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Literature Review

Sudden Death Syndrome in Infants and Children

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Abstract

Background: Unpredicted death of a nearly healthy child is considered a "sudden unexplained childhood death (SUDI)" and refers to this in a child aged 7 to 365 days. Unforeseen deaths in infants under 7 days of age are removed from the category of SUDI, according to the majority of definitions, and are called 'sudden unexpected early neonatal death' (SUEND)". Infection is the most prevalent cause of the known causes, as well as metabolic and cardiovascular diseases, although the share of cases are much lower.

Aim: In this review, we will look into the prevalence, different causes and management of sudden death syndrome among infants.

Conclusion: Mechanisms contributing to sudden death tend to be complex and multi-factorial, involving the involvement of multiple different mortality causes. Parents, particularly mothers, should be encouraged and educated on the causes and prevention of SIDS through health services and campaigns.

Keywords: *Sudden Death Syndrome; SUDI; Causes of Sudden Death in Infants; Management of SUDI*

Introduction

In a healthy balanced infant, the sudden death is called "sudden unexpected children's mortality" (SUDI). In most of the definitions unexpected deaths in infants under 7 days old were omitted from 'SUDI' group and instead were classified as 'suddenly unexplained early neonatal death' (SUEND All cases of 'SUDI' and 'SEND' are examined to ascertain the cause of death).

The primary purpose of the postmortem test, including its components and additional inquiries, is to identify or exclude certain normal and unnormal cause of death [1]. Neoplasms, disorders affecting nervous, cardiovascular or respiratory systems, and infections are the most frequently acquired causes of natural death for infants and children in England and Wales (0 - 27 days) [2]. Comprehensively, however, it is difficult to establish accurate figures about the proportions of expounded and unexplained deaths following autopsy. This is due, in part, to broad variability in the death reporting process, rendering epidemiological assessment unreliable [3] and a lack of large population-based studies, especially those investigating SUDCA Latest review records 24 public studies in 25 cohorts from 11 countries (17 in children, 4 in both children and adolescents and just 4 in females); 9% to 67% of SUDI and 22% to 86% of cases of SUDC have been found to lead to death following the full review [4].

The most common cause is the infection, as well as metabolic and cardiovascular disorders, although the number of cases is much smaller. There is now substantiation that cardiac channel gene mutations also play an important role, Sudden cardiac death (SCD) is characterised as death that is hurried, unexpected, and due to a cardiovascular cause. Death typically takes place within 1 hour after cardiovascular symptoms begin. However, young adolescents develop symptoms within just few minutes of onset. If recovery restores spontaneous circulation, it is called 'sudden death aborted'".

The prevalence estimated at 100,000 children of paediatric SCD is in the US of 0.6-6.2 Approximately 20-25 per cent of sports deaths are occurring [5]. However, it is focused on overestimated research that is inaccessible or widely available that describes these conditions. In order not to have a similar heartbreak in the same family, it is vital that anything be done to determine the cause of death [6]. In future studies SUID should be treated as a disconnected entity from post-perinatal sudden SUEND. These data may help to improve SUEND and SUID's epidemiological understanding and provide evidence for a systematic understanding of the causes of death [7].

In this review, we will look into the prevalence, different causes and management of sudden death syndrome among infants.

Literature Review

In a recent structured literary examination in the Middle East, several infant deaths have been reported as "unknown cause" for a variety of reasons and no related autopsy report or other additional data. The most important risk factor for SIDS in the ME countries reported in the literature is the high prevalence of smoking arising from less legislative controls on smoking at home and in public settings. Special attention must be paid to childcare, diets and general living conditions in high-risk ME countries with a concentrated emphasis on public health services for SIDS [8].

In another study in KSA; cause-specific infant mortality rate from Qatif region, KSA was 21.06/1000 live births. In the neonatal age, 40 fifth (70,3%) of accidents happen and about 75% were preventable (39.1 per cent), 16 (25 per cent) infections, 12 (18.8 per cent) birth defects, 4 (6.3 per cent) sudden infant mortality syndrome (SIDS) and 3 (4.7 per cent) challenging delivery [9].

A study described survey performed to record a 6-year period on the incidence and key causes of sudden deaths in a major university hospital in Eastern Saudi Arabia. Of 1273 investigated hospital record; 223 (17.5%) cases of sudden death. Men accounted for 56.0% of cases. At 32.2 percent of all deaths of all ages, the sudden infant death occurred. In 59.2 percent of cases, cardiovascular diseases are the most significant direct causes of sudden death, in 24.7 percent, in 23,8 percent for diabetes mellitus and in 20.2 percent for infectious disease.

Participants and Methods

Study design: Review article.

Study duration: Data were collected between 1 June and 30 August 2020.

Data collection Medline and PubMed public database searches have been carried out for papers written all over the world on the most notable advances in childhood sudden death disorder. The keyword search headings included "sudden death syndrome, SUDI, causes of sudden death in infants, management of SUDI", and a combination of these will be used. For additional supporting data, the sources list of each research was searched. Criteria of inclusion: the papers have been chosen on the basis of the project importance, including one of the following topics: sudden death syndrome, SUDI, sudden infant deaths, SUDI management, etc.). Criteria for exclusion: all other publications that did not have their main purpose in any of these areas or multiple studies and reviews were excluded.

Statistical analysis

No predictive analytics technology has been used. In order to evaluate the initial results and the methods of conducting the surgical procedure, the group members reviewed the data. The validity and minimization of error were double revised for each member 's results.

Prevalence

Around 1,400 deaths from SIDS, around 1,300 unexplained deaths from beds and approximately 900 cases of actual suffocation and asphyxiation were recorded in 2017 [11].

Sudden deaths due to population-specific deaths in 2015 and 2016 were 120/100 000 live births and 1.9/100 000 children from the legal records of infants (< 365 days) and children (1 - 17 years). Infants (2.7/100 000 live births) showed greater cardiac rates than infants (0.3/100 thousand infants). The accidental mortality rate in pediatric sudden epilepsy death (SUDEP) amounted to 0.2/100,000 live births. Non-white races accounted for 42% of births and 43% of deaths, but just 23% of the population. The most common cause was cardiac cause of respiratory disorder [12].

Causes

Congenital and developmental anomalies: Any part of the respiratory tract can suffer from congenital and developmental anomalies. Some of them are macroscopically visible during autopsy, while others require microscopic exams. The most likely cause of death by airway compromises is gross anomalies [13]. The complexity of the respiratory tract means that many anatomical areas may be affected by irregular changes or infections which can cause childhood death. The respiratory tract is divided anatomically into the upper and lower respiratory tract (URT, LRT). The URT starts with nasal and mouth passages, perinatal sinuses, pharynxes and the upper-vocal cord larynx component; the LRT is defined in larynx as the lower part than vocal cords, tracheas, bronchi and acini (including bronchiole, alveolary ducts and alveoli); thoracic cage and diaphragm, which normally separates them from a pleural room, restrict the lungs. A wide variety of diseases can affect either of these sites and air pathology is one of the most prevalent organ systems for natural deaths in infancy and childhood [14].

A group of lung, developing lung diseases of indefinite etiology typically present in the neonatal phase (CLDs), are key diffuse pulsation disorders (CLDs).

Acinar dysplasia (AD) tends to be the primary arrest of lung growth, because the lungs are abnormally small with apparent maturation arrest in the pseudo-glandular or early canal stage [15]. The failure of normal acini prevents proper gas exchange and infants affected are ventilating refractory and die soon after birth. The underlying cause remains uncertain but in at least some cases case reports showing positive family history indicates a genetic origin [16].

Congenital alveolar dysplasia (CAD) is a primary alveoli malformance arising from obstruction and normal pulmonary growth disorder. The lungs may be ordinary in size, but a histological analysis suggests that the late or early secular growth has been stopped. Neonates show symptoms of respiratory failure and bruising shortly after birth. Though recovery is possible, they remain dependent on the ventilator and die as a result of respiratory failure [17].

In most children, early neonatal hypertension (PPHN) is encountered by advanced breathing failure and cyanoses within 48 hours of birthing. However, delayed implementation can occur weeks or months later. Mortality reaches 100% regardless of the age at present, with most recorded cases found during autopsy [18].

Inborn errors of surfactant metabolism are erratic and extant chiefly in infancy. More than 30 inherited latent autosomal mutations of genes affecting surfactant development and treatment have been identified, and the causative gene is very much dependent on clinical ap-

pearance and histological indicators [19]. SFTPC deficiency usually affects older infants and demonstrates a form of chronic infant pneumonitis (CPI) with interstitial thickening, moderate inflammatory infiltration, edema, and type II diffuse pneumocystis hyperplasia [20].

Chronic fetal pulmonary hypertension (PPHN) is present after failure of natural cardiopulmonary artery relaxation from foetal to independent circulation. PPHN is related with a 50% mortality rate, with perinatal asphyxia, meconium aspiration, pneumonia, and pulmonary hypoplasia [21]. Surge deaths are uncommon but well described, with 10 percent of all sudden deaths recorded in the first week of existence [22].

Acquired respiratory conditions: Bronchial asthma is a common childhood respiratory condition characterized by airway hypersensitiveness to allergens, irritants or infectious diseases, cold air, exercise and emotional distress. Acute exacerbations can lead to asthma, which is a well-known cause of death. refractive therapy for asthma. But tragic death can happen in children who have previously been considered mild or had minimal symptoms of early death [23].

Anaphylaxis: Anaphylaxis is an acute life-threatening reaction to an allergen in a prone individual. Meat, followed by insect venom, is normally triggered in children [24]. The most common causes for those younger than 2 years are allergies to cow milk and eggs. Older children in pre-school years are more influenced by the reaction of nuts, including hazelnuts and cashews. Medical symptoms of a molecular edoema affecting lips, tongues and bronchospasm will rapidly lead to death without urgent medical attention. However, anaphylaxis diagnosis is difficult at autopsy, as there may be no clear gross or histological characteristics, so descriptions of death and medical history are necessary. The blood test is most reliable and indicates an improvement in mast cell striptease (samples are useful up to 3 days after death) and rises in total IgE or IgE levels of established or susceptible allergens Allergens [25].

Congenital heart disease: Congenital heart disease (CHD) is the most prevalent birth defect in about 1% of births in life [26]: 6 to 8 of 1,000 live births are predictable [27]. Despite this, undiagnosed congenital heart disease remains an significant cause of sudden unexpected death, especially in early childhood, with studies showing that as many as 25 percent of babies with serious congenital heart disease might be satisfied with an undiagnosed hospital, some of whom are first diagnosed with autopsy [28]. Most life-threatening defects in the neonatal era (0 - 27 days) have a systemic or pulmonary circulation problem based on the duct, with symptoms becoming evident when the ductus arteriosus is closed.

Detailed CHD explanations extend beyond this analysis but select examples are given based on sort of situations most commonly seen as sudden unexplained deaths in infancy and early childhood. It is recommended to regularly examine the heart in conjunction with subsequent segmental exams, which assess and decide all ties, chambers and relationships. Anomalous venous pulmonary drainage can have irregular anatomical properties, which must be studied in-situ prior to removal of the heart or at least the connexions [27].

Aortic stenosis is around 5% CHD [29] and may be above, below or in the aortic valve if secondary to cuspid abnormalities. Severe aortic stenosis is life-threatening, causing obstruction of the left ventricular outflow channel, and allowing the systemic duct-dependent circulation [30]. At the extreme end of the aortic stenosis, with or without mitral stenosis or atresia syndrome, Hyperplastic left heart HLHs fuses with the underdevelopment of the left side of the heart. The number of neonatal CHD deaths in HLHS is up to 25 percent [31]. HLHS etiology is uncertain and although serious cases can be diagnosed during mid-trimester ultrasound scanning, HLHS can occur later during pregnancy and remain undetected shortly after birth until success. In these cases it has been conjectured that an intrauterine attack arises after embryogenesis in a genetically prone fetus; in fact it may be immunological, viral, or autoimmune [31].

Central nervous system (CNS) infections: Meningitis is inflammation of the brain and spinal cord accompanying leptomeninges; it may be due to infections that are bacterial, viral, fungal or parasitic. Bacterial causes very much depend on the child's age. For example, GBS, *Escherichia coli* and other gram-negative species are the key causes of neonatal meningitis [32]. Neisseria meningitides, however, presents a higher risk for children > 1 and adolescents [33]. Fungal meningitis is typically caused by yeast-production fungal diseased diseases like the Cryptococcus or Coccidiodomycosis [34].

Sudden unexpected death in epilepsy: Sudden epileptic death (SUDEP) is a famous complication of seizure, with infancy rates of between 1.1-4.3/10,000 patient years [34]. A diagnosis of failure indicates a person with a known epileptic background without any anatomical or toxicological cause of death. Brain structure problems such as cortical malformations, sclerosis of the hippocampus, atrophy of the brain and hydrocephalus can be defined as causing epilepsy. It suggested a variety of causes, including cardiac arrhythmias and central apnea [35].

Bacteria: Bacteria such as *S. aureus* and *E. coli* developed toxins pose a particular risk to infant lives. Mucosal surface absorption of the toxins of these bacteria or a forthcoming bacteremia that primarily affect the cardiovascular and respiratory system, causing 'channels' in cell membranes that disrupt smooth ions. [36]. *Neisseria meningitides* (meningococcus) is the leading cause of early childhood infectious death [37] with a mortality rate > 20% in meningococcal sepsis children [38,39].

Sleeping environment: The sleeping environment concerns the child's health, bed consistency, bed distribution, and smoking during health. Sleep conditions are very critical. Newborns without resistance to sudden death are those who change their sleep position and are susceptible to death [38,39]. Infants sleeping on the lateral side are two times more likely to die unexpectedly than sleeping in the abdominal position [40]. Many parents and professionals of health immediately note that supine sleep impairment of the neonatal respiratory system increases the risk of gastro-esophageal reflux [41].

When the body temperature of the newborn rises due to higher room temperature, fever, sweating or excessive clothes the chances of SIDS increase. Whereas The sudden death risk two and six times is linked to combinations of risk factors like prone and soft layer or elevated body temperature and sleep [42].

The sharing of a child's bed with parents or twins often contributed to a high risk of SIDS [43]. Babies less than 13 weeks aged who slept with their parents are in higher risk to sudden death [44].

Managements of (SIDS)

The causes of SIDS are not entirely understood, but the above factors have a sudden death impact [45]. The American Academy of Pediatrics has also established in practice precautions to minimize the occurrence of SIDS. These measures are:

- The most adaptable aspect is the location of the child at bedtime. The high temperature of the child is preferred to be avoided, acceptable room temperature preserved, unjustified clothing avoided [46].
- It is better to have a firm and comfortable sleeping surface [47].
- Clinical results indicate that compromises lead to the reduction of risk of sudden sleep death. The American psychiatric association suggests that parents work hard to their children from the outset of infancy [48]. However, pacifier use is occupied in the creation of otitis media and airway obstruction [49].
- The baby crib is advised to be placed in the same location as the parent's cabin and not to modify the space for sleeping babies; Immunization in time often tends to be protective against SIDS for at least the first year of a child's life [50].
- Educational assistance for parents and healthcare professionals in the reduction of SIDS is equally relevant [51].

Conclusion

Mechanisms contributing to sudden death tend to be complex and multi-factorial, involving the involvement of multiple different mortality causes. At present, it is not possible to determine which mixture of variables will result in the death of a SIDS in infant. The main

goal of post-mortem investigation is to decrease the chance of SIDS causes and decrease incidence. Parents, particularly mothers, should be encouraged and educated on the causes and prevention of SIDS through health services and campaigns.

Bibliography

1. Office for National Statistics. Childhood mortality in England and Wales (2014).
2. Sidebotham P., *et al.* "Patterns of child death in England and Wales". *Lancet* 384.9946 (2014): 904-914.
3. Taylor BJ., *et al.* "International comparison of sudden unexpected death in infancy rates using a newly proposed set of cause-of-death codes". *Archives of Disease in Childhood* 100.11 (2015): 1018-1023.
4. Coté A. "Investigating sudden unexpected death in infancy and early childhood". *Paediatric Respiratory Reviews* 11.4 (2010): 219-225.
5. Botvinick EH., *et al.* "Hypertrophic cardiomyopathy in the young: another form of ischemic cardiomyopathy?" *The American College of Cardiology* 22.3 (1993): 805-807.
6. Coté A. "Investigating sudden unexpected death in infancy and early childhood". *Paediatric Respiratory Reviews* 11.4 (2010): 219-225.
7. References and abstracts from MEDLINE/PubMed (National Library of Medicine) Article: Distinct Populations of Sudden Unexpected Infant Death Based on Age.
8. Shereen Hamadneh., *et al.* "Sudden Infant Death Syndrome in the Middle East: An Exploration of the Literature on Rates, Risk Factors, High Risk Groups and Intervention Programs (2016).
9. Srair HA., *et al.* "Cause-specific infant mortality rate in Qatif area, eastern province, Saudi Arabia". *Annals of Saudi Medicine* 15.2 (1995): 156-158.
10. HK Nofal., *et al.* "Incidence and causes of sudden death in a university hospital in eastern Saudi Arabia". *Eastern Mediterranean Health Journal* 17.9 (2011): 665-670.
11. Cole CR and Eghtesady P. "The myocardial and coronary histopathology and pathogenesis of hypoplastic left heart syndrome". *Cardiology in the Young* 26.1 (2016): 19-29.
12. Kristin M Burns., *et al*. "Epidemiology of Sudden Death in a Population-Based Study of Infants and Children (2020).
13. Gilbert-Barness EF., *et al.* "Handbook of pediatric autopsy pathology". Springer: New York (2014).
14. GBD 2015 Child Mortality Collaborators. "Global, regional, national, and selected subnational levels of stillbirths, neonatal, infant, and under-5 mortality, 1980-2015: A systematic analysis for the Global Burden of Disease Study 2015". *Lancet* 388.10053 (2016): 1725-1774.
15. Armes JE., *et al.* "Diffuse lung disease of infancy: A pattern-based, algorithmic approach to histological diagnosis". *Journal of Clinical Pathology* 68.2 (2015): 100-110.
16. Chow CW., *et al.* "Acinar dysplasia of the lungs: Variation in the extent of involvement and clinical features". *Pathology* 45.1 (2013): 38-43.
17. Armes JE., *et al.* "Diffuse lung disease of infancy: A pattern-based, algorithmic approach to histological diagnosis". *Journal of Clinical Pathology* 68.2 (2015): 100-110.
18. Bishop NB., *et al.* "Alveolar capillary dysplasia". *American Journal of Respiratory and Critical Care Medicine* 184.2 (2011): 172-179.

19. Armes JE., *et al.* "Diffuse lung disease of infancy: A pattern-based, algorithmic approach to histological diagnosis". *Journal of Clinical Pathology* 68.2 (2015): 100-110.

20. Dishop MK. "Paediatric interstitial lung disease: Classification and definitions". *Paediatric Respiratory Reviews* 12.4 (2011): 230-237.

21. Steinhorn RH. "Neonatal pulmonary hypertension". *Pediatric Critical Care Medicine* 11 (2010): S79-S84.

22. Weber MA., *et al.* "Sudden unexpected neonatal death in the first week of life: Autopsy findings from a specialist centre". *Journal of Maternal-Fetal and Neonatal Medicine* 22.5 (2009): 398-404.

23. Champ CS and Byard RW. "Sudden death in asthma in childhood". *Forensic Science International* 66.2 (1994): 117-127.

24. Grabenhenrich LB., *et al.* "Anaphylaxis in children and adolescents: The European Anaphylaxis Registry". *The Journal of Allergy and Clinical Immunology* 137.4 (2016): 1128-1137.

25. Jortveit J., *et al.* "Sudden unexpected death in children with congenital heart defects". *European Heart Journal* 37.7 (2016): 621-626.

26. Komisar J., *et al.* "Impact of changing indications and increased utilization of fetal echocardiography on prenatal detection of congenital heart disease". *Congenital Heart Disease* 12.1 (2017): 67-73.

27. Sharland G. "Fetal cardiac screening and variation in prenatal detection rates of congenital heart disease: Why bother with screening at all?" *Future Cardiology* 8.2 (2012): 189-202.

28. Komisar J., *et al.* "Impact of changing indications and increased utilization of fetal echocardiography on prenatal detection of congenital heart disease". *Congenital Heart Disease* 12.1 (2017): 67-73.

29. Pinar H. "Postmortem findings in term neonates". *Seminars in Neonatology* 9.4 (2004): 289-302.

30. Mellander M. "Diagnosis and management of life-threatening cardiac malformations in the newborn". *Seminars in Fetal and Neonatal Medicine* 18.5 (2013): 302-310.

31. Cole CR and Eghtesady P. "The myocardial and coronary histopathology and pathogenesis of hypoplastic left heart syndrome". *Cardiology in the Young* 26.1 (2016): 19-29.

32. Shah S., *et al.* "A case report of meningococcal disease in a neonate". *Wisconsin Medical Journal* 112.1 (2013): 28-30.

33. Esposito S., *et al.* "Meningococcal B vaccination (4CMenB) in infants and toddlers". *Journal of Immunology Research* (2015): 402381.

34. Leestma JE. "Intracranial causes of death and their mechanisms". *Diagnostic Histopathology* 22.9 (2016): 327-332.

35. Milroy CM. "Sudden unexpected death in epilepsy in childhood". *Forensic Science, Medicine, and Pathology* 7.4 (2011): 336-340.

36. Morris JA., *et al.* "Sudden infant death syndrome and cardiac arrhythmias". *Future Cardiology* 5.2 (2009): 201-207.

37. Ladhani SN., *et al.* "Enter B and W: Two new meningococcal vaccine programmes launched". *Archives of Disease in Childhood* 101.1 (2016): 91-95.

38. The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. American Academy of Pediatrics Task Force on Sudden Infant Death Syndrome". *Pediatrics* 116.5 (2005): 1245-1255.

39. Mitchell EA., *et al.* "Changing infant's sleep position increases risk of sudden infant death syndrome. New Zealand Cot Death Study". *The Archives of Pediatrics and Adolescent Medicine* 153 (1999): 1136-1141.

40. Li DK., *et al.* "Infant sleeping position and the risk of sudden infant death syndrome in California, 1997-2000". *American Journal of Epidemiology* 157.5 (2003): 446-455.

41. Mitchell EA., *et al.* "Soft cot mattresses and the sudden infant death syndrome". *Zeitschrift für Medizinische Physik Journal* 109.1023 (1996): 206-207.

42. Hauck FR. "Changing epidemiology". In: Byard RW, Krous FR, editors. Sudden infant death syndrome. Problems, progress and possibilities. London: Arnold (2004): 31-35.

43. Passive smoking damages children's health". *Britton Journal Practitioner* (2010).

44. Carpenter RG., *et al.* "Sudden unexplained infant death in 20 regions in Europe: case control study". *Lancet* 363.9404 (2004): 185-191.

45. Ladhani SN., *et al.* "Two new meningococcal vaccine programmes launched". *Archives of Disease in Childhood* 101.1 (2016): 91-95.

46. Risk factors for SIDS. Mitchell EABMJ 339 (2009): b3466.

47. Simkiss DE., *et al.* "Airway obstruction by a child's pacifier--could flange design be safer?" *European Journal of Pediatrics* 157.3 (1998): 252-254.

48. Jorch G. "Prevention of SIDS". *Monatsschrift Kinderheilkunde* 151 (2003): 514-519.

49. Vennemann MM., *et al.* "Do immunisations reduce the risk for SIDS? A meta-analysis". *Vaccine* 25.26 (2007): 4875-4879.

50. Yikilkan H., *et al.* "Sudden infant death syndrome: how much mothers and health professionals know". *Pediatrics International* 53.1 (2011): 24-28.

51. Orch G. "Prevention of SIDS". *Monatsschrift Kinderheilkunde* 151 (2003): 514-519.

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