasal oxygen. Neurological examination revealed generalised hypotonia and a depressed anterior fontanelle, while the pupils were equal and reactive to light. There were no dysmorphic features, pallor, or jaundice. Respiratory examination

showed widespread bilateral coarse crepitations consistent with bronchopneumonia, while the abdomen was soft and non-distended with no organomegaly.

Investigations and Laboratory Findings

Initial investigation findings were consistent with severe bronchopneumonia with sepsis. Chest radiography demonstrated bronchopneumonic changes, while full blood count showed leucocytosis with neutrophil predominance and toxic granulations. The malaria rapid diagnostic test was negative. As part of a routine emergency assessment, a bedside random blood glucose test performed a few hours after admission was markedly elevated and unrecordably high. A repeat blood glucose level remained markedly elevated (>33.5 mmol/L). Urine dipstick testing revealed glycosuria (2+) and significant ketonuria (3+). Further biochemical evaluation showed a reduced serum bicarbonate level (11 mmol/L) and urine pH of 6.0. Serum electrolytes demonstrated mild derangements in sodium and chloride, with urea and creatinine at the upper limit of normal; these parameters remained relatively stable on serial monitoring. The details of the laboratory findings are summarised in Table I.

Table I: Serum electrolytes and renal function profile at presentation and during hospitalisation

Parameter	Reference Range	At Presentation	Day 2	Day 4	Interpretation
Sodium (Na^+) (mmol/L)	135 – 145	132	135	138	Mild hyponatremia at presentation, normalised with treatment
Chloride (Cl^-) (mmol/L)	98 – 108	95	100	102	Mild hypochloraemia initially, improved with therapy
Potassium (K^+) (mmol/L)	3.5 – 5.5	4.6	4.4	4.2	Within normal range
Bicarbonate (HCO_3^-) (mmol/L)	21 – 28	11	16	22	Markedly reduced at presentation, corrected progressively
Urea (mmol/L)	2.3 – 6.5	7.2	6	4.8	Mild elevation consistent with dehydration
Creatinine (µmol/L)	35 – 80	78	70	60	High-normal initially, but improved with rehydration

Management and hospital course

On admission, the infant was promptly stabilised with judicious fluid resuscitation aimed at correcting severe dehydration and metabolic derangements, as outlined in Table II. Urethral catheterisation was done to facilitate accurate urine output monitoring and to guide ongoing fluid therapy. Initial rehydration was achieved with isotonic saline, followed by carefully titrated maintenance fluids adjusted according to serial glycaemic measurements and overall clinical status. Continuous intravenous insulin infusion was commenced early to control hyperglycaemia and suppress ketosis, while intravenous broad-spectrum antibiotic therapy was initiated for bronchopneumonia, which was considered the precipitating factor for the metabolic crisis (Table II). During the acute phase, the child was kept *nil per os*, and dextrose-containing fluids were introduced as blood glucose levels declined, allowing safe continuation of insulin therapy. Serial urine dipstick monitoring demonstrated progressive resolution of glycosuria and ketonuria in keeping with improving metabolic control. The trend in blood glucose levels during this period is illustrated in Figure 1, showing a steady decline with appropriate therapeutic adjustments.

Table II: Stepwise fluid, insulin, antibiotic, and glycaemic management (Weight = 3.6 kg)

Day / Phase	Fluid Therapy	Insulin Therapy	Antibiotic Therapy	Urine Findings	Clinical / Glycaemic Progress
Day 0 (Admission – Acute DKA)	IV 0.9% Normal Saline 20 ml/kg over 1 hr (72 ml), then 0.45% saline at 5 ml/kg/hr (18ml/hr)	IV regular insulin infusion 0.1 IU/kg/hr (0.36 IU/hr)	Ceftriaxone 75 mg/kg/day (270 mg/day)	Glycosuria 2+, Ketonuria 3+	RBS >33.5 mmol/L; severe dehydration and acidosis
Day 1 – 2 (Correction phase)	Continued IV fluids; switched to paediatric saline + 5–10% dextrose when RBS <14 mmol/L	IV insulin infusion continued at 0.36 IU/hr	IV Ceftriaxone continued	Glycosuria 1–2+, Ketonuria 2+	Gradual fall in blood glucose; improving sensorium
Day 3 – 4 (Resolution phase)	IV fluids tapered; oral feeds reintroduced	Transitioned to SC regular insulin 0.5 IU/kg/day (1.8 IU/day) in 3 divided doses (0.6 IU per dose)	IV Ceftriaxone continued	Trace glycosuria, Ketonuria 1+	Ketosis resolving; stable glucose levels
Day 5 – 6 (Stabilisation phase)	IV fluids stopped. Breastfeeding on demand	SC insulin continued	IV Ceftriaxone continued	Urine negative for ketones; trace glycosuria	Normal consciousness and feeding
Day 7 (Transition phase)	Oral feeds only	Oral glibenclamide started at 0.17 mg/kg/day (0.6 mg/day); insulin gradually withdrawn	Switched to oral Cefixime 8 mg/kg/day (29 mg/day)	Urine sugar negative	Stable glycaemia (RBS 5–7 mmol/L)
Day 8–14 (Discharge phase)	Oral feeds only	Glibenclamide titrated to 0.3 mg/kg/day (1.1 mg/day)	Oral Cefixime completed	Normal urinalysis	Discharged stable; continued follow-up

Close monitoring of vital signs, neurological status, fluid balance, and biochemical parameters guided ongoing management.

With resolution of metabolic acidosis and restoration of clinical stability, the child regained consciousness and demonstrated progressive improvement in hydration status and respiratory function. Oxygen support was gradually weaned as respiratory distress subsided and repeated clinical and radiological assessments confirmed resolution of the chest infection. Intravenous insulin was subsequently transitioned to subcutaneous insulin, followed by a carefully monitored switch to oral glibenclamide after full stabilisation. Breastfeeding was reintroduced and well tolerated, with sustained clinical improvement thereafter.

Trial Transition to Sulfonylurea Therapy

After achieving sustained metabolic stability, subcutaneous insulin was gradually withdrawn and oral glibenclamide was introduced as a closely monitored therapeutic trial in the absence of genetic confirmation. Treatment was initiated at 0.17 mg/kg/day (≈0.6 mg/day) and titrated to 0.3 mg/kg/day (≈1.1 mg/day) based on serial blood glucose monitoring, which demonstrated progressive and sustained glycaemic stabilisation.

The child demonstrated excellent and sustained clinical and biochemical response to glibenclamide without hypoglycaemia or adverse effects, thus supporting the diagnosis of sustained hyperglycaemia from neonatal diabetes mellitus. In the absence of molecular confirmation, this response provided functional diagnostic evidence. Glycaemic control

remained stable with dose adjustments in line with growth, while urinalysis confirmed resolution of glycosuria and ketonuria. The child completed treatment for bronchopneumonia, was discharged after 14 days on oral glibenclamide, and has maintained normal growth, development, and metabolic control over two years of follow-up (Figure 2).

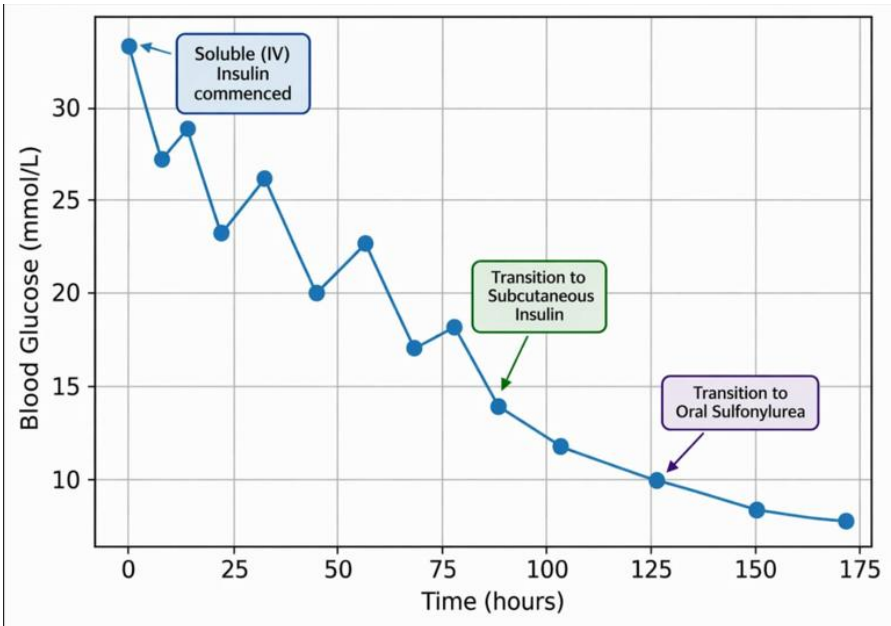


Figure 1: Blood Glucose Trend During Acute Management of Diabetic Ketoacidosis

This shows an initial fluctuation under intravenous insulin, followed by stabilisation with subcutaneous insulin and subsequent transition to oral Sulfonylurea (Glibenclamide) therapy.

Growth trajectory has been satisfactory, demonstrating progressive catch-up growth over time. Although occasional transient weight faltering occurred during episodes of mild intercurrent childhood illnesses, these were followed by appropriate recovery and continued upward growth trend, as illustrated in Figure 3. Other anthropometric parameters, including length and mid-upper arm circumference, remained within acceptable ranges for age, indicating preserved nutritional status and overall growth. Developmental milestones have been appropriately attained, with no evidence of neurodevelopmental delay. There has been no recurrence of significant respiratory symptoms or major intercurrent illnesses during the follow-up period. Caregivers have remained actively engaged in the child's care, demonstrating good adherence to home glucose monitoring, medication administration, and scheduled clinic visits following structured education and counselling.

Discussion

This case highlights the diagnostic challenge of neonatal diabetes mellitus (NDM) in resource-limited settings, where severe infections are far more commonly encountered than metabolic disorders in early infancy. In the present case, bronchopneumonia with sepsis was clinically and laboratory-confirmed and most likely served as the precipitating factor for diabetic ketoacidosis while simultaneously obscuring recognition of the underlying neonatal diabetes mellitus.[8, 28, 29] The coexistence of infection and metabolic decompensation illustrates how NDM may be overlooked or diagnosed late, particularly where access to specialised investigations is limited. This reflects a broader systemic issue in low- and middle-income settings, where infectious aetiologies dominate clinical thinking and rare metabolic conditions are not routinely prioritised in acutely ill infants.[10, 11, 30]

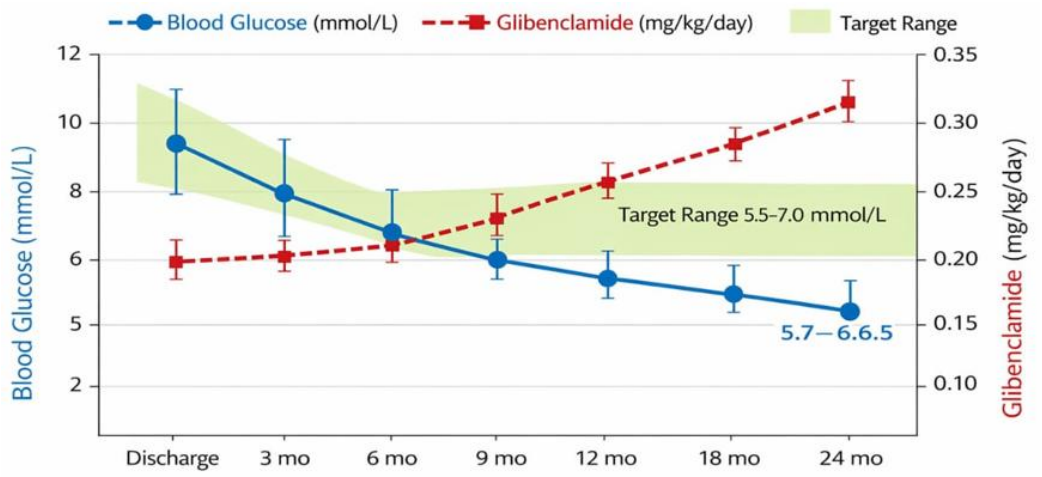


Figure 2: Trend of Blood Glucose Level During Transition from Insulin to Glibenclamide Therapy

Serial blood glucose measurements demonstrating progressive improvement in hyperglycaemia at discharge to sustained values within the target range (5.5 – 7.0 mmol/l) over 24 months, corresponding with gradual escalation of oral Glibenclamide dose. This pattern reflects restoration of ATP-sensitive potassium channel function and enhanced insulin secretion.

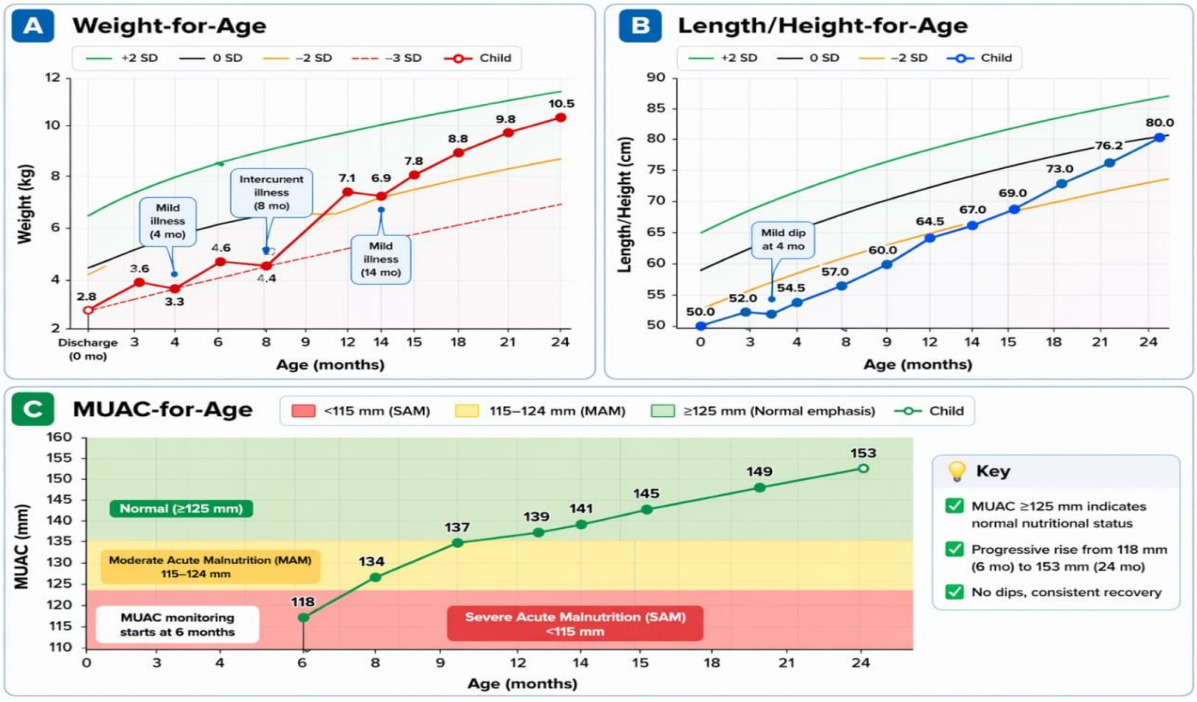


Figure 3 (A–C): Serial growth measurements show progressive recovery from initial growth failure following initiation of oral sulfonylurea therapy, with sustained normalisation over 24 months.

Both weight and length/height were affected at presentation, with early growth faltering within the first 7–8 months, followed by steady catch-up growth; weight showed mild intermittent declines during intercurrent illnesses, while length/height demonstrated gradual improvement with clear catch-up by 24 months. MUAC steadily improved throughout, reflecting restoration of normal nutritional status.

Similar diagnostic dilemmas have been reported in Nigeria and other African settings, where infants presenting with diabetic ketoacidosis were initially managed as sepsis before hyperglycaemia was recognised. [1, 26, 31, 32] In a report from East Africa, Nyangabyaki-Twesigye *et al.*[14] described delayed recognition

of neonatal diabetes due to overlapping clinical features with infection, further emphasising the need for a high index of suspicion in such settings. In contrast, reports from high-income countries often demonstrate earlier diagnosis, largely due to routine metabolic screening and better access to specialised investigations, highlighting disparities in diagnostic timelines and outcomes.[5, 10, 18, 33-35]

In the index case, clinical vigilance and simple bedside investigations were pivotal to diagnosis. Random blood glucose testing and urine dipstick analysis enabled early identification of diabetic ketoacidosis and timely intervention. This underscores the importance of routine glucose assessment in critically ill infants, particularly those presenting with dehydration, altered consciousness, poor feeding, or sepsis-like features. Such low-cost tools can facilitate early detection and prevent delays that may worsen the outcomes.[8, 10]

The concurrent presence of glycosuria and significant ketonuria pointed towards an underlying state of insulin deficiency rather than a transient physiological response to illness. Although stress hyperglycaemia may occur in acute settings, it is seldom associated with marked ketone production, and the overall biochemical profile in this case therefore raised concern for a primary disorder of glucose metabolism. While diabetes mellitus with ketosis was considered, the age at presentation (11 weeks) makes classical Type 1 diabetes mellitus unlikely, given that autoimmune-mediated beta-cell destruction is rare at this stage of infancy. Instead, the early onset of symptoms is more suggestive of neonatal or monogenic forms of diabetes. On this basis, a working diagnosis of diabetes mellitus with ketosis, likely of non-autoimmune origin, was made, guiding the decision to initiate appropriate metabolic stabilisation alongside further evaluation to establish the underlying aetiology.[26, 31, 34]

Importantly, the persistence of marked hyperglycaemia beyond what is typically expected in stress-related responses to infection, together with significant ketonuria and metabolic acidosis, provided useful clinical clues that differentiated NDM from transient stress hyperglycaemia. This distinction is critical, as stress-induced hyperglycaemia is usually mild, transient, and resolves with treatment of the underlying illness, whereas NDM requires sustained glucose-lowering therapy. This case, therefore, provides practical bedside guidance for clinicians in similar settings on when to move beyond an initial assumption of sepsis and actively consider an underlying metabolic disorder.[32, 36, 37]

Building on the initial working diagnosis of diabetes mellitus with ketosis in early infancy, the diagnosis of neonatal diabetes mellitus (NDM) was supported by the clinical course and therapeutic response. The persistence of marked hyperglycaemia beyond the acute phase, despite appropriate resuscitation and treatment of sepsis, reinforced the likelihood of an underlying metabolic disorder rather than transient stress hyperglycaemia. In the absence of genetic testing, diagnosis was based on clinical and biochemical features, including early age at presentation (11 weeks), severity of metabolic derangement, and lack of features suggestive of autoimmune diabetes, all consistent with a monogenic aetiology. Importantly, the successful transition from insulin to oral glibenclamide with sustained glycaemic control provided functional evidence of sulfonylurea-responsive neonatal diabetes. Collectively, these findings support a diagnosis of neonatal diabetes mellitus presenting with diabetic ketoacidosis, likely of non-autoimmune origin and precipitated by intercurrent sepsis, reflecting a pragmatic, response-based diagnostic approach in a resource-constrained setting.[38]

The favourable outcome in this instance demonstrates that effective management of NDM is achievable in resource-constrained

settings. Careful insulin therapy during the acute phase, followed by a structured transition to oral glibenclamide, resulted in sustained glycaemic control and progressive catch-up growth during follow-up care. This supports the role of sulfonylureas as a practical long-term option in selected cases, even in the absence of molecular confirmation, and highlights the importance of structured follow-up in maintaining stable outcomes.[20, 39]

Following successful stabilisation and transition to oral sulfonylurea therapy before discharge, the child has been maintained on regular follow-up for two years with sustained clinical stability. Glycaemic control has remained consistently within recommended paediatric target ranges (pre-prandial 4.0–8.0 mmol/L; post-prandial 4.0–10.0 mmol/L) on oral glibenclamide, with no documented episodes of hypoglycaemia or recurrent ketoacidosis. The sustained normal growth observed in the index child while on follow-up care, as demonstrated in the growth chart, further supports the effectiveness of this comprehensive management approach, contrasting with reports where delayed diagnosis was associated with poor growth outcomes. This favourable trajectory reinforces the importance of early recognition, appropriate therapy, and continuous follow-up in optimising outcomes in neonatal diabetes.[3, 20, 28, 40-42]

Comparable outcomes have been documented globally, particularly in patients with KATP channel mutations, where transition from insulin to sulfonylurea therapy has resulted in improved glycaemic control and quality of life.[15, 20, 25] However, unlike many of these reports where genetic confirmation guided therapy, our approach relied on a carefully monitored therapeutic trial, with the child's sustained response further supporting the clinical diagnosis. Similar pragmatic approaches have been described in other resource-constrained settings, where access to genetic testing remains limited.[20, 43] This further underscores the relevance of therapeutic response as a functional diagnostic tool in such environments.

The index case represents the first documented report from our centre and contributes to the limited Nigerian and African data on NDM. It underscores the need to improve awareness, strengthen basic diagnostic capacity, and integrate routine metabolic screening into paediatric emergency care. Furthermore, this case adds to the growing body of African literature highlighting the dual burden of infectious and metabolic diseases and reinforces the importance of integrating simple metabolic screening into standard paediatric emergency protocols to reduce missed or delayed diagnoses.[1, 14] In practical terms, routine early glucose testing in all critically ill infants may represent a low-cost, high-impact intervention in such settings.

Caregiver education formed an integral component of long-term management in the present case. Parents were counselled on the chronic nature of NDM, its likely genetic basis, and the importance of future genetic evaluation where feasible. This facilitated informed participation in care and supported adherence to treatment and follow-up.[44, 45] In our case, sustained caregiver engagement from admission through follow-up played a critical role in maintaining treatment adherence and achieving favourable long-term outcomes.[45-47]

Sustained caregiver engagement throughout the course of treatment contributed significantly to the favourable outcome observed in this patient. Following structured education and counselling, the caregivers demonstrated good adherence to medication administration, home glucose monitoring, and scheduled follow-up visits, thereby facilitating early recognition of illness and maintenance of glycaemic stability. Such active participation is particularly important in chronic paediatric conditions requiring long-term monitoring and treatment adherence. In addition, efforts are ongoing to

pursue genetic testing through available local opportunities and potential collaboration with external laboratories. Although financial and logistical constraints continue to limit access to molecular diagnostics in many low-resource settings, eventual genetic confirmation remains important for definitive subtype classification, prognostication, and family counselling.[12-14, 38, 44, 45]

The sustained normal growth observed in this child over follow-up, as demonstrated in the growth chart, further supports the effectiveness of this comprehensive management approach, contrasting with reports where delayed diagnosis was associated with poor growth outcomes. This favourable trajectory reinforces the importance of early recognition, appropriate therapy, and continuous follow-up in optimising outcomes in neonatal diabetes. [15, 22, 39, 48, 49]

Limitations

This report is limited by the absence of genetic testing, which precluded definitive molecular confirmation and subtype classification of neonatal diabetes mellitus.

Conclusion

This case highlights the diagnostic challenge encountered when neonatal diabetes mellitus presents with diabetic ketoacidosis in early infancy, where clinical features may closely mimic severe sepsis and thus delay recognition. It underscores the importance of routine glucose assessment in critically ill infants and the need to consider underlying metabolic disorders when hyperglycaemia is persistent and disproportionate to illness severity.

Acknowledgement: The authors acknowledge the contributions of the healthcare team involved in the care of the patient and the cooperation of the patient's family throughout the course of management and follow-up.

Authors' Contributions: SA and OPA conceived the research. SA did the literature review. All the authors participated in data curation and interpretation. SA drafted the manuscript while OPA, EEC, AU1 and AU2 revised the draft for sound intellectual content. All the authors approved the final version of the manuscript.

Consent for publication: Written informed consent was obtained from the infant's parent for the use of his data in this case report. All identifying parts of the data have been anonymised to ensure confidentiality.

Conflicts of Interest: None declared.

Financial support: The authors did not receive any financial support for this research or for the preparation of the manuscript.

Accepted for publication: 18th June 2026.

References

1. Mande BG, Batina AS, Alworong'a OJ, Ngbonda DN. Neonatal Diabetes Mellitus: A Case Report and Literature Review. *Asian J Res Rep Endocrinol* 2018;1(1):28-31
2. Attia NA. Neonatal Diabetes Mellitus: Clinical and Genetic Approach. *Glob Adv Res J Med Med Sci* 2018;7(1):1-9.
3. Beltrand J, Busiah K, Vaivre-Douret L, Fauret AL, Berdugo M, Cavé H, *et al.* Neonatal Diabetes Mellitus. *Front Pediatr* 2020;8:540718. https://doi.org/10.3389/fped.2020.18.
4. Lemelman MB, Letourneau LR, Greely SAW. Neonatal diabetes mellitus: an update on diagnosis and management. *Clin Perinatol* 2018;45:41-59. https://doi.org/10.1016/j.clp.2017.10.006.
5. Woon Y, Card C, Holcroft L, O'Connell S, Power C, Somers E, *et al.* Neonatal Diabetes in Ireland Over the Past 19 Years: Clinical Presentation, Management, Genetics, And Outcomes. *Horm Res Paediatr* 2026;5(6):24-25. https://doi.org/10.1159/000550794.
6. Dermitzaki N, Serbis A, Baltogianni M, Giapros V. The Molecular Basis of Neonatal Diabetes Mellitus and Transient

Hyperglycemia in the Neonate. *Appl Sci* 2026;16(6):2649. https://doi.org/10.3390/app16062649.

7. Le Bourgeois F, Beltrand J, Baz B, Julla JB, Riveline JP, Simon A, *et al.* Long-Term Metabolic And Socioeducational Outcomes Of Transient Neonatal Diabetes: A Longitudinal and Cross-Sectional Study. *Diabetes Care* 2020;43(5):1191-99. https://doi.org/10.2337/dc19-0324.
8. Hattersley AT, Greeley SAW, Polak M, Rubio-Cabezas O, Njølstad PR, Mlynarski W, *et al.* ISPAD Clinical Practice Consensus Guidelines 2018: The Diagnosis and Management of Monogenic Diabetes in Children and Adolescents. *Pediatr Diabetes* 2018;19 Suppl 27:47-63. https://doi.org/10.1111/pedi.12772.
9. Balboa D, Saarimäki-Vire J, Borshagovski D, Survila M, Lindholm P, Galli E, *et al.* Insulin Mutations Impair Beta-Cell Development in a Patient-Derived IPSC Model of Neonatal Diabetes. *Elife* 2018;7:e38519. https://doi.org/10.7554/eLife.19.
10. Shah B, Kataria A, Gopi RP, Mally P. Neonatal Diabetes Mellitus: Current Perspective. *Res Rep Neonatol* 2014;4:55-62. https://doi.org/10.2147/RRN.S38206.
11. Raghav A, Ahmad J, Kaul Ak, Singh P, Mamtani H, Andrabi SM, *et al.* Neonatal Diabetes Mellitus: A Tale Still to Resolve. *Curr Res Diabetes Obes J* 2017;4(4):23-7. https://doi.org/10.19080/Crdoj.2017.04.555643.
12. Okolo AA, Omene JA, Odita JC. Neonatal Diabetes Mellitus and Hypertrophic Pyloric Stenosis: A Case Report. *Niger J Paediatr* 1983;10(2):1-5.
13. Obasa TO, Adesiyun OO, Mokuolu OA. Transient Neonatal Diabetes or Neonatal Hyperglycaemia: A Case Report. *Niger J Paediatr* 2013;40(2):192-4. https://doi.org/10.4314/njp.v40i2.18.
14. Nyangabyaki-Twesigye C, Muhame MR, Bahendeka S. Permanent Neonatal Diabetes Mellitus in East Africa. *Afr Health Sci* 2015;15(4):1339-41. https://doi.org/10.4314/ahs.v15i4.37.
15. Ben Hamouda H, Chioukh F, Boussetta B, Mahjoub B, Ayadi A. Long-Term Follow-Up of Permanent Neonatal Diabetes. *Ann Endocrinol (Paris)* 2010;71(4):314-9.
16. Mackay D J, Temple I K. Transitory and Permanent Forms of Neonatal Diabetes Mellitus: Current Insights. *Front Endocrinol* 2018;9:273
17. Mohammad Golshan-Tafti, Seyed Alireza Dastgheib, Reza Bahrami, Maryam Yeganegi, Maryam Aghasipour, Zahra Marzbanrad, *et al.* Genetic Underpinnings of Neonatal Diabetes: A Review of Current Research. *Egyptian J Med Human Genet* 2024;25:119-27.
18. Taberner P, Flanagan SE, Mackay DJ, Ellard S, Taverna MJ, M F. Clinical and Genetic Features of Argentinian Children with Diabetes-Onset Before 12 Months of Age. *Diabetes Res Clin Pract* 2016;117:104-10. https://doi.org/10.1016/j.diabres.2016.04.005.
19. Bahendeka S, Wesonga R, Mutungi G. Diabetes in Sub-Saharan Africa: Burden and Challenges. *Afr Health Sci* 2016;16(4):1037–45. https://doi.org/10.4314/ahs.v16i4.36.
20. Carmody D, Bell CD, Hwang JL, Dickens JT, Sima DI, Felipe DL, *et al.* Sulfonylurea Treatment Before Genetic Testing in Neonatal Diabetes. *J Clin Endocrinol Metab* 2014;99(8):2709-14. https://doi.org/10.1210/jc.2014-494.
21. Hajji S, Aljenaee K, Garrahy A, Byrne M. Successful Transition from Insulin to Sulfonylurea, on Second Attempt, in a 24-Year-Old Female with Neonatal Diabetes Secondary to KCNJ11 Gene Mutation. *BMJ Case Rep* 2021;14(4):e239973. https://doi.org/10.1136/bcr-2020-73.
22. Garcin L, Kariyawasam D, Busiah K, Fauret-Amsellem AL, Le Bourgeois F, Vaivre-Douret L, *et al.* Successful Off-Label Sulfonylurea Treatment of Neonatal Diabetes Mellitus Due To Chromosome 6 Abnormalities. *Pediatr Diabetes* 2018;19(4):663-9. https://doi.org/10.1111/pedi.12635.
23. Yamazaki M, Sugie H, Oguma M, Yorifuji T, Tajima T, Yamagata T. Sulfonylurea Treatment in an Infant with Transient Neonatal Diabetes Mellitus Caused by an ABCC8 Mutation. *Clin Pediatr Endocrinol*

2017;26(3):165-9. https://doi.org/10.1297/cpe.26.165.

24. Li X, Xu A, Sheng H, Ting TH, Mao X, Huang X, *et al*. Early Transition from Insulin to Sulfonylureas. Pediatr Diabetes 2018;19(2):251-8. https://doi.org/10.1111/pedi.12560.
25. Beltrand J, Baptiste A, Busiah K, Bouazza N, Godot C, Boucheron A, *et al.* Glibenclamide Oral Suspension: Suitable and Effective in Patients with Neonatal Diabetes. *Pediatr Diabetes* 2019;20(2):246-54. https://doi.org/10.1111/pedi.12823.
26. Fredrick F, Sawe H, Muze K, Mally D, Majaliwa E. A Seven-Week-Old Baby with Diabetic Ketoacidosis. *Clin Case Rep* 2016;4(2):147-50. https://doi.org/10.1002/ccr3.472.
27. Mancioppi V. Case Report: Better Late Than Never, But Sooner Is Better: Switch From CSII To Sulfonylureas in Two Patients with Neonatal Diabetes Due To KCNJ11 Variants. *Front Endocrinol* 2023;14:1143736. https://doi.org/10.3389/fendo.2023.36.
28. Ilkhanipoor H, Karamizadeh Z. Changing Treatment From Insulin To Glibenclamide In Neonatal Diabetes. *Int J Prev Med* 2013;4(9):1078-81
29. Wright M, Body S, Lutman D. Management Of Diabetic Ketoacidosis In Children. *BJA Educ* 2023;23(9):364-70. https://doi.org/10.1016/j.bjae.2023.03.005
30. Grant P. Management Of Diabetes In Resource-Poor Settings. *Clin Med* 2013;13(1):27-31. https://doi.org/10.7861/clinmedicine.13-1-27
31. Abacı A, Razi CH, Özdemir O, Hızlı S, Kıslal F, Argas PI, *et al.* Neonatal Diabetes Mimicking Sepsis. *J Clin Res Pediatr Endocrinol* 2010;2(3):131-3. https://doi.org/10.4274/jcrpe.v213.131.
32. Akinola I J, Odusote O A, Oduwole A O. Neonatal Diabetic Ketoacidosis in a Nigeria Infant: A Case Report. *Niger Quart J Hosp Med* 2015;25(2):139-41.
33. Greeley SAW, Tucker SE, Naylor RE, Bell GI, Philipson LH. Neonatal Diabetes Mellitus: A Model for Personalized Medicine. *Trends Endocrinol Metab* 2010;21(8):464–72.
34. Sood S, Landreth H, Bustinza J, Chalmers L, Thukaram R. Neonatal Diabetes: Case Report Of A 9-Week-Old Presenting Diabetic Ketoacidosis Due To An Activating Abcc8 Gene Mutation. *J Investig Med High Impact Case Rep* 2017;5(1):2324709617698718. https://doi.org/10.1177/18.
35. Yang W, Sang Y, Liu M, Gu Y, Zhu C, Ni G. Clinical Analysis on 10 Patients with Permanent Neonatal Diabetes Mellitus. *Endocrinol Metab Syndr* 2011; S5-004. https://doi.org/10.4172/2161-1017.S5-004.
36. Oduwole AO, Ibitoye PK, Fajolu IB. Pattern of Childhood Diabetes Mellitus in Lagos, Nigeria. *Niger J Paediatr* 2012;39(4):155–59.
37. Akanji AO, Olatunji PO. Childhood Diabetes Mellitus in Sub-Saharan Africa: Challenges and Prospects. *Afr J Med Med Sci* 2013;42(3):219–28.
38. Habeb AM, Deeb A, Elbarbary N, Beshyah SA. Diagnosis and Management of Neonatal Diabetes Mellitus: A Survey Of Physicians' Perceptions and Practices in ASPED Countries. *Diabetes Res Clin Pract* 2020;159:107975e. https://doi.org/:10.1016/j.diabres.2019.75.
39. Bowman P, Sulen Å, Barbetti F, Beltrand J, Svalastoga P, Codner E, *et al.* Effectiveness and Safety of Long-Term Treatment with Sulfonylureas in Patients with Neonatal Diabetes Due To KCNJ11 Mutations: An International Cohort Study. *Lancet Diabetes Endocrinol* 2018;6(9):637-46. https://doi.org/10.1016/S2213-8587(18)30106-2.
40. Rabbone I, Barbetti F, Gentilella R, Mossetto G, Bonfanti R, Maffeis C, *et al.* Insulin Therapy in Neonatal Diabetes Mellitus: A Review of the Literature. *Diabetes Res Clin Pract* 2017;129:126-35. https://doi.org/10.1016/j.diabres.2017.04.007.
41. Garcin L, Mericq V, Fauret-Amsellem AL, Cavé H, Polak M, Beltrand J. Neonatal Diabetes Due To Potassium Channel Mutation: Response to Sulfonylurea

According to the Genotype. *Pediatr Diabetes* 2020;21(6):932-41. https://doi.org/10.1111/pedi.13041.

42. Pearson ER, Flechtner I, Njølstad PR, Malecki MT, Flanagan SE, Larkin B, *et al.* Switching from Insulin to Sulfonylureas in KIRA6.2 Mutations. *N Engl J Med* 2006;355(5):467-77. https://doi.org/10.1056/NEJMoa061759.
43. Babiker T, Shepherd MH, Hattersley AT. Continue with Long-Term Sulfonylureas in Patients with Mutations in the KCNJ11 Gene when there is evidence of response, even if Insulin Treatment is Still Required. *Diabetes Res Clin Pract* 2013;100(3):e63.
44. Shepherd M, Knight BA, Laskey K, McDonald TJ. Parental Experiences of a Diagnosis of Neonatal Diabetes and Perceptions of Newborn Screening for Glucose: A Qualitative Study. *BMJ Open* 2020;10(11):e037312.
45. Dhada BL, Blackbeard DR. Caregivers of Children with Diabetes Mellitus: Challenges of Caring for and Perceptions of Consultations in a South African Public Sector Context. *South Afr Fam Pract* 2019;61(4):1606491e.
46. Ola H, Jafar AA, Diana HA. Review of Diabetes Education Intervention Targeting Parent/Caregiver Of Children With Type 1 Diabetes. *Frontiers Nursing* 2026;13(1):15-23. https://doi.org/10.2478/FON-026-0003
47. Melike TB, Hatice D, Birsel M, Funda Ö, Beray SE. Parenting a Child with Type 1 Diabetes: Challenges and the Role of Social Support. *J Pediatr Nursing* 2025;85:191-99. https://doi.org/10.1016/j.pedn.2025.07.024
48. Babiker T, Shepherd MH, Hattersley AT. Continue with long-term sulfonylureas in patients with mutations in the Kcnj11 Gene when there is evidence of response, even if Insulin treatment is still required. *Diabetes Res Clin Pract* 2013;100(3):e63.
49. Bremer AA, Ranadive S, Lustig RH. Outpatient Transition To Glyburide In Neonatal Diabetes. *Pediatr Diabetes* 2008;9(3):236-39. https://doi.org/10.1111/j.399-5448.2007.00316.x.