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Role of Belgian Outcome in Injury Score in Predicting Mortality in Burns

Jibetosh Biswas¹, Ravi Kumar Chittoria², Barath Kumar Singh. P³

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Abstract

Severe burn injury and its clinical outcome a major concern in prediction of mortality of patients. Multiple models have been formulated to overcome the concern related to this. Advancements in burn management over the years have significantly decreased burn mortality. But, implementing these advancements have become a huge concern in developing countries. Therefore, for routine evaluation and management to tackle mortality issue this scoring should be implemented at the earliest. There are various scoring systems that are formalized in predicting mortality in burns. In this article we would like to describe our study in using Belgian outcome in burn injury scoring system in predicting mortality in burns patients.

Keywords: Belgian outcome in burn injury (bobi); Score; Burn.

INTRODUCTION

Burn injuries are a global public health problem, responsible for an estimated 250,000 deaths per year.^{1,2} As a result, burn injury is one of the world's health issues contributing to the burden of disease and reported at about 180,000 deaths per year. Considering this problem, a management strategy predicting the prognosis of the condition of burn

patients is needed. Studies have indicated a strong link between burn size and mortality. Predicting the mortality in burns patient on presentation to hospital helps in determining the prognosis of the patient and effective handling of resources. Burns mortality depends on types of burn injury, total body surface area involved (TBSA) as well as certain demographic factors like age, gender, comorbidities. On assessing the clinical details and presumable outcome, risk stratification can be done from before, patient mortality can be reduced and infrastructure can be improvised accordingly. There are few scoring systems available for predicting the mortality in burn patients, but all of them are performed to a limited extent. In our study, we are assessing BOBI scoring system to predict mortality in burns

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Every year, more than 200,000 deaths occur because of diverse types of burns, and the majority of these deaths occur in low income and developing.

METHODS AND MATERIALS

This study was conducted in tertiary care centre in department of plastic surgery, JIPMER after getting the department ethical committee approval. Informed consent was obtained from patients. The subject was a 2 year old child who allegedly falls on hot water while playing at home and incurred burn injury on the right side of anterior chest, right arm, forearm. On examination she had 2nd degree burns with total of 15% surface area involvement (Fig. 1). On presentation, Belgian outcome in burn injury score was calculated based on 3 parameters (Table 1) - age (0 point), TBSA (0 point), inhalation injury (0 point), with a predicted mortality of zero percent. Patient burn wound bed preparation done with Autologous platelet rich plasma (APRP), collagen scaffold dressing. Patient underwent Wound debridement, tangential excision followed

by split skin grafting and discharged successfully at 4 weeks.



Fig. 1: 20% burns over chest, abdomen and right arm

Table 1: Belgian outcome of burn injury score

	0	1	2	3	4
Age (years)	<50	50-64	65-79	>80	
TBSA (%)	(20	20-39	40-59	60-79	>80
Inhalation injury	No			Yes	
Total Score					
	0	1	2	3	4
Mortality Prediction	0.1	15	5	10	20
	30	50	75	85	95
	50	75	100	115	120

BOBI scoring system for predicting mortality in burn patients

RESULTS

BOBI score came to a total of 0 point. On using mortality prediction risk of belgian outcome in burn injury (BOBI) scoring result came to be of 0.1% with very less threat to life. Intra-operative and post-operative periods were uneventful for the patient. The raw area took up the split thickness skin graft well and burn wounds healed well and patient discharged successfully (Fig. 2). There were no complications.



Fig. 2: Burn wounds healed well at the time of discharge

DISCUSSION

Several prognostic indices for burn injuries have been created over time. But, none of them could accurately formulate to tackle the major concern faced by burn patients. Even though advancements in burn injury management have significantly reduced the burn mortality, due to limited resources burn mortality is still high in developing countries. Every year in India, around 10,00,000 people sustain moderate to severe burns.^{3,4} The first prognostic factors found to be effective in predicting the mortality in patients with burns was the Total surface area (TBSA) and age, which was first proposed by Weidenfeld, who in 1902 correlated TBSA and age with the mortality. The effectiveness of these two parameters was affirmed later 1949 by Bull and Squire in 1949 and later by Baux in 1963 as Baux score.⁵ Abbreviated Burn Scoring Index (ABS), Ryan et al., Belgian Outcome in Burn Injury (BOBI), Smith et al., McGwin et al., are some of the scoring systems available which can be used to predict the mortality in burn patients. However, this was highly limited in application as several factors

modify survival probability. The BOBI score uses values of age, TBSA and presence of inhalational injury. The maximum score is 10 which give a 99% risk of mortality. In our study, the patient's BOBI score was 0 with a mortality risk of 0.1%. In our case study, Belgian outcome in burn injury (BOBI) was a good indicator of mortality prediction as the patient was vitally and nutritionally stable at the time of discharge. Hence, Belgian outcome in burn injury (BOBI) scoring is a reliable predictor of mortality in the patient with burns evaluated in our case study.

CONCLUSION

The study shows that Belgian outcome in burn injury (BOBI) score can be used as a mortality predictor of burn patient and help in treating the patients for the best use of resources available in developing countries like India. It consists of

three parameters which was easy to calculate and reproducible by any triage system.

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Respiratory Distress in Neonates in A Tertiary Care Medical College Hospital: A Cross-Sectional Study

Sakshi Satish Rane¹, Abhijeet Shinde², Sunil Natha Mhaske³

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Abstract

Introduction: Neonatal Respiratory Distress (NRD) is the most frequent reason for admissions to NICUs. Studies on this topic are limited in our setup and mostly focused on finding the incidence of NRD. The aim of this study was to find the incidence and the most common causes of neonatal respiratory distress in our setting to better prepare treatment protocols and improve overall outcomes.

Methodology: A cross-sectional study was conducted in a tertiary care medical college hospital from February 2022 to October 2022. Data of all neonates admitted with respiratory distress during this term was collected and analyzed.

Results: A total of 550 neonates were admitted to the NICU out of which 207(37.6%) developed respiratory distress. The most common cause of NRD was Respiratory Distress Syndrome 129 cases (62.3%) followed by Pneumonia 25 cases (12.1%), Birth Asphyxia 21 cases (10.1%) and Sepsis 17 cases (8.21%). The highest mortality was observed in cases with RDS (8.35%).

Conclusion: The study highlights the importance of NRD which has a high incidence rate in NICUs and a mortality rate of 7.2%. The most common causes are also preventable and treatable, and suggestions have been made to help reduce incidence and mortality and improve outcomes.

Keywords: Common Causes; Neonates; Respiratory Distress.

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INTRODUCTION

While the first cry of a newborn is an important indicator of neonatal well-being and adaptation to extra-uterine life, respiratory distress is one of the common reasons for admissions to the Neonatal Intensive Care Unit (NICU) within a few hours after birth.¹ The incidence of neonatal respiratory distress (NRD) in developing countries like India ranges from 0.7% to 8.3%.² Respiratory distress is recognized by signs of increased work

of breathing like: tachypnea, chest retractions, nasal flaring, wheezing, grunting, stridor, cyanosis etc.³ A number of maternal, foetal and obstetric factors are implicated in the development of respiratory distress. The most common causes according to most literature include: respiratory distress syndrome or hyaline membrane disease (RDS or HMD), transient tachypnea of the newborn (TTN), meconium aspiration syndrome (MAS), birth asphyxia, pneumonia, pneumothorax, and congenital malformations etc.^{4,5} Risk factors for NRD include: low birth weight, prematurity, caesarean section, gestational diabetes to name a few.^{6,7} Regardless of the cause of respiratory distress, it is responsible for a significant amount of preventable morbidity and mortality in neonates if not detected and treated early.⁸ Studies on neonates admitted with respiratory distress in NICUs in Maharashtra are limited. This study was conducted in a tertiary care medical college hospital in western Maharashtra, to evaluate the common neonatal causes of respiratory distress in our setting to help improve further outcomes and reach closer to achieving our millennium development goal of reducing neonatal deaths in high mortality countries.

METHODOLOGY

This was a retrospective, descriptive, cross-sectional study carried out from February 2022 to October 2022 at a tertiary care medical college hospital.

The study included all neonates admitted to the NICU with signs of respiratory distress during the study period.

Informed consent was obtained from parents of all babies enrolled in the study.

The study excluded neonates whose parents/guardians refused to participate in the study or if any information required for data collection could not be obtained.

Simple convenience non-probability sampling technique was used for data collection.

The cases were diagnosed clinically by the presence of at least 2 of the following criteria: respiratory rate (RR) of 60 breath/ min or more, subcostal indrawing, xiphoid retraction, suprasternal indrawing, flaring of alae nasi, expiratory grunt and cyanosis at room air.

They were also assessed by scoring systems using Silvermen Anderson Scoring system for preterm babies and Downe's Scoring system for

term babies.

Data was collected for each patient using hospital records. Neonatal demographic and clinical data was collected under the following headings in a pre-structured proforma: name, age at admission, sex, body weight, singleton or multiple births, date of admission and date of discharge or death. All were weighed using an electronic scale and classified to small for gestational age (SGA) ≤ 2.5 kg, large for gestational age (LGA) ≥ 4 kg and normal weight group (NGA)(2.5-4 kg). The relative investigations were done and treatment as per unit protocol was followed. All neonates were examined daily till discharge from hospital or death.

The prevalence of respiratory distress varies with gestational age: 30.0% among preterm, 20.0% among post terms to 4.0% in term babies. Assuming prevalence of respiratory distress as 16% and 95% confidence interval with 5% margin of error, sample size was calculated using formula $N = z^2 pq/d^2$ where, p= prevalence of respiratory distress in newborns. q = 1-p, N= sample size, z=1.96, d= maximum tolerable error. Estimated sample size was 207.

All the data was tabulated in Microsoft Excel and Statistical analysis was done using SPSS program (version 20). Categorical data are expressed as frequency and percentage. Continuous data (if any) are expressed with mean and standard deviation. Chi-square test was used to compare two categorical data. A P-value of <0.05 was considered statistically significant.

Ethical clearance was obtained from the institutional ethics committee before starting the study.

RESULTS

Out of 550 NICU admissions during the study period, 37.6% (207) neonates were admitted for respiratory distress. Our study comprised of 58.9% (122) males and 41.1% (85) females. The male:female ratio was 1.4:1. 61.4% (127) neonates were full term while 38.6% (80) were preterm. 72.9% (151) babies were in the normal birth weight group (2.5-4kg) and 27.1% (56) were small for gestational age (SGA) (≤ 2.5 kg). There were no post-term admissions or large for gestational age (LGA) (≥ 4 kg) babies in our study. 94.2% (195) were singleton births whereas there were 12(5.80%) twin births.

88.36% (183) of the cases were of respiratory origin, whereas the other 11.64% (24) had a non-respiratory origin.

The commonest cause of neonatal respiratory distress was Respiratory Distress Syndrome (RDS): 62.3% (129), followed by Pneumonia: 12.1% (25), Birth Asphyxia: 10.1% (21) and Sepsis 8.21% (17). Bronchiolitis comprised 3.86% (8) of the cases and Congenital Heart Diseases (CHD): 3.38% (7).

44.9% (58) of the RDS cases were born preterm and 30.2% (39) were SGA, while 6.2% (8) out of them were twins. 36% (9) of the cases of Pneumonia were preterm births, with 28% SGA babies. 71.4% (15) of Birth Asphyxia 70.6% (12) of Sepsis cases were full

term births with 76.2% (16) and 82.3% (14) of them being of normal birth weight respectively. No case of Bronchiolitis were preterm, SGA or twins. 28.6% (2) of CHD cases were preterm, while 28.6% (2) of them were SGA and 1 (14.3%) was a twin birth. All cases showed a male preponderance.

92.8% (192) were treated and subsequently discharged while 7.2% (15) died. Maximum mortality was seen in cases with Respiratory Distress Syndrome (RDS) 8.35%.11

Table 1: Features associated with common causes of Neonatal Respiratory Distress

Diagnosis	Sex		Term		Weight		Singleton/Twin		Total
	Male	Female	Full Term	Preterm	NGA	SGA	Singleton	Twin	
RDS	76 (58.9%)	53 (41.1%)	71 (55.1%)	58 (44.9%)	90 (69.8%)	39 (30.2%)	121 (93.8%)	8 (6.2%)	129 (62.3%)
Pneumonia	15 (60%)	10 (40%)	16 (64%)	9 (36%)	18 (72%)	7 (28%)	24 (96%)	1 (4%)	25 (12.1%)
Birth Asphyxia	12 (57.1%)	9 (42.9%)	15 (71.4%)	6 (28.6%)	16 (76.2%)	5 (23.8%)	20 (95.2%)	1 (4.8%)	21 (10.1%)
Sepsis	10 (58.8%)	7 (41.2%)	12 (70.6%)	5 (29.4%)	14 (82.3%)	3 (17.7%)	16 (94.1%)	1 (5.9%)	17 (8.21%)
Bronchiolitis	5 (62.5%)	3 (37.5%)	8 (100%)	0 (0%)	8 (100%)	0 (0%)	8 (100%)	0 (0%)	8 (3.86%)
CHD	4 (57.1%)	3 (42.9%)	5 (71.4%)	2 (28.6%)	5 (71.4%)	2 (28.6%)	6 (85.7%)	1 (14.3%)	7 (3.38%)
Total	122 (58.9%)	85 (41.1%)	127 (61.4%)	80 (38.6%)	151 (72.9%)	56 (27.1%)	195 (94.2%)	12 (5.8%)	207 (100%)

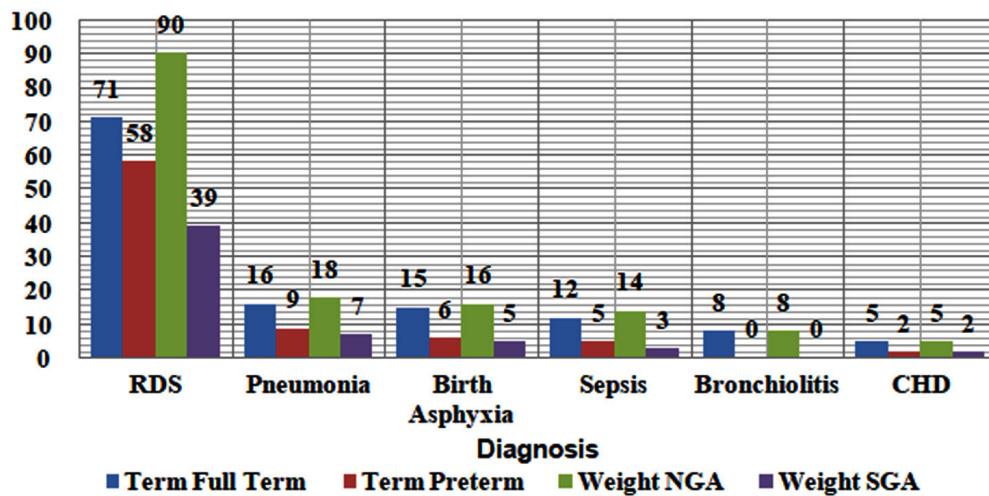


Chart 1: Features associated with the common causes of Neonatal Respiratory Distress

Table 2: Outcome of various causes of Neonatal Respiratory Distress

Diagnosis	Outcome		Total
	Discharged	Death	
RDS	118 (91.5%)	11 (8.35%)	129 (62.3%)
Pneumonia	23 (92%)	2 (8%)	25 (12.1%)
Birth Asphyxia	20 (95.2%)	1 (4.8%)	21 (10.1%)
Sepsis	16 (94.1%)	1 (5.9%)	17 (8.21%)
Bronchiolitis	8 (100%)	0 (0%)	8 (3.86%)
CHD	7 (100%)	0 (0%)	7 (3.38%)
Total	192 (92.8%)	15 (7.2%)	207 (100%)

DISCUSSION

While respiratory distress remains one of the most common cause of admissions to the NICU according to most studies^{9,10}, it has not been extensively studied in context of India despite its prevalence and mortality.¹¹ Very limited studies have tried to find out the common causes of neonatal respiratory distress in settings similar to ours. Respiratory distress is often associated with neonatal mortality which is an important chunk of under-five mortality. While infant mortality rates have decreased, neonatal death rates have remained almost the same.² Knowledge of the causes of respiratory distress is important for preparing protocols for treating neonates presenting with respiratory distress, especially in low resource settings, and for arranging basic treatment facilities to help improve outcomes.

Our study found the incidence of neonatal respiratory distress among hospital admissions to be 37.6%, which is similar to admissions rates of NRD reported in other studies.^{12,13} The most common cause of neonatal respiratory distress in our study was Respiratory Distress Syndrome (62.3%), followed by Pneumonia (12.1%), Birth Asphyxia(10.1%) and Sepsis(8.21%).

Respiratory distress syndrome was the most common cause of respiratory distress in our study, following the trends seen in other studies^{14,15} and 44.9% cases were preterm and 30.2% were SGA. It further verifies findings of other studies which state that frequency of RDS is increased with low birth weight and prematurity.^{16,17} Further highest mortality was found amongst RDS cases with 8.35%. While mortality due to RDS has reduced due to surfactant therapy¹⁸, we did not have facilities for the same at our institute. However, we can aim to provide good antenatal and obstetric care to help reduce preterm births and get better results.

Pneumonia was the second common cause of respiratory distress with 12.1%, as also shown by Dutta et al with 24.3% incidence¹⁹, however, a paper by Mathur et al. reported pneumonia to be the most common cause with 68.7% incidence.²⁰

Birth asphyxia was seen in 10.7% of neonates in a study done by Prakash et al²¹, very similar to our incidence rate (10.1%). Sepsis constituted 8.21% cases in our study, while much higher incidence has been noted in other studies.^{9,11}

Bronchiolitis was seen in 3.86% cases, which has not been previously reported in other studies. Congenital heart disease was seen in 3.38% of our

cases, while almost double incidence has been reported in other studies.^{11,22}

This study showed a male preponderance for respiratory distress in line with findings of other studies^{23,24,25}. However, no plausible explanation could be found for the same. Our study did not have any neonate who was post-term or large for gestational age (>4kg).

The results show that neonatal respiratory distress associated with RDS, Pneumonia, Birth Asphyxia and Sepsis is quite common, and as these causes are either preventable or treatable, the overall burden of NRD and associated mortality can be greatly reduced. More emphasis should be laid to deal with the cause that is the most prevalent and also one that causes maximum mortality, as tackling them on a priority basis will vastly help improve our overall outcomes.

CONCLUSION

Respiratory distress was the most common cause of admission in our NICU and consisted of 37.6% of total admissions, during the study period. The most frequently encountered cause was Respiratory Distress Syndrome (62.3%), followed by Pneumonia (12.1%), Birth Asphyxia (10.1%) and Sepsis (8.21%). In our setup, the outcomes were fairly good with a 92.8% cure rate and 7.2% mortality rate.

RECOMMENDATIONS

Efforts need to be made to further lower the mortality rate by providing good antenatal care to decrease the incidence of premature labor, or administration of steroids to the mother in anticipated premature deliveries and use of surfactant immediately after delivery for all prematurely born infants. Also, NICU personnel need to be trained to identify early signs of respiratory distress and provide neonatal resuscitation and initiate treatment soon after. This will help in decreasing the incidence and mortality associated with neonatal respiratory distress.

LIMITATIONS

Our study was conducted for a short time period, had a limited sample size and was a hospital based study. Further, we have not included maternal antenatal and obstetric history in our study. So, more extensive studies need to be conducted on a larger scale to substantiate our findings. However,

this study provides a good idea about the common causes of respiratory distress in neonates in a tertiary care setup of western Maharashtra.

Conflict of interest: None.

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Osteogenesis Imperfecta: A Rare Case Report

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Abstract

Introduction: Osteogenesis Imperfecta also known as brittle bone disease is a heterogeneous disorder which is rare and characterized by bone fragility, multiple fracture, bone deformity and short stature.¹ It has varying degree of classification based on varying degree of fragility and various clinical presentations.² Osteogenesis imperfecta (OI) is a rare skeletal dysplasia, with an incidence of 1/15,000–20,000.³

Case report: A 7-month-old male child visited our hospital for fever cough, cold & breathlessness. Ultrasonography at 8th month revealed mild polyhydramnios with Fetal growth retardation and shortening of fetal long bones of upper and lower limb & 9th month Ultrasonography revealed moderated Polyhydramnios with short limb Dwarfism. On head to toe examination, patient had triangular shaped face with broad forehead, blue sclera, short neck short statured limbs with cylindrical like appearance that is circumferential fat pads, both the hips and knees were flexed and rotated inwards. Barrel shaped rib cage was present.

Discussion: OI, commonly known as brittle bone disease, is a hereditary ailment that includes a diverse range of illnesses. It is characterised by a propensity for bone fractures, which can range in severity from a minor break to a prenatal fracture. Blue sclera, DI, hyperlaxity of ligaments and skin, hearing impairment, small height, and bone abnormalities are further characteristic clinical symptoms.¹¹ Sillence et al categorised OI into four kinds based on their clinical severity and genetic characteristics because OI is a diverse disease with different clinical presentations.¹²

Conclusion: In conclusion, experimental treatments including gene based therapy and bone marrow and stem cell transplantation offer prospective treatments for OI. But these methods are not yet prepared for clinical testing.¹⁵ There should be availability of these approaches in each tertiary care center for better diagnosis & treatment of osteogenesis imperfecta.

Keywords: Osteogenesis; Brittle bone; Multiple fracture; Short stature; Blue sclera.

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INTRODUCTION

Osteogenesis Imperfecta also known as brittle bone disease is a heterogeneous disorder which is rare and characterized by bone fragility, multiple fracture, bone deformity and short stature.¹ It has varying degree of classification based on varying degree of fragility and various clinical presentations.² Osteogenesis imperfecta (OI) is a rare skeletal dysplasia, with an incidence

of 1/15,000–20,000.³ As the production of type I collagen in various tissues is impaired, individuals with OI may also suffer from other clinical symptoms such as brittle teeth, blue sclerae, hearing loss, reduced respiratory function, and cardiac valvular regurgitation.⁴

The severity of OI varies from mild to extremely severe, with the most severe form being perinatally lethal.⁵ Five clinical forms of OI are identified by the revised Nosology and Classification of Genetic Skeletal Disorders: OI type I, which is non-deforming with persistently blue sclera; type II, which is perinatally lethal; type III, which is gradually deforming; type IV, which is moderate; and type V, which has calcification of the interosseous membranes and/or hypertrophic callus.⁶ Among patients who survive infancy, those with OI type III are the most seriously affected, with many fractures, scoliosis, small stature, and limited mobility. OI kind I possess the most modest phenotype. X-linked, dominant, or recessive inheritance patterns exist for OI.^{7,8} Most frequently, pathogenic mutations in either COL1A1 or COL1A2 produce the dominant illness (encoding components of type I collagen).

Null alleles (i.e. deletions, splice variants that change the reading frame, or variations that are truncating) in COL1A1 result in haploinsufficiency that is typically associated with mild OI (type I). COL1A1 or COL1A2 missense mutations, which commonly result in glycine substitutions in a Gly-X-Y repeat, or splice mutations, which do not alter the reading frame, typically cause deadly, severe, or moderate OI (categories II, III, or IV, respectively). An OI type V recurrent pathogenic mutation in the 5' untranslated region of IFITM5, which codes for the interferon induced transmembrane protein 5 (BRIL), causes the dominant form of the disease, which is rarer.⁹ There is currently no known cure for OI, and available therapies do not deal with the underlying molecular illness.

The purpose of treatment is to retain mobility, encourage normal function, increase overall bone strength to prevent fractures, and enhance quality of life. Physiotherapy is used to build up the muscles and increase mobility, and lifetime orthopaedic procedures like rods in the long bones to rectify bone abnormalities are used to achieve this goal. Bisphosphonates are prescribed to treat osteoporosis and boost bone mineral density. The impact of bisphosphonates on OI has been debated, and a recent systematic review found that nonrandomized open label uncontrolled studies show that oral and intravenous bisphosphonate

administration objectively improved function and mobility while randomised controlled trials did not show a significant improvement in function and mobility with oral bisphosphonate administration.¹⁰

CASE REPORT

A 7-month-old male child visited our hospital for fever cough, cold & breathlessness. The mother had non consanguineous marriage with 6 pregnancies out of which 2 were aborted and live births were 4. She was on calcium and iron supplements at the time of Pregnancy. No history of taking teratogenic drugs. No any illness to mother during pregnancy. She also had history of Polyhydramnios during the delivery. Ultrasonography at 8th month revealed mild polyhydramnios with Fetal growth retardation and shortening of fetal long bones of upper and lower limb & 9th month Ultrasonography revealed moderated Polyhydramnios with short limb Dwarfism. Baby was born full term normal delivery and cried immediately after birth. Birth weight was 2.5 kg & other anthropometry of at birth was not recorded. Now head circumference is 39 cm, chest circumference 33 cm and length from heel to crown was 45 cm. So patient had microcephaly & severely stunted. He was normally breastfed and had no history of NICU stay. Mild tachypnoea was present & rest vitals were stable. On auscultation of both lungs, crepts were heard, mild hypotonia was present & other systemic examination was normal. X ray lungs showed pneumonia. Patient also had history of recurrent respiratory tract infections.

On head to toe examination, patient had triangular shaped face with broad forehead, blue sclera, short neck short statured limbs with cylindrical like appearance that is circumferential fat pads, both the hips and knees were flexed and rotated inwards. The baby also had long flexible fingers and toes. Barrel shaped rib cage was present

All routine investigations sent & were normal. In order to confirm diagnosis, genetic sequencing of the most common problematic genes, COL1A1, COL1A2, and IFITM5 was planned but patient was unaffordable.

Calcium, phosphorous & vit D levels were low for which appropriate supplements started.

Limb physiotherapy was started & advised to continue after discharge.

Patient was given IV antibiotics, oxygen by nasal prongs, nebulization & patient responded to treatment for pneumonia. At last patient was referred to higher centre for further management of

osteogenesis imperfecta.



Picture 1: Osteogenesis imperfecta patient showing BLUE SCLERA



Picture 2: Osteogenesis imperfecta patient showing *Barrel Shaped Chest*

DISCUSSION

OI, commonly known as brittle bone disease, is a hereditary ailment that includes a diverse range of illnesses. It is characterised by a propensity for bone fractures, which can range in severity from a minor break to a prenatal fracture. Blue sclera, DI, hyperlaxity of ligaments and skin, hearing impairment, small height, and bone abnormalities are further characteristic clinical symptoms.¹¹ Sillence et al. categorised OI into four kinds based on their clinical severity and genetic characteristics because OI is a diverse disease with different clinical presentations.¹² Type I, mild nondeforming; type II, prenatal deadly; type III, severely deforming; and type IV, moderately deforming were the categories used. However, more OI cases have since been discovered and looked into. As a result, OI categories V through VIII have been added to the original Sillence classification based on the unique clinical, radiological, and molecular aspects.¹³ There are four of these: type V, which is moderately deforming and has normal teeth and sclera; type VI, which is moderately ill and has a fishscale pattern of bone lamellation, normal sclera, and teeth; type VII, which is clinically similar to type II but the patients have a smaller head and normal sclera; and type VIII, in which the patients have defects in growth and mineralization.¹⁴ Type I collagen, a main and predominate extracellular

matrix protein in bone tissues, is synthesised with a quantitative or qualitative deficiency, causing OI, an autosomal dominant condition.¹⁵ The mutation could be in either of the two COL1A1 or COL1A2 genes, which produce the pro-I or pro-II chains of type I collagen, respectively.¹⁶ However, depending on which chains are impacted, where in the collagen structure the mutation occurs, and the type of amino acid substitute used, different phenotypes are produced.¹¹ In this instance, the patient had a history of recurrent respiratory tract infections, which could range in severity from mild symptoms to life-threatening discomfort. The physical examination revealed a triangular shaped face, blue sclera, and hypermobile joints.

DNA or collagen protein analysis can be used to confirm an OI diagnosis, but in many cases, the presence of other clinical signs including blue sclera and the occurrence of bone fractures with little damage is enough to make the diagnosis. A multidisciplinary team evaluation of the patient is required following the confirmation of the OI diagnosis. The cornerstones of OI management include orthopedic surgery, physical therapy, and rehabilitation. In this example of mild type I OI, the aim was also to provide a normal quality of life for the patient. The purpose of multimodality therapy is to maximise the mobility and functional capabilities of patients.¹⁶ For the treatment of all forms of OI, oral and intravenous bisphosphonates (BPs) and strong antiresorptive medicines are frequently used. Although BPs cannot treat OI, they are a useful supplement to complete therapy. The best way to utilise BPs, whether those with lesser symptoms should use them, and any negative effects are still unknown. To improve their therapeutic applicability, a sizable randomised double blind placebo controlled experiment is needed. There are other medicinal treatments available, like GHs and PTHs, but their results and side effects need to be assessed and analysed further.¹⁵ When medicinal treatments are ineffective, surgical surgery is a backup plan. No surgical intervention was necessary in the present case because no abnormalities were seen and the response to pamidronate was positive. In order to avoid contractures and immobility related bone loss, it was also essential to implement monitored, moderate physical activity regimens after the patient was discharged from the hospital.¹⁷

CONCLUSION

In conclusion, experimental treatments including gene based therapy and bone marrow and stem cell

transplantation offer prospective treatments for OI. But these methods are not yet prepared for clinical testing.¹⁵ There should be availability of these approaches in each tertiary care center for better diagnosis & treatment of osteogenesis imperfecta.

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Reactivation at BCG Vaccination Site in a Case of Multisystem Inflammatory Syndrome in Children

Praveen Unki¹, Sujay Rangaswamy², Sri Raksha Satya³, Shreyas Vishwanath⁴

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Abstract

Background: Kawasaki disease is a well known entity in pediatric age group and so is its counterpart Multisystem Inflammatory Syndrome in Children (MIS-C). MIS-C term became more popular during covid era as the infection started to affect pediatric age group. Now, a large number pediatricians have seen cases of MIS-C. Reactivation of BCG scar is less commonly seen in Kawasaki disease and rare to never in a case of MIS-C.

Case Presentation: We report a 6-month-old male child who presented with cold and fever. Covid Rapid Antigen Test (RAT) and Covid RTPCR were negative. He continued to have high grade fever which is unusual for a case of bronchiolitis and started on antibiotics. Clinical suspicion of MIS-C made and started to work up for the same. Examination revealed reactivation at BCG site with peeling of skin. Further investigations showed elevated CRP and d-dimer with positive anti-SARS COV2 antibody. Diagnosis of MIS-C confirmed and treated with methylprednisolone and IVIG.

Conclusion: Reactivation at BCG site is well known entity in Kawasaki disease and as a post vaccination immune cross reactivity. But never reported in a case of MIS-C as less known about human immunological response towards Covid-19 infection and its sequel. It can be considered as an early diagnostic tool in resource poor settings and at community level.

Keywords: Multisystem Inflammatory Syndrome in Children; BCG scar; IVIG; Methylprednisolone.

INTRODUCTION

Pandemic of Covid 19 had created chaos all over the world. It did not spare any age group

from its devastating effects. It was more deadly initially in high-risk groups and old ages with little or no effects among children. At first it was a new infection and its behaviour, early and late effects were not known. As time passed, we could be able to appreciate spectrum of manifestation of the Covid infection. Protocols were made to curtail the infection and so for its early detection and management. Research on drugs that are effective in treating Corona infection started and various drugs were proposed to be effective. However, only a countable number of drugs showed benefits. Covid-19 even had varied effects on the foetus of

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Covid infected mother such as intrauterine death, hydrops fetalis, congenital covid infection and so on. As children started infecting with Corona virus, we could see a variety of presentation from asymptomatic to devastating complication of the infection so called Multisystem Inflammatory Syndrome in Children (MIS-C).¹ Reactivation of the BCG scar has been described in children during viral infections and following influenza vaccination, but is mostly associated with Kawasaki's disease, a disease entity with pathogenesis likely similar to the Covid-19 complication, MIS-C.² We present a case of MIS-C with reactivation at BCG site.

CASE PRESENTATION

A 6-month-old male child presented with history of cold and fever of 3 days duration. Child was admitted in view of tachypnoea and decreased air entry over left mammary region. Chest X-ray was suggestive of bilateral diffuse haziness with hyperinflation suggesting bronchiolitis as in fig.



Fig. 1: Chest X-ray suggestive of Bronchiolitis.



Fig. 3: Healing at BCG reactivation site 1 week after IVIG treatment.

1 and hence initial diagnosis of bronchiolitis was made and started on humidified oxygen. Complete hemogram was done suggestive of anemia (Hb-9.2g/dl) with elevated total leukocyte count and normal platelet count (Table 1). Child continued to have fever spikes of 101 to 103. Dengue serology (NS1 antigen, IgM and IgG), Rapid malarial antigen test, peripheral smear for malaria parasite, Covid RAT and Covid RTPCR were negative. All possible causes of acute febrile illness were ruled out and started on inj. Amoxicillin and clavulanic acid. Blood culture and Urine culture were sterile without any bacterial growth. Child continued to have persistent fever and had 2 episodes of vomiting. Child developed erythema at BCG site with peeling of skin. Child had 2 system involvement with fever for 5 days and elevated CRP and D-dimer levels (Table 1). Hence, Possibility of MIS-C considered and level for anti-SARS COV2 antibody sent.³ 2D Echocardiogram was done which was suggestive of small ASD with normal coronary vessels. ECG was normal with negative Trop I level. Child was



Fig. 2: BCG site reactivation.

started on inj. Methylprednisolone (2mg/kg/day) in view of strong evidences in favour of MIS-C (reactivation at BCG site along with other criteria) along with inj. Low molecular weight (LMWH) (2mg/kg/day) and aspirin (5mg/kg/day). Child started responding and temperature returned to baseline. On receiving Anti-SARS COV2 antibody levels (29.6U/ml), diagnosis of MIS-C confirmed.³ Patient was started on IVIG 2g/kg. Inj. Methylprednisolone was changed to equivalent dose of oral prednisolone and tapered slowly over

5 days and repeat D-dimer was 435 ng/ml at the time of discharge. Patient was advised to follow up for 2D echocardiogram.

DISCUSSION

Infections with SARS-CoV-2 in pediatric age

Table 1: Investigations with normal reference range.

Parameter	Observed value	Reference value
Hemoglobin	9.2 g/dl	11.1-14.1 g/dl
Leukocyte count	15620 cells/cumm	6000 - 18000 cells/cumm
Platelet count	3.58×10 ⁵ /cumm	2-5.5×10 ⁵ /cumm
CRP	42.8 md/l	0-10 mg/dl
ESR	56 mm/ at 1hour	2-15 mm/ at 1 hour
PT	12 seconds	11-16 seconds
aPTT	26 seconds	26-36 seconds
INR	0.8	-
BUN	15 mg/dl	15-50 mg/dl
Creatinine	0.4 mg/dl	0.4-14 mg/dl
Blood culture	Negative	-
Urine culture	Negative	-
D-dimer	1345 ng/ml	Up to 500 ng/ml
Urine Routine and microscopy	Within normal limits	-
Anti-SARS COV-2 antibody	29.6U/ml	< 0.80 U/ml (negative) ≥ 0.80 U/ml (positive)

group are usually mild, but there may be dreaded complications associated with post infection inflammatory disorder, which can lead to serious short term and long term consequences, referred to here as MIS-C. Most cases of MIS-C associated with COVID-19 are treated following the standard protocols for Kawasaki disease. Dufort *et al.* highlight that the incidence of MIS-C was 2 per 100,000 in children and young adults of less than 21 years of age.¹ MIS-C is equivalently severe to Kawasaki disease, based on six main diagnostic elements: pediatric age, persistent fever, presence

of laboratory inflammatory markers (ESR, CRP), signs or symptoms of organ dysfunctions, without any alternate diagnosis, and history of COVID-19 infection or exposure with possible cardiac involvement.³ World Health Organization and the Centers for Disease Control and Prevention (CDC), have defined the diagnostic criteria of MIS-C.(Table 2)

The presence of coronary aneurysm is a serious complication, well known in Kawasaki disease, and present in MIS-C. Coronary artery inflammation

Table 2: Multi-system Inflammatory Syndrome in Children (MIS-C): WHO Definition³

0-19-years-old child with fever >3 days

And: Two of the following:

- 1) Rash or bilateral non-purulent conjunctivitis or muco-cutaneous inflammation signs (oral, hands or feet).
- 2) Hypotension or shock
- 3) Features of myocardial dysfunction, pericarditis, valvulitis, or coronary abnormalities (ECHO findings or elevated Troponin/NT proBNP)
- 4) Evidence of coagulopathy (by PT, PTT, elevated d-Dimers)
- 5) Acute gastrointestinal problems (diarrhoea, vomiting, or abdominal pain)

And Elevated ESR, C-reactive protein or Procalcitonin

And no other obvious microbial cause of inflammation, including bacterial sepsis, staphylococcal or streptococcal shock syndromes.

And Evidence of COVID-19 (RT-PCR, antigen test or serology positive), or likely contact with patients with COVID-19

will progress to aneurysm which acts as a nidus for thrombus formation with partial or complete artery block resulting in myocardial infarction, and cardiac arrhythmias. The timely diagnosis and treatment with Intravenous Immunoglobulins (IVIG) until the end of first week of the disease reduced the possibilities of these complications.⁴ The child described in this case report had MIS-S without heart involvement as confirmed by ECG, echocardiography and acute cardiac markers and was treated using steroid and IVIG.

In countries like India where tuberculosis is still a major public health issue, BCG is recommended to all neonates at birth or as soon thereafter as possible. In countries with low incidence of TB (European countries) BCG vaccine is only given to high-risk groups. Up to 97% of people who receive BCG vaccine experience a skin reaction at the injection site 2 to 4 weeks after vaccination.^{5,6} The reaction heals within 2 to 5 months with a scar of approximately five mm in diameter. Several research studies concluded that BCG at birth is associated with a 30% to 50% reduction in neonatal mortality.^{2,7} BCG also gives protection against sepsis and respiratory infections.⁷

Reactivation at BCG site has also been witnessed in children during viral infections such as measles and human herpes virus type 6.^{8,9} However, reactivation of BCG scars has commonly associated with Kawasaki disease (KD), and suggested by many pediatricians as a diagnostic tool for KD.¹⁰ A study from Singapore, where BCG is universally given at birth, reported that 43% of patients with KD had reactivation at the BCG scar site.⁹ The study indicated that BCG site reactivation was in direct correlation with time gap between BCG vaccination and onset of KD. Shorter the duration more chances of reactivation.⁹ This could possibly explain why we only observed reactivation in the most recent BCG scars. As expected, MIS-C had behaved similar to KD in many aspects from clinical and laboratory diagnostic criteria to less common manifestations such as BCG scar reactivation.¹¹ However, the pathophysiology of MIS-C is still unknown.¹¹ The local reaction was attributed to cross-reactivity between BCG microbial components persisting at the site of vaccination and SARS-CoV-2 vaccines. In favour of the above proposed hypothesis there are evidence of eight BCG derived peptides with significant sequence homology to either SARS CoV-2 non-structural protein 3 (NSP3) or non-structural protein 13 (NSP 13) derived peptides were recently identified.¹² Similarly, reactivation of the BCG scar experienced by two health care workers after

mRNA vaccination might therefore have been caused by an immunological reaction due to the cross reactivity between BCG and SARS-CoV-2.¹³ This phenomenon in KD has been attributed to immunological cross-reactivity of mycobacterial heat shock protein (HSP) 65 with human homologue HSP 63.5. Likely mechanism may be proposed in our case as a cause for reactivation at BCG site in a case of MIS-C.

CONCLUSION

Any child with irritability and persisting fever (>3 days) not responding to antipyretics should be suspected to have MIS-C in this Covid era. All criteria need not be fulfilled; incomplete MIS-C may be present similar to incomplete Kawasaki disease.⁴ In view of risk higher incidence of coronary involvement in infancy, an early diagnosis and prompt treatment are essential. Erythema at the site of BCG inoculation is rare, but it is a specific sign of Kawasaki disease and so seems to be with MIS-C. Hence, it can be used as a tool for an early diagnosis in places where it takes time to get investigations report and in resource poor settings. Children have been diagnosed early by looking at the BCG scar on admission like its counterpart Kawasaki disease.¹⁴ This should be particularly useful in countries where BCG vaccination is universal.

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Introduction

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Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. *J Oral Pathol Med* 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone-iodine antisepsis. State of the art. *Dermatology* 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. *Applied logistic regression*, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O,

Kidd EAM, editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. pp 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979–2001. www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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