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A STUDENT PERSPECTIVE: NEWBORN SCREENING FOR CONGENITAL HYPOTHYROIDISM (CH): CASE SCENARIOS FOR PREVENTION OF DISABILITY

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ABSTRACT:
Newborn screening (NBS) is a preventive public health program aimed at identifying congenital metabolic disorders soon after birth, before clinical symptoms appear. The Congenital hypothyroidism (CH) the most common NBS disorder used globally to prevent childhood disability. The CH is the significant preventable cause of intellectual disability worldwide, affecting approximately 1 in 2,000–3,000 live births. Thyroid hormone is essential for normal brain development. Delayed diagnosis and treatment of CH can lead to irreversible neurodevelopmental impairment. The CH also fulfils NBS criteria and hence it is considered as the ideal NBS candidate. The efficiency of NBS program with CH has been proved in many countries. However, India does not have NBS in its mandatory health policy and the risk of childhood disabilities/death therefore is higher due to CH.
The CH cases encountered by the student during her internship are reported here to emphasize the significance of NBS in India to prevent childhood disabilities & the need of saving babies across the country due to CH, thereby help to reduce the national health burden. The case scenarios were developed as composite evidence-based examples from the published literature.
The aim is to summarize current evidence on newborn screening (NBS) strategies for CH and their impact on neurodevelopmental outcomes. It also highlights clinical decision-making through three case scenarios illustrating the diverse presentations and outcomes of CH. The methodology used was a narrative literature review using PubMed/MEDLINE, EMBASE and the Cochrane Library. Publications from 1974 to 2025 were reviewed, with emphasis on systematic reviews including major guidelines, like the American Academy of Pediatrics (AAP, 2023) and European Society for Paediatric Endocrinology (ESPE, 2014).
It is evident that despite 50 years of established NBS programs, only 29.6% of global births were covered by NBS for CH by 2024. The case scenarios further demonstrate that optimal outcomes depends on early detection within first 14 days of life, confirmatory testing, timely recall systems, appropriate therapy, consistent follow-up, and recognition of high-risk groups such as preterm infants. It is concluded that universal newborn screening for CH is a critical ethical and public health priority, as early diagnosis and treatment substantially reduce preventable intellectual disability and improve long-term neurodevelopmental outcomes.

KEYWORDS: Congenital hypothyroidism, Newborn screening, Neurodevelopment, disability; neonatal TSH, Dried Blood Spot (DBS)

INTRODUCTION

Congenital hypothyroidism (CH) is a condition in which an infant is born with insufficient thyroid hormone production. It may result from abnormal development of the thyroid gland (thyroid dysgenesis), defects in thyroid hormone synthesis (dyshormonogenesis), or dysfunction of the hypothalamic–pituitary axis causing central CH. The CH is among the most common endocrine disorders identified during the neonatal period and remains one of the leading preventable causes of permanent intellectual disability worldwide (1).
Prior to the introduction of organized newborn screening (NBS) programs, untreated CH frequently led to severe neurodevelopmental consequences, historically described as cretinism, characterized by profound cognitive impairment, motor dysfunction, deafness, and reduced survival. A major breakthrough occurred in 1974 when Dussault and colleagues in Quebec, Canada, demonstrated that thyroid-stimulating hormone (TSH) could be reliably measured from dried blood spot (DBS) samples obtained through neonatal heel-prick testing (2, 3). This innovation marked the beginning of modern neonatal screening and preventive endocrinology. Over the following decade, NBS programs for CH became widely implemented across North America, Europe, and several Asian countries, resulting in a dramatic decline in severe CH-associated disability (4).

Incidence: Congenital hypothyroidism (CH) is considered the most frequently occurring endocrine disorder in newborns, with an estimated worldwide incidence ranging from 1 in 2,000 to 1 in 3,000 live births (1). A systematic review and

meta-analysis published by Liu et al. in 2023, evaluated studies conducted between 1969 and 2020 and estimated the global prevalence to be nearly 1 in 2,500 births, while also highlighting marked geographic variability. (5). Regions such as South Asia, the Middle East, and North Africa have reported comparatively higher rates, ranging from 1 in 1,000 to 1 in 1,500 live births (6). These increased rates are thought to be associated with factors such as consanguineous marriages, iodine imbalance, and a greater burden of dyshormonogenesis. A clear female predominance, approximately two females for every affected male, has been repeatedly documented in thyroid dysgenesis, although the precise biological explanation for this sex difference is still unclear (1).

In the Indian population, CH appears to occur more frequently than in many Western countries. A systematic review and meta-analysis published in The Lancet Regional Health (2022) estimated the prevalence in India to be approximately 1 in 1,706 live births, with a comparatively greater contribution from thyroid dyshormonogenesis than that reported in Western populations (7). The ICMR pilot study in 1 lakh babies from India revealed 1: 900 incidence in newborns (8).
Aetiology: Most cases of CH are classified as primary CH, meaning the disorder originates from intrinsic abnormalities of the thyroid gland. Thyroid dysgenesis which includes thyroid agenesis, ectopic thyroid tissue, and thyroid hypoplasia is responsible for nearly 80% of permanent primary CH cases and usually occurs sporadically. Genetic mutations involving transcription factors, as well as mutations in the thyroid-stimulating hormone receptor (TSHR) gene, have been identified in approximately 2–5% of dysgenesis cases (9).
Another important cause is dyshormonogenesis, where the thyroid gland is anatomically present but unable to synthesize thyroid hormones effectively. This form contributes to nearly 15–20% of permanent CH cases again due to genetic mutation (9). Central CH, resulting from hypothalamic or pituitary dysfunction, is uncommon and may remain undetected in NBS programs which rely solely on TSH-based screening. Temporary or transient CH can occur secondary to maternal influences such as transplacental passage of antithyroid drugs, maternal TSH-receptor blocking antibodies, or abnormalities in iodine exposure, either deficiency or excess (10).
Several factors are associated with a higher likelihood of severe disease or delayed recognition of CH. One of the most important is the absence of a newborn screening program in the region where the infant is born. Premature infants, (born before 32 weeks of gestation) are at increased risk because of an attenuated neonatal TSH surge (10). Similarly, very low birth weight infants weighing less than 1500 grams may have delayed biochemical abnormalities (11). Monozygotic twin pregnancies can also complicate detection due to dilution effects on TSH levels between twins. Additional risk groups include neonates with trisomy 21 and critically ill infants requiring neonatal intensive care unit (NICU) admission, where heel-prick sampling may be delayed (12).

Pathophysiology: Thyroid Hormones and Neurodevelopment
The role of thyroid hormone in brain maturation follows a predictable developmental sequence. During early fetal life, particularly throughout the first trimester, the fetus is entirely dependent on maternal thyroxine (T4) transferred across the placenta because the fetal thyroid gland does not begin functioning until around 10–12 weeks of gestation. As pregnancy progresses, endogenous fetal thyroid hormone production gradually increases and becomes the primary source by mid-gestation. After birth, thyroid hormones become critically important for neurological development, especially during the first 6–12 months of life, a period marked by rapid myelination, cerebellar maturation, hippocampal development, and refinement of auditory neural pathways. This narrow developmental window explains why early recognition and immediate treatment of congenital hypothyroidism (CH) are essential for preserving normal neurodevelopment (13).
When CH remains untreated or treatment is delayed, a wide range of neurological complications may occur. Cognitive impairment is among the most significant consequences, with lower intelligence quotient (IQ) scores strongly associated with both the severity and duration of thyroid hormone deficiency. Affected children may also demonstrate difficulties in memory, learning, attention, and psychomotor performance (14). Sensorineural hearing loss can develop because of impaired maturation of the cochlea and auditory pathways. In severe longstanding cases, additional manifestations such as spasticity, cerebellar dysfunction, and seizures may be observed. Importantly, the classic physical signs of CH including persistent neonatal jaundice, enlarged posterior fontanelle, macroglossia, hypotonia (Fig.1), constipation, hypothermia, and a hoarse cry often become apparent only several weeks or months after birth. This delay in clinical presentation highlights the indispensable role of biochemical newborn screening in detecting the disorder before irreversible neurological injury occurs (1, 5, 14 & 15).

Figure 1: The 3 months old baby with physical features of CH such as hyperglossia, hepatomegaly, coarse facial features, & lethargy

Newborn Screening: Scientific Basis, Methods, and Protocols :
Wilson-Jungner Criteria are followed for NBS screening & CH satisfies all ten Wilson-Jungner criteria for population-

based screening as below (6 & 16).

1. An accepted treatment is available (levothyroxine).
2. Facilities for diagnosis and treatment are available.
3. A recognisable latent or early symptomatic stage exists.
4. A suitable and acceptable screening test is available (DBS for TSH/T4 test).
5. The natural history of the condition is well understood.
6. An agreed policy on who to treat exists (society guidelines: AAP 2023, ESPE 2014).
7. The cost of case finding is economically balanced with overall expenditure on medical care.
8. Case-finding is a continuing process, not a once-and-for-all project.
9. The test is acceptable to the population.

Specimen Collection (Dried Blood Spot): The standard NBS specimen is a dried blood spot (DBS) obtained by heel-prick, collected after 48 hours of life (Fig. 2). The TSH is first measured from DBS using universally accepted ELISA (Enzyme Linked Immuno Assay) method (12). Cut-off thresholds vary by laboratory, gestational age, and the analytical method used. The following evidence-based thresholds from the AAP (2023), ESPE (2014), and Medscape clinical guidelines are applied in practice (Table-1).

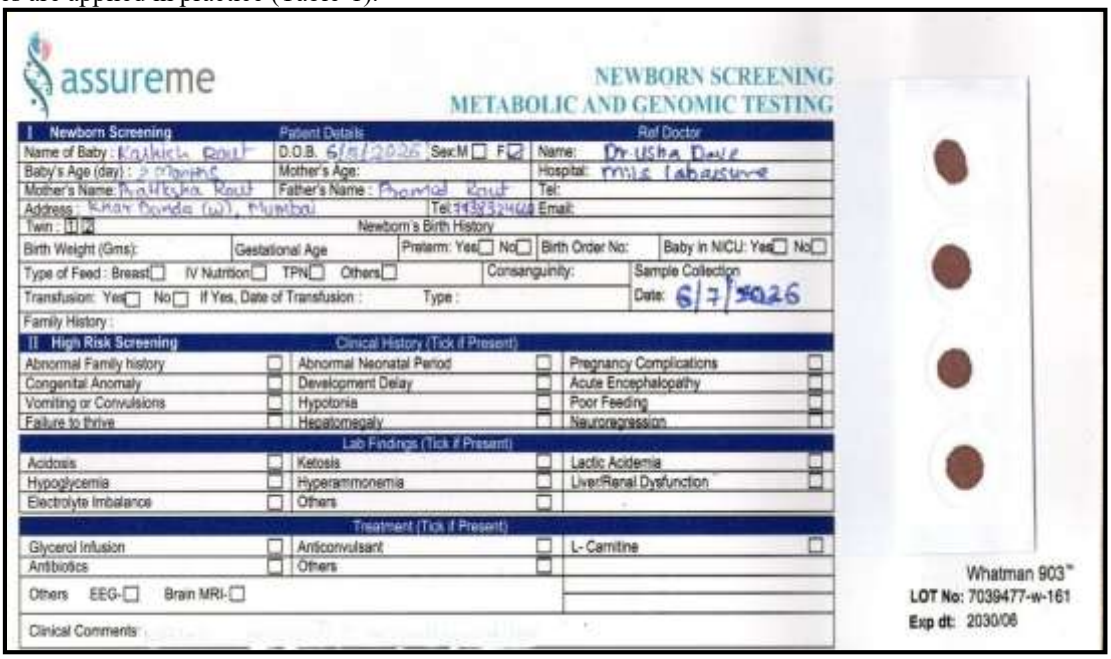

Figure 2: Dried blood spot card showing details of the infant for CH test

Note the 4 dried blood spots on filter paper circles sent for testing.

DBS TSH (mU/L)	Serum TSH (mU/L)	Free T4	Recommended Action
> 40	Any elevated	Low	Immediate LT4; no delay for
20–40	> 20	Low or	Confirmatory serum testing; treat if TSH
6–20	> 6 beyond day 21	Low	Initiate LT4; reassess at 3 years
< 20	Normal	Normal	Observe; repeat screen per protocol

Table 1. Action Thresholds Based on DBS TSH and Confirmatory Serum Values

(DBS: dried blood spot; LT4: levothyroxine; TSH values in mU/L. Based on AAP 2023 and ESPE 2014 guidelines.)

All screen-positive infants require confirmatory serum testing: Free T4 (FT4), total T4 (TT4), and TSH, using venous blood. Additional investigations helpful in determining CH aetiology and informing prognosis include (12):

- Thyroid ultrasound: assesses gland presence, size, and location; detects ectopia, hypoplasia, or agenesis.
- Radionuclide thyroid scintigraphy (99mTc or 123I): identifies functional thyroid tissue and ectopic location with high sensitivity; may be omitted if ultrasound is diagnostic.
- Thyroglobulin (Tg): undetectable in athyreosis; elevated in dyshormonogenesis.
- Thyroid autoantibodies (TPO-Ab, TSH-R blocking Ab): identifies maternal autoimmune aetiology for transient CH.
- Genetic testing: indicated for suspected dyshormonogenesis or familial CH; guides recurrence risk counselling.

Treatment: Levothyroxine (LT4) is the sole recommended treatment for CH. The AAP (2023) (12) and ESPE (2014) (17) guidelines agree on a starting dose of 10–15 mcg/kg/day administered orally, with the higher end of this range (12–

15 mcg/kg) recommended for infants with severe biochemical disease (very low FT4, TSH >40 mU/L) or confirmed athyreosis (12). Treatment should begin as soon as possible and no later than 2 weeks after birth; for neonates with DBS TSH >40 mU/L, LT4 should be initiated immediately after confirmatory blood samples are drawn, without awaiting results (15 & 18).
Delaying LT4 initiation beyond 6 weeks of life is associated with a substantial, documented risk of impaired cognitive development (12 & 19).

Case Scenarios: Clinical Spectrum and the Impact of Screening
The following 3 cases are evidenced composites derived from published clinical literature.

CASE 1: Optimal NBS Outcome in Term Neonate with Thyroid Ectopia
Presentation: A female neonate with birth weight 3.2 kg, 39 weeks gestation, was born to a mother in a tertiary referral centre with an established NBS program. DBS was collected at 54 hours of life. NBS returned a TSH of 94 mU/L (laboratory cut-off: 20 mU/L). Parents were recalled at day 5 for repeat sample.
The confirmatory serum TSH 112 mU/L; FT4 5.2 pmol/L (severely low; normal >12 pmol/L). Thyroglobulin 6 ng/mL (low-normal). Thyroid ultrasound showed no identifiable thyroid tissue in normal position and reported as lingual thyroid ectopia. The treatment -LT4 was initiated on day 9 at 13 mcg/kg/day. TSH normalised to 1.8 mU/L by 6 weeks; FT4 within upper half of normal range. Monitoring every 4–6 weeks in infancy. Dose adjusted with weight gain. At 6 months, baby achieved normal developmental milestones.
Evidence Base: Albert et al, 2013 demonstrated that high-dose LT4 with close monitoring in screen-detected CH is associated with normal intellectual and motor development, consistent with this case (20).
Clinical Outcome: At 4-year neurodevelopmental assessment revealed age-appropriate language, motor, and social development and disability entirely prevented by timely NBS and treatment.

CASE 2: No NBS Available, Delayed Diagnosis, Severe CH with Permanent Disability
Presentation: A male infant was born at home in a rural district without access to any NBS program. He presented to a district paediatric facility at 5 months with severe hypotonia, macroglossia, umbilical hernia, hoarse cry, prolonged jaundice, marked constipation, and delayed motor developmental milestones (no social smile, no head control at 5 months). The clinical presentation was classic for severe untreated CH, yet had been attributed to 'weakness' and nutritional deficiency by local practitioners. Serum TSH >500 mU/L; FT4 undetectable. Bone age radiograph: severely retarded (consistent with 0–1 months). Brain MRI: diffuse hypomyelination of white matter tracts; thin corpus callosum. Audiological assessment: bilateral moderate-to-severe sensorineural hearing loss.
The LT4 was initiated at 12 mcg/kg/day; dose up-titrated over 4 weeks. TSH declined slowly, reaching 8 mU/L at 3 months post-initiation. Early intervention commenced: physiotherapy, hearing aids, and special education.
Evidence Base: Oron et al, 2015 demonstrated that time to treatment initiation and severity of biochemical disease at diagnosis are the strongest predictors of early neurodevelopmental outcome (15). Treatment after 3 months does not reverse established neurological injury from severe CH.
Clinical Outcome: At 5-year follow-up: IQ 48 (severe intellectual disability); non-verbal communication primarily; dependent for activities of daily living; bilateral hearing aids; physiotherapy ongoing. The child's disability was entirely preventable with NBS and treatment before 2 weeks of life.

CASE 3: Missed Diagnosis in Preterm Infant, Protocol Gap and Repeat Screening Failure Presentation: A male infant born at 28+3 weeks gestation (930g birth wt.) was admitted to the NICU. Heel-prick DBS was obtained at 72 hours; TSH returned at 5.8 mU/L — within the
laboratory cut-off (< 20 mU/L), reflecting the blunted TSH surge and hypothalamic-pituitary
immaturity characteristic of extremely preterm neonates. A second NBS (recommended per AAP protocol for neonates <32 weeks) was documented as 'planned' but was not performed before NICU discharge at 36 weeks corrected age. No community follow-up NBS occurred.at re-referral: At 11 weeks chronological age (3 weeks corrected age), routine thyroid function ordered by a community paediatrician revealed TSH 84 mU/L; FT4 7.1 pmol/L. Thyroid ultrasound showed a small but present gland in situ, suggesting possible dyshormonogenesis or delayed TSH rise. The missed second NBS was the critical system failure. The LT4 was initiated at 10 mcg/kg/day. TSH normalised by week 16.
Evidence base: A study by Nolan et al in 2024 found that 14.6% of preterm infants born VPT (very preterm) or VLBW had abnormal thyroid function on repeat screening, which was missed by the initial NBS (11). Klosinska et al, in 2022 reviewed the specific challenges of CH diagnosis in preterm neonates, noting that hypothyroxinemia may be delayed and that single- point TSH screening is insufficient (10).
Clinical Outcome: At 2 years corrected age: mild expressive language delay; normal motor development; receiving speech and language therapy. Developmental age: 86 (borderline low). Outcome was suboptimal but better than untreated severe CH, due to relatively lower degree of biochemical hypothyroidism. Failure of the repeat NBS protocol was the avoidable system error.

DISCUSSION
The three case scenarios presented above (Table 2) encapsulate the central argument for universal NBS. The outcome difference between Case 1 (normal IQ, disability prevented) and Case 2 (severe intellectual disability, irreversible) is not a difference in disease severity alone, but a difference in the presence or absence of a screening infrastructure. This is the

quintessential demonstration of secondary prevention at the population level; intervening before symptoms emerge to prevent irreversible harm.

NBS for CH is considered one of the most cost-effective public health interventions in medicine. Published health economic analyses estimate that for every dollar invested in NBS for CH, between 8 and 12 dollars are saved in downstream disability care, special education, and lost productive years. In the Indian context, the ICMR multicentre pilot study (8) validated this economic argument, finding CH incidence slightly higher than with the global literature and recommending universal NBS as a cost-justified health program (3, 7).

In India, where approximately 27 million births occur annually, the national NBS program remains fragmented. The Indian Society for Pediatric and Adolescent Endocrinology (ISPAE) has issued guidelines recommending universal NBS, and pilot programs across Delhi, Chennai, Kolkata, Mumbai, and Hyderabad validated feasibility, but a government-funded national universal program remains aspirational (7 & 8). Key barriers include laboratory cold-chain infrastructure costs, shortage of paediatric endocrinologists for confirmatory evaluation and follow-up, home birth rates in rural settings, and low health literacy among families (21 & 22).

The Case 3 reflects a documented gap in screening fidelity for preterm neonates. The blunted neonatal TSH surge, relative HPT axis immaturity, and the clinical complexity of the NICU environment all conspire to make a single-point TSH screen inadequate for this population. The AAP 2023 guidelines specifically mandate repeat NBS at 2–4 weeks for all infants born

<32 weeks or with birth weight <1500 gm (12) Nolan et al. in 2024 demonstrated that 14.6% of VPT/VLBW infants had abnormal thyroid function detectable on repeat screening, making protocol adherence non-negotiable (11).

Table 2: Comparative Summary of Three Congenital Hypothyroidism (CH) Cases: Impact of Newborn Screening (NBS):

Parameter	**Case 1** Optimal NBS Outcome — Term Neonate, Thyroid	**Case 2** No NBS Available — Severe, Delayed Diagnosis	**Case 3** Missed Diagnosis in Preterm Infant — Protocol Gap
Setting / Screening Status	Tertiary centre with established NBS program	Home birth, rural district; no NBS program available	NICU; DBS done, but recommended repeat NBS at <32 weeks not performed
Background	Female, term (39 wks), 3.2 kg, vaginal delivery, Apgar 9/10; primigravida mother	Male infant, born at home; no antenatal/perinatal screening access	Male, extreme preterm (28+3 wks, 930 g); admitted to NICU
Age at NBS / Presentation	DBS at 54 h of life; recalled day 5	First presented to a facility at 5 months of age	First DBS at 72 h (false-negative); re- referred at 11 weeks chronological (3 weeks corrected)
Clinical Features	Asymptomatic at screening (typical for screen-detected CH)	Severe hypotonia, Macroglossia, Umbilical hernia, Hoarse cry, Prolonged jaundice, No social smile/head control at 5 month	Initially asymptomatic/preterm-related issues; thyroid dysfunction picked up only on routine community testing
Key Investigations	Confirmatory TSH 112 mU/L; FT4 5.2 pmol/L; Tg 6 ng/mL; USS — no thyroid in normal position; 99mTc scan — lingual ectopic focus	TSH >500 mU/L; FT4 undetectable; bone age retarded (0–1 mo at chronologic 5 mo); MRI — diffuse hypomyelination, thin corpus callosum; bilateral SNHL on audiology	Initial TSH 5.8 mU/L (within cut-off, blunted preterm surge); at re-referral TSH 84 mU/L, FT4 7.1 pmol/L; USS — small gland in situ (? dyshormonogenesis)
Treatment (LT4)	Started day 9, 13 mcg/kg/day; TSH normalised (1.8 mU/L) by 6 weeks	Started at 5 months, 12 mcg/kg/day, up-titrated over 4 weeks; TSH only 8 mU/L by 3 months post-start; multidisciplinary early intervention (physio, OT, hearing	Started at 11 weeks, 10 mcg/kg/day; TSH normalised by week 16
Supporting Evidence	Albert et al., JCEM 2013 — high- dose LT4 with close monitoring in screen-detected CH → normal cognitive/motor outcome	Oron et al., J Pediatr 2015 — time-to- treatment and biochemical severity at diagnosis are the strongest predictors of outcome; injury after 3 months is largely irreversible	Nolan et al., J Pediatr 2024 — 14.6% of VPT/VLBW infants have abnormal thyroid function missed on initial NBS; Klosinska et al., Front Endocrinol 2022 — single-point TSH screening is
Neurodevelopmental Outcome	IQ 103 at 4 years (normal); age- appropriate language, motor, social development	IQ 48 at 5 years (severe intellectual disability); non-verbal; dependent for ADLs; bilateral hearing aids; ongoing	Bayley cognitive composite 86 at 2 years (borderline-low); mild expressive language delay; normal motor development; speech

Key Take-Home Lesson	Timely NBS + early high-dose LT4 + close monitoring → disability fully prevented	Absence of NBS → diagnosis delayed to symptomatic stage → severe, permanent, irreversible disability	A single early TSH is not sufficient in preterm infants; failure to perform the recommended repeat NBS allowed a system-level error that
Reference(s)	Albert S, et al. Levothyroxine dose in congenital hypothyroidism and neurodevelopmental outcome. J Clin Endocrinol Metab. 2013.[12]	Oron T, et al. Effect of age at initiation of treatment on neurodevelopmental outcome in congenital hypothyroidism. J Pediatr. 2015.[13]	Nolan EE, et al. Thyroid dysfunction on repeat newborn screening in very preterm/very low birth weight infants. J Pediatr. 2024.[14] Klosinska M, et al. Congenital hypothyroidism in preterm newborns

Dose and Timing is crucial: The optimal LT4 starting dose remains a subject of ongoing research. Guidelines recommend 10–15 mcg/kg/day, with higher doses for severe disease.
Esposito et al, in 2022 in a prospective multicenter Italian study found that initial doses between 10–12.5 mcg/kg/day and 12.6–15 mcg/kg/day both achieved normal developmental quotients at 24 months and IQ at 48 months, with no significant difference between groups (18). This underscores that beyond pharmacological parameters, social determinants of health significantly influence developmental trajectories.

Recommendation: A few recommendations for effective CH-NBS program for Indian scenario are given below as the outcome of this study-

A) For Health Systems and Policymakers-

- Universal NBS for CH (and CAH, G6PD deficiency) should be mandated and government-funded, prioritising where coverage remains below 10%.
- Point-of-care TSH devices and community health worker-led DBS collection should be piloted and scaled in areas with poor laboratory access.
- National registries for CH should be established to monitor incidence, screening coverage, time-to-treatment, and long-term neurodevelopmental outcomes.

B) For Clinicians & Health providers

- Adhere to NBS guidelines (23): screen between 24–72 hours, initiate LT4 within 14 days, and target FT4 in upper half of normal range.
- Perform repeat NBS for all high-risk neonates (preterm <32 weeks, VLBW, NICU, multiples, trisomy 21) at 2–4 weeks and again at 6–8 weeks if still <36 weeks corrected age.
- Conduct thorough aetiological work-up to distinguish permanent from transient CH, enabling appropriate duration of treatment decisions.
- Provide clear, empathetic parent communication about the screening process, recall, confirmatory testing, and long-term prognosis.

C) For Medical Students and Future Physicians:

- Understand that NBS is not merely a laboratory test. It is a system of care requiring recall protocols, confirmatory pathways, treatment initiation, and longitudinal follow-up.
- Advocate for equitable access to NBS as a health justice issue during training and throughout a career.
- Recognise the social determinants that shape CH outcomes beyond screening status, including family education, economic status, and access to specialist care.

CONCLUSION

Congenital hypothyroidism remains the paradigm of preventable neurodevelopmental disability. The science is settled: early identification through NBS, followed by prompt LT4 therapy, prevents intellectual disability in the vast majority of affected neonates. Yet 70% of the world's newborns are not screened. The contrast between Case 1—an infant who receives NBS and develops normally, and Case 2—an infant born outside a screening system who develops severe intellectual disability—is not a contrast between different diseases or different treatments. It is a contrast between systems that have chosen to prioritise prevention and those that have not. The same has been demonstrated with evidence in the present 3 case scenarios.

As an Indian student, I understand that childhood disability due to CH is an already well solved biological problem. Our responsibility is to ensure that the solution reaches every child and this requires clinical competence in neonatal thyroid disorders, advocacy for universal screening programs, commitment to equitable health systems, and the human skill to support families navigating a difficult diagnosis. The NBS for CH at its core, is both a scientific and a moral exercise and

demands our full responsibility & engagement.

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