DOI: https://doi.org/10.47275/2379-6707-110
Volume 7 Issue 2
Commentary

Scholit Publishers

Journal of Pediatrics and Congenital Disorders

Commentary: The Blind Spot in SIDS Research

Pontus M Siren*

Independent researcher, Crassier, Switzerland

Abstract

Despite decades of intense research, the cause of death in Sudden Infant Death Syndrome (SIDS) remains unknown. Two communications published in Nature Pediatric Research (Blind Spot and The Missing Link) on April 6 and June 5th, 2021, respectively, highlight the fact that there is a potentially consequential blind spot in SIDS research. This short communication draws attention to the blind spot and highlights five reasons why the diaphragm should be investigated in the context of SIDS.

Keywords: Sudden infant death syndrome; SIDS; Diaphragm failure; Pediatric critical care

Commentary

Despite decades of intense research, the cause of death in Sudden Infant Death Syndrome (SIDS) remains unknown. Two communications published in Nature Pediatric Research (Blind Spot and The Missing Link) on April 6 and June 5th, 2021, [1,2] respectively, draw attention to the fact that there is a potentially consequential blind spot in SIDS research. This short communication draws attention to the blind spot and highlights five reasons why the diaphragm should be investigated in the context of SIDS:

1. There is broad consensus that SIDS likely has a respiratory origin
2. All key cardiorespiratory organs, except the diaphragm, have been extensively investigated in the context of SIDS
3. The diaphragm powers the vital respiratory pump and is critical for survival
4. There is no compelling evidence to suggest the diaphragm can be excluded from SIDS research
5. There is a substantial body of evidence to suggest that the diaphragm plays a central role in SIDS, and the SIDS – Critical Diaphragm Failure (SIDS-CDF) hypothesis posits that all SIDS risk factors either increase the workload of the diaphragm or reduce its force-generating capacity.

The origin of SIDS remains a mystery, but there is broad consensus that the syndrome likely has a respiratory origin because research and clinical investigation have ruled out airway occlusion or trauma as the cause of the syndrome. Similarly, cardiac failure has been excluded through clinical observations, and heart rate and respiratory recordings of infants who subsequently succumb to SIDS [3].

The number of articles published on PubMed is a good indicator of the focus of SIDS research. On October 5th, 2021, there are 12,365 publications on sudden infant death syndrome. Of these, 2285, relate to SIDS & the heart, 714 to SIDS & lungs, 982 to SIDS & central nervous system. However, there are only 59 publications on SIDS & diaphragm of which seven pertain to the SIDS-CDF hypothesis. Interestingly, even the possible role of the liver in SIDS has been more actively investigated with 281 published articles.

The diaphragm is critical for survival because it powers the vital respiratory pump. This is even truer in young infants who have underdeveloped secondary respiratory muscles and a pliable chest wall. Diaphragm failure is a well-known terminal event in adults.

The 59 articles on SIDS & diaphragm, contain no evidence to suggest that we can rule out the diaphragm as a causative factor in SIDS. Indeed, the scarcity of the research speaks to the fact that the diaphragm has not been adequately investigated in the context of SIDS and as such should not be excluded from SIDS research.

The SIDS-CDF hypothesis explains why the diaphragm should be investigated in the context of SIDS. The hypothesis posits that all SIDS risk factors either increase the diaphragm workload or decrease its force-generating capacity. Six articles and communications published since 2011,

Correspondence to: Pontus M Siren, Independent researcher, Crassier, Switzerland; E-mail: pontusmax@hotmail.com

Citation: Siren PM (2021) Commentary: The Blind Spot in SIDS Research. J Pediatr Congenit Dis, 7(2): 110. DOI: https://doi.org/10.47275/2379-6707-110

Received: October 06, 2021; **Accepted:** October 20, 2021; **Published:** October 25, 2021

[1-6] argue that there is compelling evidence to suggest that SIDS risk factors such as non-lethal infections and the prone sleeping position among others, directly affect diaphragm function. There is a strong correlation between SIDS and non-lethal infections, and it is well established that non-lethal infections can significantly reduce diaphragm force-generating capacity without leaving pathological signs in the affected tissue. Similarly, the prone sleeping position has been shown the significantly increase the workload of the infant diaphragm. Furthermore, there is direct genetic evidence to suggest that the diaphragm plays a role in the etiology of SIDS. A recent report in the Lancet showed that pathogenic gene variants of SCN4A that encode for NaV1.4, a skeletal muscle voltage-gated sodium channel that is crucial for the generation of action potentials and excitation of muscle, are overrepresented in infants who died of SIDS compared with ethnically matched controls. These SCN4A variants have been shown to impair the ability of respiratory muscles to respond to hypoxia [7].

There is also compelling evidence directly linking other SIDS risk factors such as hypoxia, REM sleep, hyperthermia, altitude, absence of breastfeeding, and hypomagnesemia to critical diaphragm failure. Furthermore, the incidence of intrathoracic petechial hemorrhages that are found in over 70% of SIDS cases, is consistent with the SIDS-CDF hypothesis: to form, intrathoracic petechiae require an infection combined with gasping or a "struggle to breathe" episode(s), and the clinical signs of respiratory muscle fatigue are rapid shallow breathing, paradoxical chest wall movements, and respiratory pauses combined with hyperventilation (see Siren PM publications 2011-2021).

Conclusion

SIDS likely has a respiratory origin and despite decades of intense research, the cause of death is unknown. Besides the diaphragm, all other key cardiorespiratory organs have been comprehensively investigated in the context of SIDS. There is no evidence to suggest the diaphragm should be excluded from SIDS research. Yet, the diaphragm remains a collective blind spot that may contribute decisively to our inability to uncover the cause of SIDS.

Competing Interest

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Funding

None.

References

1. Siren PM (2021) Blind spot. Pediatr Res. https://doi.org/10.1038/s41390-021-01508-4
2. Siren PM (2021) Sudden infant death syndrome and the diaphragm: Is there a link? Pediatr Res. https://doi.org/10.1038/s41390-021-01640-1
3. Siren PM, Siren MJ (2011) Critical diaphragm failure in sudden infant death syndrome. Upsala J Med Sci 116: 115-123. https://doi:10.3109/03009734.2010.548011
4. Siren PM (2013) The SIDS--critical diaphragm failure hypothesis revisited. Upsala J Med Sci 118: 62-64. https://doi:10.3109/03009734.2012.744373
5. Siren PM (2016) SIDS-CDF hypothesis revisited: explaining hypoxia in SIDS. Upsala J Med Sci 121: 199-201. https://doi:10.1080/03009734.2016.1176972
6. Siren PM (2017) SIDS-CDF Hypothesis Revisited: Cause vs. Contributing Factors. Front Neurol 7: 244. https://doi:10.3389/fneur.2016.00244
7. Männikkö R, Wong L, Tester DJ, Thor MG, Sud R, et al. (2018) Dysfunction of NaV1.4, a skeletal muscle voltage-gated sodium channel, in sudden infant death syndrome: a case-control study. Lancet 391: 1483-1492. https://doi.org/10.1016/S0140-6736(18)30021-7