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Antenatal-Onset Clues to Congenital Myopathy in a Term Neonate With Birth Asphyxia: A Case Report

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Abstract

Hypoxic-ischemic encephalopathy (HIE) readily explains hypotonia, seizures, and respiratory failure in a term neonate who is depressed at birth, so a coexisting congenital myopathy may go unrecognized. We describe a term female neonate born after reduced fetal movements and meconium-stained liquor, with Apgar scores of 3 and 6 at one and five minutes, who was assessed as having stage II HIE; therapeutic hypothermia was unavailable. From the first day of life, she also had contractures of both elbows and knees, bilateral pes cavus, areflexia, and facial and bulbar weakness. An elder sister had died on the sixth day of life of an undetermined cause. Total creatine kinase was not elevated (30 U/L). Weakness did not improve as expected for moderate encephalopathy, and extubation failed seven times because of inadequate respiratory effort, often with a clear chest radiograph. Over 65 days, she developed refractory shock, recurrent seizures, and eight cardiac arrests, and she died. Whole-exome sequencing, reported two days after her death, identified a homozygous in-frame deletion of uncertain significance in ryanodine receptor 1 (RYR1) and a homozygous OGDH variant of uncertain significance; tandem mass spectrometry showed elevated very-long-chain species without a corresponding genetic finding. Parental segregation analysis, muscle biopsy, electromyography, electroencephalography, and magnetic resonance imaging were not feasible. Contractures and pes cavus present at birth, together with reduced fetal movements, indicate weakness that began before labor and cannot be attributed to the intrapartum event. With areflexia, failed extubation despite clear lungs, and a normal or low creatine kinase, these findings should prompt evaluation for congenital myopathy in an asphyxiated neonate. These clues are clinical and inexpensive, and they remain useful where confirmatory testing is slow or unavailable.

Categories: Pediatrics
Keywords: birth asphyxia, congenital myopathy, extubation failure, hypoxic-ischemic encephalopathy, neonatal hypotonia, ryanodine receptor 1

Introduction

Hypoxic-ischemic encephalopathy (HIE) is a frequent cause of hypotonia in the newborn, and central causes account for most cases of neonatal hypotonia [1]. When a term infant is depressed at birth and then shows hypotonia, seizures, and respiratory failure, HIE offers a complete explanation, and the diagnostic process often stops there.

A congenital myopathy presenting at birth produces hypotonia, weak respiratory effort, feeding difficulty, and failure to establish spontaneous respiration, all of which are also features of perinatal depression. Respiratory failure caused by the myopathy can itself lead to hypoxic-ischemic injury at delivery, so the two conditions may coexist, and the myopathy is easily concealed [2]. Two recent reports describe infants with ryanodine receptor 1 (RYR1)-related myopathy whose presentation was initially attributed to HIE [2,3].

We report a term neonate in whom a probable congenital myopathy coexisted with birth asphyxia, and in whom the most informative clues were findings present at birth that could not have arisen during labor. We use the case to propose bedside features that should prompt evaluation for a neuromuscular disorder in an asphyxiated infant, particularly where therapeutic hypothermia, neuroimaging, and genetic confirmation are not readily available.

Case Presentation

A term female neonate (40 weeks and six days) was born by vaginal delivery after pre-labor rupture of membranes eight hours before delivery to a 28-year-old third-gravida mother. The pregnancy was notable for reduced fetal movements and meconium-stained liquor. The birth weight was 2.76 kg, appropriate for gestational age. The parents reported no consanguinity, although both originated from the same locality. An elder sister had died on the sixth day of life of undetermined cause, a six-year-old brother was healthy, and a paternal uncle was receiving treatment for epilepsy (Figure *1*).

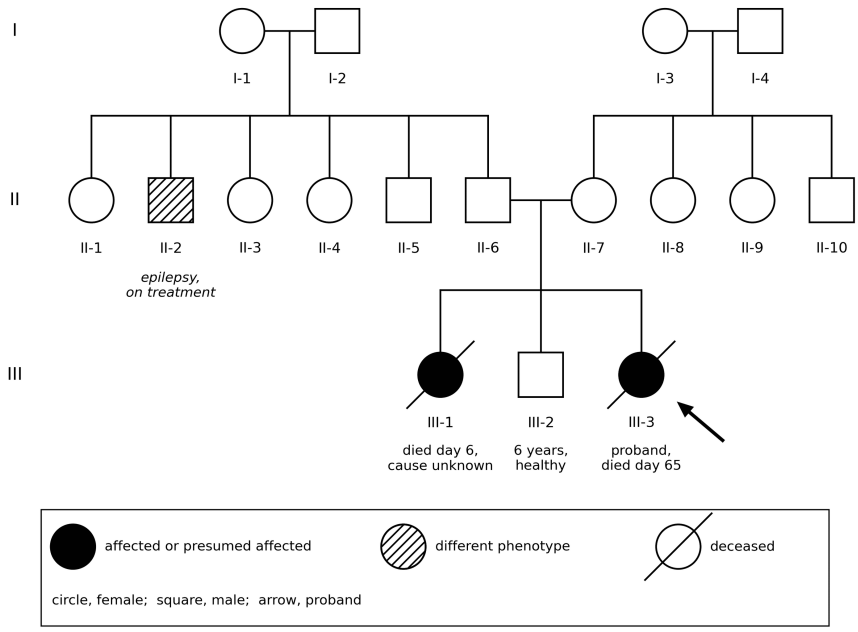


FIGURE 1: Three-generation pedigree.

The proband (III-3, arrow) and an elder sister (III-1), who died on the sixth day of life of undetermined cause, are shown as affected or presumed affected and deceased; a brother (III-2) is healthy. A paternal uncle (II-2) receives treatment for epilepsy and is shown hatched to indicate a different phenotype. The parents (II-6 and II-7) reported no consanguinity but originate from the same locality.

The infant did not cry at birth and required bag-and-mask ventilation for approximately 60 seconds. Apgar scores were 3 at one minute and 6 at five minutes [4]. A single umbilical artery was noted, and cord blood gas analysis was not available. She was assessed as having stage II HIE by Sarnat criteria [5]. Therapeutic hypothermia was not administered because servo-controlled cooling was not available in the unit.

On the first day of life, she had profound axial and appendicular hypotonia with a frog-leg posture [1], proximal more than distal weakness, facial and bulbar weakness with a weak suck and impaired swallowing, and absent deep tendon reflexes. She was lethargic, with poor spontaneous activity. She also had fixed contractures of both elbows and knees and bilateral pes cavus, findings that, unlike the encephalopathy, could not have been produced by an intrapartum event.

Blood glucose, ionized calcium, ammonia, lactate, blood gas, and thyroid function were unremarkable. Total serum creatine kinase (CK) was 30 U/L. C-reactive protein rose from 15.2 to 23.0 mg/L, and two blood cultures were sterile, most likely because empirical antibiotics had been started before sampling. Lumbar puncture was deferred throughout the admission because of hemodynamic instability on inotropic support, and clinical meningitis was treated empirically. Deletion analysis of SMN1, the gene deleted in most cases of spinal muscular atrophy, was negative. A point-of-care ultrasonography on day 4 showed poor cardiac contractility and no intracranial hemorrhage, and a later cranial ultrasound showed ventricular dilatation.

She was intubated within the first hours of life for poor respiratory effort and shock, for which dobutamine was started. She was extubated to non-invasive support the following day, re-intubated on day 4, and thereafter remained ventilator-dependent apart from a single day on non-invasive support (Figure *2*). Over 65 days, she required escalating circulatory support with dobutamine and dopamine up to 20 mcg/kg/min each, with adrenaline, noradrenaline, and hydrocortisone during refractory episodes; antibiotic escalation from ceftriaxone and amikacin to meropenem with vancomycin; two red cell transfusions; and treatment of recurrent seizures, which began on day 10, with phenobarbital and levetiracetam. Eight cardiac arrests occurred, and seven were followed by return of spontaneous circulation.

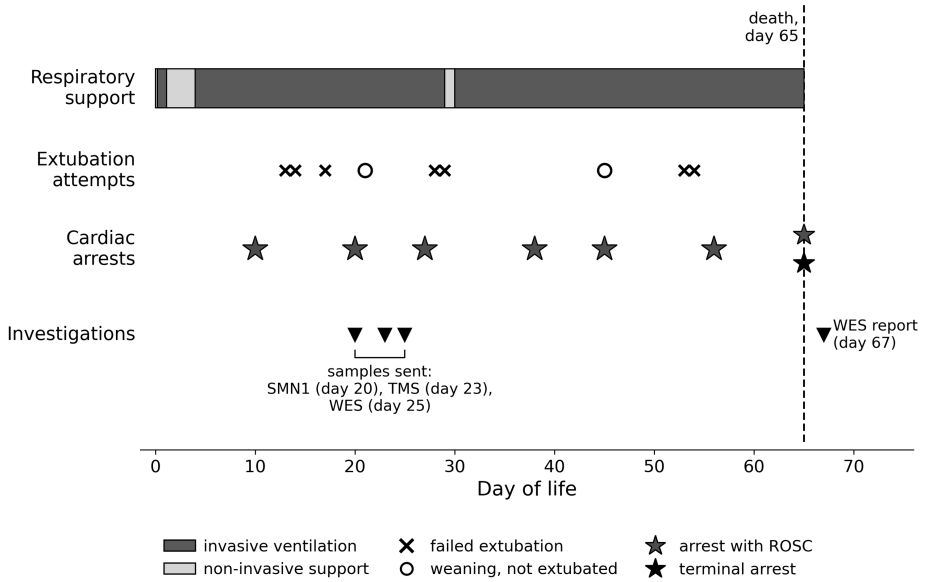


FIGURE 2: Clinical course over the 65-day admission.

The infant was intubated within hours of birth, extubated the following day, and re-intubated on day 4, after which she remained ventilator-dependent apart from days 29 to 30. Extubation failed on seven occasions, and weaning on two further occasions did not progress to extubation, each time because of inadequate respiratory effort. Eight cardiac arrests occurred, seven with return of spontaneous circulation; two occurred on the final day. The WES report was issued two days after death.

ROSC: return of spontaneous circulation; SMN1: survival of motor neuron 1 gene; TMS: tandem mass spectrometry; WES: whole-exome sequencing

The most informative feature was ventilatory. Extubation failed on seven occasions, on days 13, 14, 17, 28, 29, 53, and 54, and weaning on days 21 and 45 did not progress to extubation. Each failure followed inadequate respiratory effort rather than failure of oxygenation, often with a clear or improving chest radiograph. Weakness did not improve over the first two weeks, as would be expected with moderate encephalopathy, and remained static thereafter.

Tandem mass spectrometry (TMS) of a dried blood spot collected on day 23 showed elevated very-long-chain acylcarnitines and lysophosphatidylcholines, whereas the long-chain acylcarnitines that mark mitochondrial fatty acid oxidation disorders were within reference ranges (Table *1*). An isolated mild elevation of hexanoylcarnitine, with normal octanoylcarnitine, was considered nonspecific.

Analyte	Result (µmol/L)	Reference range (µmol/L)	Interpretation
Long-chain acylcarnitines			
Tetradecenoylcarnitine (C14:1)	0.05	0.03-0.42	Normal
Hexadecanoylcarnitine (C16)	2.81	0.81-8.34	Normal
3-hydroxyhexadecanoylcarnitine (C16-OH)	0.02	0.01-0.07	Normal
Octadecenoylcarnitine (C18:1)	1.29	0.50-2.50	Normal
Medium-chain acylcarnitines			
Hexanoylcarnitine (C6)	0.31	0.02-0.12	Elevated
Octanoylcarnitine (C8)	0.08	0.02-0.17	Normal
Very-long-chain acylcarnitines			
Eicosanoylcarnitine (C20)	0.14	0.01-0.06	Elevated
Docosanoylcarnitine (C22)	0.05	0.00-0.01	Elevated
Tetracosanoylcarnitine (C24)	0.17	0.00-0.02	Elevated
Hexacosanoylcarnitine (C26)	0.19	0.00-0.02	Elevated
Lysophosphatidylcholines			
C20:0-LPC	2.68	0.06-0.45	Elevated
C22:0-LPC	1.02	0.07-0.41	Elevated
C24:0-LPC	1.38	0.22-0.95	Elevated
C26:0-LPC	0.69	0.13-0.58	Elevated

TABLE 1: Selected tandem mass spectrometry findings.

Values are from a dried blood spot collected on day 23. The long-chain acylcarnitines that mark mitochondrial fatty acid oxidation disorders were within reference ranges, whereas very-long-chain acylcarnitines and lysophosphatidylcholines were elevated.

LPC: lysophosphatidylcholine; OH: hydroxy

Whole-exome sequencing (WES) was sent on day 25 to an accredited laboratory (mean on-target depth 84x, GRCh38 reference) and reported two homozygous variants of uncertain significance (VUS). The first, in RYR1 (c.7769_7777del; p.Ser2590_Arg2593delinsCys; exon 48), is a nine-base-pair in-frame deletion that is absent from population databases, including the Genome Aggregation Database [6]; the laboratory assigned American College of Medical Genetics and Genomics criteria PM2 and PM4 [7]. It was predicted damaging by MutationTaster2 [8], although the affected region is not conserved across species. The second, in OGDH (c.1978G>A; p.Ala660Thr), which encodes a subunit of the oxoglutarate dehydrogenase complex, was reported as an additional finding; it is absent from the same databases [6] and was predicted damaging by SIFT [9], the likelihood ratio test [10], and MutationTaster2 [8], and possibly damaging by PolyPhen-2 [11]. No reportable variant was identified in genes associated with peroxisomal disorders. Sanger confirmation and parental segregation analysis were not affordable for the family.

On the final day, she developed shock refractory to maximal support. She was resuscitated from one arrest in the evening, but a second arrest did not respond to resuscitation, and she died on the 65th day of life. No autopsy was performed. The WES report was issued two days after her death.

Discussion

Each element of this infant's early presentation was compatible with perinatal asphyxia: she was depressed at birth, required positive pressure ventilation, and was lethargic and hypotonic with poor respiratory effort. A diagnosis of stage II HIE accounted for all of it, and we believe she did sustain hypoxic-ischemic injury.

Three findings, however, could not have been produced by labor. Fixed contractures of large joints require sustained intrauterine immobility over weeks to develop, pes cavus is a structural deformity of antenatal onset, and both were present on the first day of life; reduced fetal movements had been noted before

delivery. Together, they indicate that she was already weak before labor began. This reverses the usual causal reasoning. Rather than the asphyxial insult producing the weakness, pre-existing weakness plausibly contributed to her depressed state at birth, because a weak fetus tolerates labor poorly and an infant with a weak respiratory pump cannot readily establish effective ventilation [2]. Decreased fetal movement and arthrogryposis are recognized features of severe neonatal RYR1-related myopathy [12].

The overlap also distorts how encephalopathy is graded. Sarnat staging scores tone, reflexes, and spontaneous activity [5], all of which a congenital myopathy depresses independently of brain injury, so a myopathy can inflate the apparent severity of HIE [2]. Level of consciousness is the more discriminating criterion, as infants with a pure neuromuscular disorder generally remain alert [2]. Our patient was lethargic, which supports a genuine encephalopathic component, but the profound hypotonia and areflexia that contributed to her stage II assessment were at least partly attributable to the myopathy.

Table *2* lists features that should prompt evaluation for a neuromuscular disorder in an asphyxiated term neonate and indicates which were present in our patient. They fall into four groups: findings of antenatal onset, examination findings not expected from encephalopathy, deviation from the expected course, and supporting investigations and history. Two require no special resources. The first is the pattern of extubation failure. Failure driven by inadequate respiratory effort while the chest radiograph is clear points to the respiratory pump rather than the lung; in our patient, it recurred seven times over six weeks, and repeated extubation failure from apnea was also prominent in the infant described by Sanghamitra et al. [2]. The second is CK. A normal or low CK in a profoundly weak infant is counterintuitive but characteristic of the congenital myopathies, whereas marked elevation suggests a muscular dystrophy [13], so a normal value should not be read as evidence against muscle disease. Central causes of hypotonia, by contrast, are usually accompanied by normal or brisk reflexes [1], so areflexia in an asphyxiated infant is itself a reason to look further.

Feature	Why it points beyond intrapartum asphyxia	Present in this infant
Antenatal onset		
Reduced fetal movements or polyhydramnios	Reflect reduced fetal motor activity before labor	Yes (reduced fetal movements)
Contractures or arthrogryposis at birth	Require weeks of intrauterine immobility to develop	Yes
Pes cavus or other fixed foot deformity at birth	A structural deformity of antenatal onset	Yes
Examination		
Areflexia	Central causes of hypotonia usually spare or increase reflexes	Yes
Hypotonia out of proportion to the encephalopathy	Weakness exceeds that expected from the assigned Sarnat stage	Yes
Facial or bulbar weakness, ptosis, or ophthalmoplegia	Suggest weakness of muscle rather than depressed consciousness	Yes (facial and bulbar weakness)
Alertness preserved despite profound hypotonia	Infants with a pure neuromuscular disorder generally remain alert	No (lethargic)
Course		
Extubation failure from poor respiratory effort with a clear chest radiograph	Indicates a weak respiratory pump rather than lung disease	Yes (seven times)
Weakness static beyond the first one to two weeks	Moderate encephalopathy is expected to improve	Yes
Investigations and history		
Normal or low creatine kinase	Typical of congenital myopathy; marked elevation suggests muscular dystrophy	Yes (30 U/L)
Sibling with unexplained neonatal death, consanguinity, or community endogamy	Raises the prior probability of a recessive disorder	Yes (sister died on day 6)

TABLE 2: Features in an asphyxiated term neonate that should prompt evaluation for a congenital myopathy.

Features are drawn from this case and from published descriptions of neonatal congenital myopathy [1-3,12,13].

This case adds to two recent reports in which RYR1-related myopathy was initially attributed to HIE [2,3]. Patial and Anand described an Indian neonate with birth asphyxia, reduced fetal movements, and persistent ventilator dependence in whom WES identified a heterozygous RYR1 nonsense variant [3]. Sanghamitra et al. described a late preterm infant treated with therapeutic hypothermia who had a de novo RYR1 VUS and died at one month of age [2]. Our patient differs in three respects that bear on management. First, her variant was homozygous, and an elder sister had died in the neonatal period, which suggests autosomal recessive inheritance with a 25% recurrence risk in each pregnancy if both parents are carriers, in contrast to the low recurrence risk of a de novo variant. Second, she was managed without therapeutic hypothermia, neuroimaging, or electroencephalography, so recognition had to rest on bedside findings. Third, the genetic result arrived only after her death, so throughout her 65-day admission the diagnosis was either clinical or not made at all.

RYR1 encodes the principal calcium-release channel of the skeletal muscle sarcoplasmic reticulum, and RYR1-related myopathies are the most common congenital myopathies [14]. Recessive variants predominate among severe neonatal presentations [12] and are associated with a greater need for gastrostomy feeding than dominant variants [15]. Our patient's phenotype corresponds closely to this description, and her CK is concordant. We therefore regard RYR1-related myopathy as the most probable explanation for her neuromuscular disease, while stressing that it is not confirmed: the variant remains a VUS, the affected region is not conserved, and neither segregation analysis nor functional studies were possible. Her course was also more severe than is typical, since in a large pediatric cohort all patients with RYR1-related myopathy survived without long-term ventilation, including those with severe neonatal onset [15]. We attribute her death to the combination of myopathy, hypoxic-ischemic injury, and infection rather than to the myopathy alone.

The OGDH variant cannot be placed with confidence. Biallelic OGDH variants cause a neurodevelopmental disorder with hypotonia and seizures [16], but our patient had no lactic acidemia, and the laboratory reported the variant only as an additional finding.

Two homozygous variants absent from population databases are unexpected in a couple who report no consanguinity. The likeliest explanation is shared ancestry through community endogamy. The parents come from the same locality, and South Asia comprises many small endogamous groups in which founder events raise the frequency of recessive disease independently of marriage between close relatives [17]. Parental genotyping would have tested this directly.

The TMS findings remain unresolved. The elevated very-long-chain species suggest impaired peroxisomal beta-oxidation, as in Zellweger spectrum disorders [18], but WES identified no corresponding variant, and secondary elevation during critical illness is a reasonable alternative explanation.

This report has important limitations. It describes a single infant without a confirmed diagnosis. Both variants are VUS, and neither was confirmed by Sanger sequencing or segregation analysis. Muscle biopsy, electromyography, magnetic resonance imaging, and electroencephalography were not performed, either because the infant was never stable enough for transport or because the investigation was unavailable, so the relative contributions of myopathy and hypoxic-ischemic injury cannot be quantified. The timing of events was reconstructed retrospectively from clinical records. The features in Table *2* are drawn from this case and from published descriptions and have not been validated prospectively; they are intended to prompt further evaluation rather than to serve as a diagnostic instrument.

Earlier recognition would not have changed this infant's survival, as no disease-modifying treatment exists for RYR1-related myopathy [14]. It would, however, have tempered expectations of extubation, prompted earlier discussion of tracheostomy or of the limits of escalation, allowed counseling about recurrence while the infant was alive, and prompted anesthetic precautions, because RYR1 variants are associated with susceptibility to malignant hyperthermia [14]. It would also have prompted avoidance of aminoglycosides. Amikacin was given empirically for the first four days, before a neuromuscular disorder was suspected, and aminoglycosides, including amikacin, can impair neuromuscular transmission and worsen weakness in patients with neuromuscular disorders [19]. It may have contributed to her poor respiratory effort in the first days of life, but it had been stopped well before the first failed extubation on day 13 and cannot account for the repeated failures that followed. Where therapeutic hypothermia, the standard of care for moderate to severe HIE [20], is unavailable, as it was in our unit, the bedside clues in Table *2* carry proportionally greater weight.

Conclusions

A congenital myopathy can coexist with, and contribute to, birth asphyxia in a term neonate, and its features are easily attributed to HIE. Findings that predate labor, such as joint contractures, fixed foot deformities, and reduced fetal movements, cannot be explained by an intrapartum insult and should prompt evaluation for a neuromuscular disorder, as should areflexia, repeated extubation failure despite clear lungs, and weakness that fails to improve as expected. Because these signs require only careful bedside examination, they are most valuable where genetic confirmation is delayed or unavailable.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Abhishek Navik, Nikita Kumari, Sweta Kumari, Ravi Sachan

Acquisition, analysis, or interpretation of data: Abhishek Navik, Nikita Kumari, Sweta Kumari

Drafting of the manuscript: Abhishek Navik, Nikita Kumari, Sweta Kumari, Ravi Sachan

Critical review of the manuscript for important intellectual content: Abhishek Navik, Nikita Kumari, Sweta Kumari, Ravi Sachan

Supervision: Abhishek Navik, Ravi Sachan

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have

declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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