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# The Journal of Ad-din Women's Medical College

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Each manuscript requires an unstructured abstract that should include objective, methods, results, conclusion and key words in not more than 150 words for any review article or case report and 250 words for structured abstract only for original article summarizing the significant information and findings. Authors must give two to five key words identifying the most important topics covered by the manuscript. Abbreviations, diagrams, and references in the abstract should be avoided.

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The body of the manuscript/text should be divided into the following sections: i) Introduction, ii) Materials and Methods, iii) Results (include tables and diagrams), iv) Discussion, v) Conclusion, and vi) Acknowledgement if any (particularly on funding, study subjects and co-author).

**Introduction**

It includes a short yet robust background purpose and the rationale for the study (or summarized observation), including pertinent references, but data or conclusion from any work should not to be included.

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**Acknowledgements**

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Acknowledgement for funding, donated resources, or significant contributions of research materials be made as well, if author(s) wish.

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**(i) Reference from the Journals**

1. Parkin DM, Clayton D, Blook RJ, Massyer E, Fried HP, Iranov E et al. Childhood leukaemia in Europe after Chernobyl: 5 years follow up. Br J Cance 1996; 73: 1006-1012
2. Paganini HA, Chao A, Ross RK, Henderson Aspirin use and chronic diseases: a cohort st of the elderly. BMJ 1989; 299: 1247-1250

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1. Gyton AC, Hall JE The thyroid metabolic hormones In Textbook of Medical Physiology. 10th edn. NewTork: WB Saunders Company. 2000: 858-86

**(iii) Internet**

I. Harverd medical school Available https:// en.wikipedia. org/wiki/havard medical college, accessed October 2011

**(iv) Thesis/Dissertations**

1. Khan MAH. Lipid profile and renal function status of hypothyroid patients [MD Thesis). Dhaka Bangabandhu Skeikh Mujib Medical University:2005

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- Study concept
- study design
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- statistical analysis
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# Editorial

# Research Capacity Building: 'Newer Strategy of Boosting Research, Medical Education and Health Care Services Delivery' in Medical College Hospitals

**Kazi Selim Anwar**

Research capacity building (**RCB**) remains a crucial step towards the initiation and development of research activities in any medical college hospitals that adjuncts medical education and health care service delivery more prudently.

**RCB** is defined as 'a process of developing sustainable abilities and skills enabling individuals/ organization to perform high quality research.'[1] In recent years interest in strategic planning for RCB for healthcare professionals has boomed in Australia[2] -which, based on my widely acquired in-depth experiences in organizations/ universities, home and abroad, seems that no medical faculty/university teachers can reach the peak of success solely by virtue of completing academic curriculum. Rather, teaching-learning (T-L) facilitates following modern medical education can excel with best outcome when adjunct with internationally collaborated research, as Barrett et al experienced on comparative education learnt through Ed-Qual experiences.[3] **RCB** contributes prudent impact not only research and developmental (R & D) issues but it impacts on policy issues as the gate way in building institutional research capacity.[3]

*Janine Matus* et al in their systematic review identified existing integrated models & framework to guide **RCB** for health care professionals showing some future directions like, behaviour change (BCC) and knowledge translation theories to guide as evidenced in some developed countries to engage in research is a priority issues, particularly in **Australia.** Thus, RCB brings multifaceted benefit, for: i) healthcare professionals, ii) given organization, and iii) for the patient themselves; which in turn, translated consequently, into overall societal development.[4]

Further, **RCB** accelerated medical research in Australian hospitals improved research productivity within acute & allied health departments towards improving service delivery & cost-effectiveness towards optimizing health care to maximize benefits in Australia's healthcare system. It enhances clinician's level of attitude, increased uptake of research utilizing evidence-based practice and experience more job satisfactions, too.[5, 6, 7]

According to ***Hulcombe J*** et al **RCB-framework** includes leadership and governance, to support **public health**, in several phases of **RCB** implementation. Starting from preliminary phase of establishing research positions to run research activity with several successes of **capacity-building strategies,** with some success stories. These successes were crucially linked to solid partnerships/ **collaborations** with **universities,**[8] with additional benefit that yields quality healthcare and efficient services delivery[6] prioritizing healthcare workforces,[9] and professionals involved in quality research that positively influence on infrastructure and patient care[10] with stronger strategic planning and policy making.[11]

Though **RCB** has been introduced in some developing countries as WHO reports,[12] RCB tends to mobilize added resources to boost department specific research aptitude targeting academic and clinical faculties, in Bangladesh, **RCB drives** is noticed only in few medical research departments/units in Bangladesh where it has been developed, though medical research in Bangladesh still remains primitive[13] and sub-standard.[14]

Head, Ad-din Medical Research Unit (MRU), Ad-din Women's Medical College (AWMC), Dhaka and Editor in Chief, Journal of AWMC

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In 2016, Biswas RS et al., commented in their paper titled **"Success and failure in medical research"** that most Bangladeshi doctors lack in knowledge in medical research, both among the physicians and students. This deficiency start growing up right from their MBBS course when preliminary foundation on research (biostats & epidemiol) are introduced in their early learning process. This inadequacy often leaves our MBBS doctors ignorant in research unless some of them gets little exposure during their post-graduation. Even, then, those processes (biostats, research methodology for due to postgraduate thesis/dissertation) that often lack honesty in conceptualizing, writing protocol and getting it accepted including often lacks in data collection, analysis and report writing, as Rajat Sanker, RB commented in this paper. He also added "due to premeditated outcomes, being a major shortcoming in dealing with their data management, that often creates a wider range of issues in getting their own thesis published in time.[14]

**RCB Bangladeshi perspectives:**

In Bangladesh, drives on RCB have been noticed, rarely, except few large NGOs like institutions like, **icddr, b, BRAC, CIPRB**, etc. and few GoB institutions like **BSMMU, DMCH, DSH,** etc.

Recently, Biswas RS commented that medical research in Bangladesh remains in a very primitive way, even in 2016.[13]

So, we few academicians, clinical experts and public health physicians got involved to start RCB to strengthen research activities in **AWMC** led by the Ad-din Research unit (**ARU**) through our Director General (Hospitals & Nursing) which later on transformed into Medical Research Unit (**MRU**) by the Executive Director. We at the **MRU** envision to bring all the 4 existing Ad-din Medical Colleges located in two large cities (Dhaka & Khulna) and in two district towns (Jessore & Kushtia), under one umbrella, through imbedding a Hi-Fi Satellite Server.

Thus, based on literature showing globally practiced RCB in a wide range of medical institutions and hospitals, we at the AWMCH can foresee the impact RCB on our new initiative to run our MRU, more fruitfully yet prudently enough. We now need to develop it further to provide our talented academicians and clinical experts a modest yet sound ready-to-go platform to facilitate scientific growth and research potentiality so that they can ventilate their research potentiality nationally and globally through publishing important findings by the talented Ad-din hub.

To train up our clinicians, bio-medical experts, nurses and technicians with adequate input, technical support and proper training under the modest umbrella we definitely need some financial assistance in this expensive endeavor with costly logistic back up and prudent laboratory support. Since ARC currently suffer from a bit of inadequate financing, we essentially require extra funding/grant from other sources. Such grants will help boost up running our ARC in full swing, more smoothly yet effectively and sustainably.

**Applied for a good Funding to run AWMC's RCB mission**

With this view, we from MRU (earlier ARC) had already applied for a 'Research Capacity Building' project from the Ministry of Health & Welfare GOB (PM initiated **IHSRD Fund activities 2021**), as a praise-worthy initiative of releasing a grand research funding to strengthen research potentials in health sector. **The objective of this RCB project in AWMC, was, to:**

i) Facilitate our clinicians/medical teachers in boosting knowledge, skill expertise on research,

ii) Provide a strong platform for them in producing quality research, and,

iii) Assisting them in growing their departmental capability of conducting a wide range of research projects to produce quality research output.

Further, we, share knowledge and research skills through bi-lateral collaborations with some national (DU, JNU, NSU, DMCH, DSH, BIRDEM, BSMMU, etc.) and international institutes/ universities (with Malaysia, Japan, and recently, trying with Univ of NSW in Sydney).

**Impact of RCB in future research:**

Global reports evidence that RCB drives assist in establishing good rapport in health through doctor's enhanced training and intensive research. However, it is less observed in Bangladesh, though doctors at the icddr, b and/or other GoB-ran universities/hospitals (like BSMMU, DMCH, DSH, etc.) yielded better research, based on good training/exposure that provenly impacted on patients care in those hospitals. So, outcome of this RCB should also impact on patient's management and in clinical research, in our AWMCH, as well.

**Impact of this RCB at the National Level**

Since good quality research reportedly provide improved healthcare service delivery, globally, outcome of this RCB may logically derive positive impact on patients care at Ad-din hospitals. And, outcome of this

RCB will be translated into better patient management and care leading to better public health consequences at least among catchment population. Further, once our ARU proves to be beneficial, profitable and income generating organ then this idea may be translated to copy by some of other medical colleges and hospitals. This is how the urge towards, and positive vibrations inside our doctor's mind/thought on medical research can be seeded in, speeded up & spread out through medical research countrywide. This will not only intensify interest in research in among our clinical/non-clinical academicians but also will pave ways to develop, nurture & practice medical research in other medical colleges/ institutes/ Universities!

**How RCB will influence UN's SDG goals**

This RCB project has got a definite link with the 3rd point of Sustainable Development Goal (Goal-3: Good health and well-being: seeks to ensure health and well-being for all, at every stage of life). Since public health is not possible to improve without a strong heath service delivery system in any hospital and recent literature yielded to achieve that we need to develop our clinical and bio-medical research, further. That is what we have aimed to achieve through this RCB drive.

Now that our ARC suffers from a degree of financial hardship, we, essentially, look for certain funding to cover up **RCB** research & development activities by Ad-din medical college clinical professionals with the ultimate goal of improved patient care and management.

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Original Article

# Early Maternal and Neonatal Outcome of Diabetic Mothers in Tertiary Care Hospitals

Md. Abu Sufian[1], Kazi Morjina Begum[2], Mahmuda Hassan[3], Shahnaz Akter[4], Tania Noor[5]


## Abstract

**Background:** Diabetes is the most common medical complication in pregnancy. In developing countries, management of diabetes in pregnancy still poses a challenge. Infants of diabetic mother are at increased risk of periconceptional, fetal, neonatal and long-term complications.

**Objective:** To evaluate the early neonatal outcome of diabetic mothers in Tertiary care hospital.

**Material and Methods:** It was a cross sectional observational multi-centric study carried out in the Department of Paediatrics, Sheikh Hasina Medical College, Hobigonj and in the Department of Obstetrics & Gynaecolgy, Ad-din Medical College Hospital, Dhaka, during the period of January 2019 to December 2019. Total 300 samples were included in this study. The patients were divided two groups, 150 patients were diabetic pregnancy (Group I) and 150 patients were normal pregnancy (Group II). Patient`s age, parity, mode of delivery, level of glycemic control and outcome were recorded.

**Results:** In this study the mean age of the patients was 29.5 years in diabetes mellitus (DM). Maternal complications during pregnancy were UTI, pre-ecalampsia, polyhydramnios & vulvovaginitis. The incidence of caesarean section was 59.3%. There was no maternal mortality as an effect of DM but many patients (30.1%) developed complications like PPH, wound infection & urinary tract infections. In majority of cases birth weight was within 3.1 to 3.5 kg in 52.7% babies, in 14% babies birth weight was 3.6 to 4 kg, in 5.3% babies birth weight was less than 2.5 kg and 7.3% babies had birth weight more than 4 kg. Both maternal & perinatal complications were more among DM cases.

**Conclusion:** Our findings show DM is not uncommon and it is associated with higher incidence of maternal morbidity and perinatal mortality and morbidity. It is responsible for a significant higher rate of caesarean deliveries though DM alone is not an indication of caesarean section. Further study is needed in different hospitals to find out the real picture of outcome of DM.


**Keywords:** Maternal, Neonatal Diabetic Mothers, Tertiary Care Hospitals

## Introduction

Diabetes is a common medical complication in pregnancy. The prevalence of diabetes mellitus (DM) in pregnancy ranges from 1 to 14%.[1] It may be pre-gestational diabetes mellitus (pre-GDM) or may be gestational diabetes mellitus (GDM). The World Health Organization (WHO) has predicted that between 1995 and 2025, there will be a 35% increase in the worldwide prevalence of diabetes.[2] Moreover, born in Asian countries shows the highest prevalence of GDM, with up to women 17% of women likely to develop GDM.[3-4] The prevalence of diabetes in Bangladesh is 8.1% in urban and 2.3% in rural area.[2] The prevalence of GDM in urban Bangladeshi population is about 7.5%.[5]

Infants born to diabetic mother (IDM) are at increased risk of complications which may be periconceptional, fetal, neonatal and even long term.[5] GDM increases the risk of complications via a myriad of biological mechanisms. Overt maternal diabetes mellitus can adversely influence intrauterine development. Spontaneous abortions and congenital anomalies may be induced in the first trimester. Excessive foetal growth, neonatal hypoglycaemia, jaundice, polycythemia and stillbirth may be induced during the second and third trimesters.[6]

Infants of diabetic mother are at higher risk of complications and congenital anomalies like macrosomia, hypoglycemia, hypocalcemia, hypo-magnesemia, polycythemia, hyperbilirubinemia, prematurity, transient tachypnea of newborn, respiratory distress syndrome, birth asphyxia, congenital heart diseases like interventricular septal hypertrophy,


1. Principal and Associate Professor (Pediatrics) Sheikh Hasina Medical College -Hobigonj
2. Associate Professor (Obstetrics & Gynaecology), AWMCH, Dhaka.
3. Professor (pediatrics) AWMCH - Dhaka.
4. Assistant Professor (Obstetrics & Gynaecology), AWMCH, Dhaka.
5. Assistant Professor (Obstetrics & Gynaecology), AWMCH, Dhaka.

**Correspondence :** Kazi Morjina Begum, Cell: 01817002304, Email: drmorjina. awmch@gmail. com


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transient hypertrophic subaortic stenosis, cardio-myopathy, cleft lip, cleft palate, sacral agenesis, jitteriness, seizures, movement disorders.[7-8] A strong association between congenital anomalies and maternal glycemic control has been documented.[9]

Although in developed countries there has been significant improvement in the outcome of diabetic pregnancies due to better metabolic control before and during pregnancy and better neonatal care, the management in developing country still poses a major challenge. Due to increased perinatal morbidity and mortality.[9] The present study aimed to know the early neonatal outcome of diabetic mothers in a tertiary care hospital.

## Materials and Methods

This cross sectional observational study was carried out Department of Obstetrics & Gynaecology, Ad-din Women's Medical College and Hospital, Dhaka, Bangladesh January 2020 to December 2020. Patients with diabetic pregnancy and normal pregnancy admitted in obstetrics & gynaecology department. Total 300 samples were included in this study. Patients were divided into two groups, 150 patients were diabetic pregnancy (Group I) and 150 patients were normal pregnancy (Group II). Data were collected by using a preformed questionnaire. The purpose of the study was explained to all study population. Relevant history was taken, gestational age was determined by last menstrual period, previous antenatal records were collected, clinical examination was done in all the cases. All these collected information was recorded in a pre-designed data collection sheet. Data were processed and analyzed by computer software SPSS (Statistical Package for Social Sciences) version 22.

## Results

**Table I:** Age distribution of the patients (n=300)

| Age in years | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| 20-25 | 28 | 18.7 | 49 | 32.7 | 0.631 |
| 26-30 | 43 | 28.7 | 64 | 42.7 | |
| 31-35 | 62 | 41.3 | 26 | 17.3 | |
| 36-40 | 17 | 11.3 | 11 | 7.3 | |
| Mean±SD | 29.50±5.29 | | 27.78±4.57 | | |

Group I: Diabetic pregnancy

Group II: Normal pregnancy

Table: 1 shows that among total 300 cases, 150 were diabetic (50%) and 150 were in normal pregnancy (50%). Among all, the largest age group was 26-30 years, which consists of 107 cases followed by age group of 31-35 years, having 88 cases. P value is 0.631 which is not significant.

**Table II:** Antenatal maternal complications of current pregnancy (n=300)

| Complication | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| UTI | 26 | 17.3 | 2 | 1.3 | 0.001 |
| Vulvovaginitis | 11 | 7.3 | 4 | 2.7 | |
| Polyhdramnios | 19 | 12.7 | 2 | 1.3 | |
| PIH | 7 | 4.7 | 2 | 1.3 | |
| PE | 16 | 10.7 | 7 | 4.7 | |
| Premature rupture of membranes | 9 | 6.0 | 5 | 3.3 | |
| None | 62 | 41.3 | 128 | 85.3 | |

Group I: Diabetic pregnancy

Group II: Normal pregnancy

According to Table II, 26 patients who presented with UTI were diabetic (17.3%). Of 150 diabetic patients, 19 had polyhydramnions (12.7%), followed by pre-eclampsia in 16 patients (10.7%). Majority of the pregnant women from the non-diabetic group presented with no antenatal complications (85%).

**Table III:** Mode of delivery in study subjects (n=300)

| Mode of delivery | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| Normal vaginal delivery | 61 | 40.7 | 113 | 75.3 | 0.001 |
| Caesarean section | 89 | 59.3 | 37 | 24.7 | |

Group I: Diabetic pregnancy; Group II: Normal pregnancy

Table III shows that caesarean section was done in majority of the pregnant women having diabetes mellitus (59.3%), whereas, the normal pregnancy group mothers mostly underwent normal vaginal delivery (75.3%).

**Table IV:** Postnatal complication of GDM patients (n=300)

| Maternal Complication | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| PPH | 16 | 10.7 | 4 | 2.7 | 0.011 |
| Wound infection | 13 | 8.7 | 0 | 0.0 | |
| UTI | 16 | 10.7 | 4 | 2.7 | |
| None | 105 | 70.0 | 142 | 94.7 | |

Group I: Diabetic pregnancy; Group II: Normal pregnancy

According to Table IV, most common postnatal maternal complications were seen in both PPH and UTI (10.7% followed by wound infections (8.7%) in diabetic group. In the normal pregnancy group there was no wound infection.

**Table V:** Birth weight of the baby (n=300)

| Birth weight | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| ≤2.5 Kg | 8 | 5.3 | 7 | 4.7 | 0.001 |
| 2.5-3 Kg | 31 | 20.7 | 104 | 69.3 | |
| 3.1-3.5 Kg | 79 | 52.7 | 39 | 26.0 | |
| 3.6-40 Kg | 21 | 14.0 | 00 | 00 | |
| >4 Kg | 11 | 7.3 | 0 | 00 | |

Group I: Diabetic pregnancy; Group II: Normal pregnancy

According to Table V, 11 babies were born with birth weight > (7.3%) from the diabetic group. While 104 babies weighed between 3.1 to 3.5 who were mother from normal pregnancy group.

**Table VI:** Neonatal complications of GDM patients (n=300)

| Neonatal complications | Group I (n=150) | | Group II (n=150) | | P value |
|---|---|---|---|---|---|
| | No | % | No | % | |
| Hypoglycemia | 13 | 8.7 | 0 | 0.0 | 0.001 |
| Hyperbilirubinaemia | 7 | 4.7 | 0 | 00 | |
| Prematurity | 16 | 10.7 | 9 | 6.0 | |
| RDS | 4 | 2.7 | 0 | 00 | |
| Birth asphyxia | 10 | 6.7 | 0 | 00 | |
| Umbilical cord sepsis | 3 | 2.0 | 2 | 1.3 | |

Group I: Diabetic pregnancy; Group II: Normal pregnancy

Table VI, describes that 10.7% babies were born with prematurity, from the diabetic group, followed by hypoglycaemia in 8.7% and birth asphyxia in 6.7% babies. Moreover, 6% babies were both the non-diabetic mothers with prematurity. Umbilical cord sepsis was present in 2% babies from the diabetic group and in 1.3% babies from the normal pregnancy group.

**Discussion**

Diabetes mellitus is the commonest endocrine disorder during pregnancy. The duration and severity of maternal diabetes and quality of its control during pregnancy determine the outcome of the offspring.[10] The aim of this study was to evaluate the early neonatal outcome of diabetic mothers.

In this study the mean age of patients with DM was 29.50±5.29 years with age range from 20 to 40 years. Similar data was found in a study conducted by Xilin Yang in China where the mean age of patients with DM was 28 years ± 0.38SD.[11] A study was conducted by Zargar AH in India showed that prevalence of DM steadily increased with age (from 1.7% in women below 25 years to 18% in women 35 years aged or older.[12] Maximum patients (40%) were in age group of 31 to 35 years.

In this study 4.7% patients had PIH, 10.7% had PE, 17.3% had urinary tract infection, 12.7% had polyhydramnios, 6% had PROM & 7.3% had vulvovaginitis. Some patients had more than one complication and some patients had no complications. A study in India was conducted by Jindal revealed that the incidence of hydramnios was 28% in patients with GDM.[13] Marked dissimilarities was detected between two studies. Though polyhydramnios was significantly higher in Jindal's study, infection was common in this study candidal vulvovaginitis was reported in 4% in the GDM group as compared to 1.3% in controls. A hospital-based series of 447 pregnant women conducted by Rizk in UAE found that the prevalence of UTI in patients with GDM was 7.9%.[14]

This study found common risk factors related to the patients were family history of diabetes, history of GDM in previous pregnancy, multiparity and maternal age more than 30 years. This finding consistent with several studies.[2,9] A recent large epidemiological survey conducted in India to determine the risk factors of DM revealed significant association between DM and advanced maternal age, pre-pregnancy obesity and family history of diabetes.[9]

In present study DM patients had higher frequency of caesarean section than vaginal delivery (59.3% vs. 24.7%) because of complications of mother and fetus due to poor control of DM. This findings consistent with Beugm et al.[2]

This study shows that no maternal mortality was detected in this study. In control cases postpartum complications were less. Wound infection was present in one case & urinary tract infection also found in 4 case among control group. But the patients with DM, 16 patients had PPH, 13 patients had wound infection and 16 patients had UTI. The difference was significant between control and DM cases. A study in Australia by Ju et al. revealed that maternal adverse outcomes were detected among 12.9% DM patients. Maternal death was also not found in that study.[15]

In majority of cases birth weight was within 3.1 to 3.5 kg in 79 babies, in 21 babies birth weight was 3.6 to 4 kg, in 7 babies birth weight was less than 2.5 kg and 11 babies had birth weight more than 4 kg in this study (7.3%). Begum et al. study revealed that 12% baby had normal birth weight and 15% was macrocosmic baby.[54]

The pregnancy related mortality & morbidity in DM patients is less than that of established diabetes as better understanding and modern management of DM has resulted in reduced neonatal mortality and morbidity. The neonatal complications were more in DM cases. Perinatal morbidity was present in 13 cases due to hypoglycaemia in 16 case & prematurity. Among neonates of control, 9 babies had prematurity and 2 neonates was umbilical cord sepsis. The difference was statistically significant in between two groups. A study was conducted by Seshiah et al. revealed a dissimilar data which showed hyperbilirubinaernia, hypoglycemia and RDS in 3.8%, 4.0% and 2.6% respectively with no neonatal death.[4] Another study was conducted by Anjum in India found that hyperbilirubinaemia, hypoglycemia, RDS, congenital anomaly, hypocalcaemia and neonatal death was 11.11%, 5.5%, 8.33%, 0.0%, 0.0% and 2.78% respectively.[9] In this study, hypoglycemia and hyperbilirubinaernia were more frequent neonatal complications.

**Conclusion**

It concluded that assessing the risk factors and identifying those women as high risk group for DM is important for the early diagnosis of DM. However there was significant difference in the incidence of maternal and neonatal complications between control and

diabetes mellitus (DM). If the diagnosis of DM is made in a timely fashion and optimum glucose control can be achieved and maintained, the outcome would be favourable. There are evidences which suggest that adequate blood sugar control during pregnancy reduces the incidence of congenital anomalies, maternal and fetal morbidity and mortality. The best results can be obtained if diabetic care is given by a team consisting of obstetrician, dietician, endocrinologist and neonatologist by maintaining blood sugar level as near as normal.

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Original Article

# Perinatal Outcome of Vaginal Birth After Earlier Caeserian Section: Findings from Tertiary Care Teaching Hospital

Kazi Morjina Begum[1], Md. Abu Sufian[2], Mahmuda Hassan,[3] Bonika Biswas[4], Rahima Khatun[5], Mst. Nilufar Jahan[6]

**Abstract**

**Background:** Vaginal Birth After Caesarean (VBAC) may be one of the strategies developed to control the rising rate of cesarean deliveries in our country. Analyzing outcome of previous caesarean pregnancies will provide an insight for reducing the caesarean rates and formulating protocols and policies for trial of labor.

**Objective:** To evaluate the perinatal outcome of vaginal birth after caesarean section (VBAC) in a tertiary care teaching hospital.

**Materials and Methods:** It was a cross sectional study carried out at the department of Obstetrics and Gynaecology, Ad-din Women's Medical College and Hospital, Dhaka, Bangladesh from January 2019 to December 2019. (Pregnant women who were admitted in the Department of Obstetrics and Gynaecology). Total 5098 patients were included in this study. Data were processed and analyzed by computer software SPSS (Statistical Package for Social Sciences) version 22.

**Results** Total number of deliveries during the study period was 5098. There were 4913 (96.4%) vaginal deliveries and only 185(3.6%) vaginal birth after caesarean section (VBAC). Feto-maternal outcome was better in VBAC patients.

**Conclusion:** The rate of caesarean section is increasing alarmingly now a days, this study tried of VBAC in appropriate group of patients. National policy and guidelines are necessary after large multicenter prospective studies.

**Keywords:** Vaginal, Birth after C/S, Perinatal outcome

## Introduction

The term 'caesarean section' (CS) denotes the delivery of fetus, placenta and membranes through an incision in the abdominal and uterine walls[1]. Trial of labour (TOL) for vaginal birth after caesarean section (VBAC) is a well-established standard practice[2]. The success rates for VBAC range between 60%–80% after one previous lower segment caesarean incision[3]. Factors associated with successful vaginal birth in a trial of labour include age < 40 years, previous history of vaginal birth, non- recurrent indication for previous CS, strength of the previous scar associated obstetrical complicating factor/factors, pelvis adequate for fetus, number of previous CS, informed consent of the patient, available resources (anesthesia, blood transfusion, and theatre) for emergency CS, cervical effacement greater than 75% on admission, and cervical dilatation 4 cm or more on admission[2]. Trial of VBAC is not practice in case of previous classical or Inverted "T" shaped uterine incision, previous two or more lower segment CS, presence of other complications in pregnancy - Obstetric (pre-eclamsia, malpresentation, placenta-previa)or medical, resources limited for emergency caesarean delivery or patient refusal for VBAC-TOL.

There is a definite risk of uterine rupture in vaginal birth after cesarean delivery (VBAC) often leading to catastrophes which can be avoided by rapid diagnosis and prompt intervention. Evidence confirming the safety

1. Associate Professor (Department of obstetrics & Gynaecology) -AWMCH - Dhaka.
2. Principal and Associate Professor (Department of Pediatrics) -Sheikh Hasina medical College -Hobigonj.
3. Professor (Department of Pediatrics) -AWMCH - Dhaka.
4. Assistant Professor (Department of obstetrics & Gynaecology) -AWMCH - Dhaka.
5. Assistant Professor (Department of obstetrics & Gynaecology) - AWMCH -Dhaka.
6. Assistant Professor (Department of obstetrics & Gynaecology) - AWMCH - Dhaka.

**Correspondence:** Kazi Morjina Begum, Associate Professor, Department of obstetrics & Gynaecology, AWMCH - Dhaka. Cell: 01817002304, Email : drmorjina. awmch@gmail. com

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of VBAC within proper guidelines has been available for more than 10 years[4-6]. However, wide variations in VBAC rates still exist between hospitals and physicians. VBAC offers distinct advantages over a repeat CS since the operative morbidity and mortality are completely eliminated, the hospital stay is much shorter and expenses involved are much less[7]. The rate of cesarean section needs to be reduced and this can be achieved to a small extent by avoiding primary CS done without explicit indications and more importantly by resorting to a trial of vaginal delivery after previous CS which is safe for the fetus[7]. The purpose of this study was to evaluate perinatal outcome of vaginal birth after caesarean section (VBAC).

**Materials and Methods**

It was a cross sectional study carried out at the Department of Obstetrics and Gynaecology, Ad-din Women's Medical College and Hospital, Dhaka, Bangladesh from January 2019 to December 2019. Pregnant women who were admitted in the Department of Obstetrics and Gynaecology. All the cases were booked in the antenatal clinic and on regular antenatal check-up. Total 5098 samples were included in this study. Data were collected by using a preformed questionnaire. The purpose of the study was explained to all study population. Relevant history was taken, gestational age was determined by last menstrual period, previous antenatal records were collected, clinical examination was done in all the cases. All these collected information was recorded in a pre-designed data collection sheet. Data were processed and analyzed by computer software SPSS (Statistical Package for Social Sciences) version 22.

**Results**

**Table I:** Age distribution of the subjects (n= 5098)

| Age | Frequency (n) | Percentage (%) | Mean±SD |
|---|---|---|---|
| ≤ 20 Years | 661 | 13.0 | 25.88±4.78 |
| 21 - 25 Years | 2648 | 51.9 | |
| 26 - 30 Years | 1326 | 26.0 | |
| > 30 Years | 463 | 9.1 | |

Table I describes the age distribution of pregnancy women with a mean age of 25.9±4.8 years more than 50% longing to young age groups of 21-25 years, followed by 26% from 26-30 years. However 13 year were so young as <20 years & 9 year older than 30 years (Table-I).

**Table II:** Vaginal birth after caesarean section (January to December 2019)

| | Normal]y vaginal delivery (NVD) | | Vaginal birth after caesarean section (VBAC) | | Ruptured uterus | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| January (n=459) | 443 | 96.5 | 16 | 3.5 | 0 | 00 |
| February (n=356) | 351 | 98.6 | 15 | 1.4 | 0 | 00 |
| March (n=439) | 425 | 96.8 | 14 | 3.4 | 0 | 00 |
| April (n=232) | 220 | 94.8 | 12 | 5.2 | 0 | 00 |
| May (n=403) | 385 | 95.5 | 18 | 4.5 | 0 | 00 |
| June (n=418) | 400 | 95.6 | 18 | 4.4 | 0 | 00 |
| July (n= 410) | 394 | 96.1 | 16 | 3.9 | 0 | 00 |
| August (n=455) | 441 | 96.9 | 14 | 3.1 | 0 | 00 |
| September (n=509) | 496 | 97.4 | 12 | 2.3 | 1 | 8.3 |
| October (n=472) | 456 | 96.6 | 16 | 3.4 | 0 | 00 |
| November (n=463) | 446 | 96.3 | 17 | 3.7 | 0 | 00 |
| December (n=473) | 456 | 96.4 | 17 | 3.6 | 0 | 00 |
| Total (5098) | 4913 | 96.4 | 185 | 3.6 | 1 | 0.54 |

Table II describes the month specific prevalence of NVD & VBAC among these children, with only 1 ruptured uterus (Table-II) showing almost a similar tern in both normal & CS deliveries in every months.

**Table III:** Fetal outcome

| Fetal outcome | No. of patients | Percentage (%) |
|---|---|---|
| Alive | 5098 | 100 |
| Still born | 0 | 00 |
| Neonatal death | 0 | 00 |

Table-III show no fetal died among all these delivery cases (0%).

**Discussion**

Several studies are raising the issue that VBAC may not be as safe as originally thought[8,9] but reports are contradictory and these factors along with medico-legal concerns have led to decrease interest in obstetrician offering and women accepting trial for VBAC in various parts of the world[10]. It is well established that repeat CS increases the risk of maternal and perinatal morbidity, including bleeding, wound infection, postpartum thromboembolism, increased risk of blood transfusion, anesthetic complications.

This study shows majority of the respondents (77.9%) were 20-30 years of age with the range of 18-40 years and the mean age of 25.88±4.78 years this study consistent with findings of the study[7]. According to this study the age group was 20 to 30 years. This study was in similarity with the study[3] where no patient was below the age of 20 years.

Vaginal delivery is associated with lower maternal morbidity and mortality as against CS. The morbidity associated with successful vaginal birth is about one-fifth than the that of elective caesarean. Perinatal risk is more after a failed trial of labour compared to elective repeated CS without labour in other study[11,12]. Failed trials of labour, with subsequent CS involve almost twice the morbidity of elective section. This information is important for informed consent, counseling about VBAC and making decision about their choices of delivery after a previous CS. The adverse events include chorioamnionitis, postpartum endometritis, and uterine rupture may require hysterectomy, blood transfusion, perinatal and neonatal deaths and neonatal neurological impairment. Many of these adverse events seen in trial of labour (VBAC) are attributable to the failure of labour and the requirement for a repeated emergency CS. This study represents our observations for a period of 1 year. The selection of women for VBAC is mainly influenced by woman's desire and conditions favorable for vaginal delivery. In general, this institution offers a conservative approach both in the selection of women and in the management of their labor. Generally speaking women belonging to higher socioeconomic status were either not keen for VBAC or opted out of the study.

In this study a total of 5098 patients, among them 4913 had successful vaginal delivery (96.4), 185 (3.6%) patients under went VBAC 1(0.54%) was ruptured uterus but no need of hysterectomy due to emergency laparotomy and scar repair which is comparable Nepal, Pakistan and lower than in many others studies done in Western Country.[10,13,14] In the present study, suitable women were selected for VBAC during early pregnancy after a thorough assessment, and adhering to strict inclusion and exclusion criteria as mentioned earlier. This is in line with the fact that the history of a previous normal vaginal delivery is the single most important predictor for a successful VBAC.[15,16]

In this study only one case of scar dehiscence occurred. Now a days, there is a significant increase in primary CS for various indications, like fetal distress, caesarean delivery for matrenal request and many other non-recurrent indications thus increasing the rate of pregnant women with previous scarred uterus[17]. Vaginal Birth After Cesarean (VBAC) can be one of the strategies developed to control the rising rate of CS. It is a TOL in selected cases of previous CS in a well-equipped tertiary care hospital. In the present era of lower CS, the dictum now is once there is a CS, always it is mandatory hospital delivery in a well-equipped hospital. Rising rates of CS is a matter of great concern and trial of labor in previous CS is an attractive alternative[18]. Analyzing outcome of previous caesarean pregnancies will provide an insight for reducing the caesarean rates and formulating protocols and policies for trial of labor in previous CS deliveries. The most important event because of which obstetricians still hesitate to attempt planned VBAC is the uterine scar integrity. There is a definite risk of uterine rupture in vaginal birth after caesarean delivery often leading to catastrophies which can be avoided by early diagnosis and prompt intervention.

In this study good maternal and fetal outcomes were evident in successful VBAC group. This findings consistent with Pokhrel et al. study[10]. This findings were comparable to other studies done by Goel SS et al[19]. In the context of rising rate of primary CS, management of patient with previous CS with the appropriate mode of delivery is the challenge in obstetric practices. Regular and intensive antenatal surveillance, proper selection of patients, vigilant monitoring with competent technical

team and dedication on the part of healthcare giver can increase safety of VBAC. There is no doubt that trial of labor is safe if followed with great care but it is not risk free[19]. There were no serious complications like hysterectomy, emergency blood transfusion and visceral injury in patients with successful VBAC group.

**Conclusion**

The study suggests that successful VBAC is associated with better feto-maternal outcomes. Prior vaginal birth is a good predictor for the outcome of VBAC. Screening for this should preferably begin at antenatal booking itself to minimize the associated risks. Proper selection, appropriate timing and suitable methods of induction with close supervision by competent staff are the key factors to achieve greater degree of success.

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Original Article

# Limited Screening of Thyroid Function for Preterm and Clinically Suspected Hypothyroidism Term Neonates

Banita Mistry[1], Shamim Ara Begum[2], Dipa Saha[3], Shahjahan Chowdhury[4], Farzana Binte Rashid[5], Md Allauddin[6]

**Abstract:**

**Purpose:** To help develop a nationwide guideline for screening and appropriate measure for prevention of mental handicap due congenital hypothyroidism.

**Introduction:** Congenital hypothyroidism is an important cause of mental retardation which is preventable if early diagnosis is done and treatment start within 3 months of life especially within 4 weeks of life. Severe cretinism may present within few days of life but milder degree of cretinism is difficult to diagnose until later infancy or early childhood [8]. Early diagnosis and treatment prevent brain damage and allows the child to grow like a normal child. Late diagnosis leads to severe neurodevelopment and intellectual deficit.

**Method:** This Purposive prospective study conducted in Dhaka Shishu Hospital, Dhaka from May, 2014 to October, 2014. A total of 100 babies were selected among them 50 preterm and 50 term neonates for clinically suspected congenital hypothyroidism (CH). In preterm neonate blood sample was taken on $5^{th}$–$7^{th}$ days, because after birth the TSH level is transiently high and it goes to base line level usually after 72 hours. In this study blood sample of term neonates was taken after 7 to 28 days after birth.

**Result:** The mean gestational age of the baby was 35.58 ±4.29 weeks among them 62 male and 38 female. Mean body weight was 2.14±0.71kg and mean TSH level were 1.69 ±0.7mIU/L. Out of 50 premature neonates, mean age was 31.57 ± 1.94 weeks. 30 males and 20 females. Mean body weight 1.50±0.27kg.Mean TSH level 1.60 ± 0.84mIU/L. Suspected term babies mean age was 39.37 ± 1.34 weeks, 64%(32) male and 36% (18) female, mean body weight 2.77±0.34kg and Mean TSH level 1.84±0.83mIU/L. The mean maternal age was 23.04±0.53 years.

**Conclusion:** There was no significant difference of TSH level of the study babies. It is not possible to make any comment regarding congenital hypothyroidism with this small sample size study.

**Keywords:** Suspected term baby, Hypothyroidism, TSH, preterm baby.

1. Assistant Professor, Department of Paediatrics, National Institute of neurosciences and Hospital, Dhaka.
2. Assistant Professor, Department of Paediatrics, National Institute of neurosciences and Hospital, Dhaka.
3. Assistant Professor, Department of Paediatrics, Bashundhara Addin Medical College Hospital, Dhaka
4. Junior Consultant, Department of Paediatrics, National Institute of neurosciences and Hospital, Dhaka.
5. Junior Consultant, Department of Paediatrics, National Institute of neurosciences and Hospital, Dhaka.
6 Junior Consultant, Department of Paediatrics, National Institute of neurosciences and Hospital, Dhaka.

**Address of correspondence:** Dr. Banita Mistry, Assistant Professor, Department of Paediatrics Neurology, Institute of neurosciences and Hospital, Dhaka. E-mail: banitamistry08@yahoo.com

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## Introduction:

Thyroid hormone is essential for human being in maintaining basal metabolic rate and metabolism of all nutrients.[1] It plays a crucial role in foetal life, infancy and childhood for normal somatic and brain growth and development. 80% of brain development occurs in intrauterine life and rest 20% development occurs within first few years of life mainly within two years after birth.[2] During this time normal serum level of thyroid hormone is essential. Thyroid function is also affected by gestational age, birth weight, birth asphyxia, respiratory distress and severe illness.[3] Congenital hypothyroidism is one of the commonly recognized disorders on screening and its incidence has been reported as 1:2700 to 1:6900 in different literature.[4]

Congenital hypothyroidism is an important cause of mental retardation which is preventable if early

diagnosis in done and treatment start within 3 months of life especially within 4 weeks of life.[5-7] It is more common in preterm babies than term babies. Some preterm babies suffer from transient hypothyroidism. That is why it is very important to screen the newborns for hypothyroidism. Early diagnosis prevents brain damage and allows the child to grow like a normal child. Klein showed the IQ of those who were treated before 3 month of age was 89: IQ was 70 if started between 3 to 6 months and 54 Where treatment started after 6 months of age.[8] Neonatal screening program for detection of congenital hypothyroidism is now establish in all developed and many developing countries [7].In most of the countries it is done along with the screening of other inborn errors of metabolism like phenylketonuria, Tyrosinaemia (Guthrie test).[9,10,11-15]

**Methods:**

This purposive prospective study was conducted in Dhaka Shishu Hospital, Dhaka from May, 2014 to October, 2014. Preterm and clinically suspected hypothyroidism in term neonates in Neonatal unit of Paediatrics department of Dhaka Shishu Hospital, Dhaka. Due to time and economic constraints sample determined purposively, one hundred were selected, 50 preterm and 50 term neonates forclinically suspected congenitally hypothyroidism (CH). Each case was selected randomly until the desired sample size was fulfilled. In this limited screening. Only risk group like preterm, low birth weight and those term neonate showing sign symptoms suggestive of congenital hypothyroidism were included as because these group has more chance to develop congenital hypothyroidism.

**Inclusion criteria:**

Preterm neonates less than 35 weeks of gestational age and weight less than 2000gm (2kg). Term neonate after one week of birth with sign-symptoms related to Congenital Hypothyroidism. Exclusion criteria Full term babies without sign-symptoms of congenital hypothyroidism. Preterm babies weighting more than 2 kg and gestational age more than 35 weeks. Informed consent was taken from the parents of the neonates about the prospect and procedure of the study before drawing the heel prick blood sample. A total of 100 cases were included in this study. Heel prick capillary blood was taken between $5^{th}$ and $7^{th}$ days after birth for preterm babies and after 7 days for clinically suspected hypothyroidism in term babies.

Under aseptic precaution capillary blood was collected by pricking heel applying single drop of blood ample enough to spread out over the required area penetrating the filter paper from one side to other. Blood spots were allowed to dry in air in a horizontal position for one hour at room temperature avoiding direct light and not touch the surface. These filter papers were sent to laboratory of the Institute of Nuclear Medicine, Shahbag, Dhaka for thyroid stimulating hormone estimation.

Statistical Methods and data Analysis Collected data was compiled, and analysed by 'Special Package for Social Science (SPSS), appropriate statistical test (Un Paired Student 't' test) was done and presented in the form of table, pie diagram, bar diagram.

**Results:**

In this study a total of 100 case, 50 premature baby, age less than 35 weeks and body weight less than 2000gm and 50 suspected term baby were included for screening the TSH level for diagnosis of congenital hypothyroidism. The mean age of the baby was 35.58 ±4.29 (table-1) weeks (Mean ± SD) among them 62 male and 38 female (Table-II). The Mean body weight were 2.14±0.71kg (Table-VI) and Mean TSH level were 1.69 ±0.7mIU/L (Figure-3). Out of 100 mothersMean age were 22.98 ±4.02 years (Figure-1) and educational qualification 13mother passed HSC, 45 mother passed SSC, 20 mother studied in secondary school, 15 mother in primary level and 7 illiterate (Figure-2).

Gestational age was diagnosed by Ultrasonography (USG) 48 % and Last menstrual period (LMP) 15%and USG &LMP were 37% (Table-IV). Among 100 mothers, 45% from Dhaka district, 11% from Gazipur, from Mymensingh, 10% from Faridpur, 5% from Madaripur, 10% Narshingdi, 12 from Narayangonj and 3% from Pirojpur (Table-3).Out of 50 premature neonates, Mean age was 31.57 ± 1.94 weeks (Table-I) and 30 male and 20 female (Table-II). Mean body weight 1.50±0.27kg (Table-VI).

Mean maternal age 22.70 ± 3.39 years (Figure-1) and Mean TSH level 1.60 ±0.84mIU/L(Figure-3). Out of 50 suspected term babies sign-Symptoms of congenital hypothyroidism were respiratory distress 20% (10), prolong jaundice 14 (7%), cry little and hoarse6%(3), abdomen distension 12%(6), poor feeding 16%(8), decrease heart rate 8%(4), delayed passes of meconium 4%(2), cool body 8%(4) reflex diminished8%(4) and umbilical hernia 4%(2), (Table-V).

Mean age was 39.37 ± 1.34 weeks (Tables-1), 64%(32) male and 36% (18) female (Table-II), Mean body weight 2.77±0.34kg (Table-VI) and Mean TSH level 1.84±0.83mIU/L (Figure-3). The mean maternal age was 23.04±0.53 years (Figure-1).

**Table-I:** Age distribution of the preterm and clinically suspected hypothyroidism term neonates n=100

| Study cases | Number | Mean Age in Wks (Mean± SD) |
|---|---|---|
| Premature baby (less than 35 weeks) | 50 | 31.57±1.94 |
| Hypothyroidism suspected term neonates | 50 | 39.37±1.34 |
| Mean age value of 100 babies | 100 | 35.58±4.29 |

**Table-II:** Sex distribution of the preterm and clinically suspected hypothyroidism term neonates n=100

| Study babies | Male | Female | Total |
|---|---|---|---|
| Premature baby | 30 | 20 | 50 |
| Hypothyroidismsuspectedterm neonates | 32 | 18 | 50 |
| Grand Total | 62 | 38 | 100 |

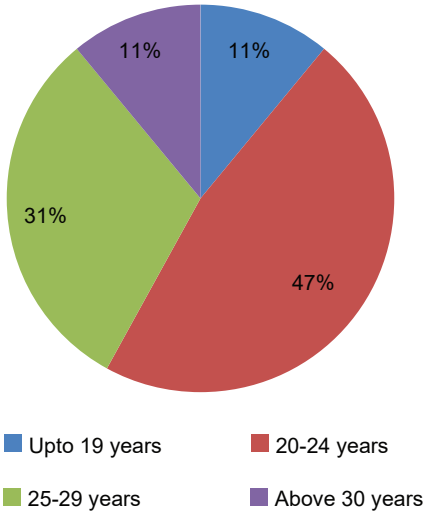


**Figure-1:** Maternal age distribution for study babies

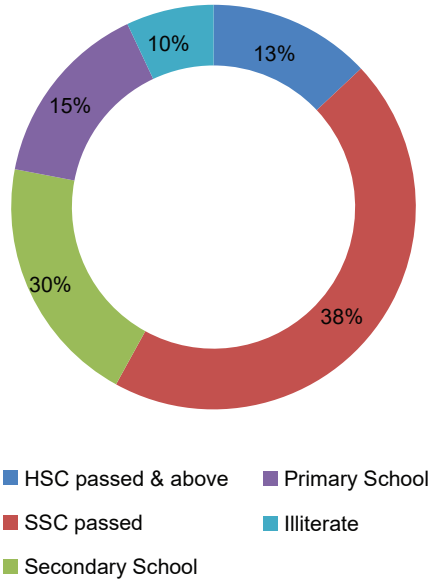


**Figure-2:** Maternal Educational Qualification of the study babies

**Table- III:** Locality or residence of Mothers of the study babies

| Locality | Number | Percentage |
|---|---|---|
| Dhaka District | 45 | 45% |
| Gazipur District | 11 | 11% |
| Mymensingh | 04 | 4% |
| Foridpur | 10 | 10% |
| Madharipur | 05 | 5% |
| Narshingdi | 10 | 10% |
| Narayangang | 12 | 12% |
| Pirojpur | 03 | 3% |

**Table-IV:** Technique of Gestational age determination of the study neonates

| Investigation | Number | Percentage |
|---|---|---|
| Ultrasonography | 48 | 48% |
| Last menstrual period (LMP) | 15 | 15% |
| USG &LMP | 37 | 37% |
| Total | 100 | 100% |

**Table-V: Si**gn-Symptoms of clinically suspected hypothyroid term neonates. N=100

| Sign and symptoms | Number | Percentage |
|---|---|---|
| Respiratory distress | 10 | 20% |
| Prolong Jaundice | 7 | 14% |
| Cry little and hoarse | 3 | 6% |
| Abdomen distended | 6 | 12% |
| Poor feeding | 8 | 16% |
| Decrease heart rate | 4 | 8% |
| Delayed passes of meconium | 2 | 4% |
| Cool body | 4 | 8% |
| Reflex diminished | 4 | 8% |
| Umbilical hernia | 2 | 4% |

**Table-VI:** Body Weight of the study Neonates.

| Study baby | Weight in kg (Mean±SD) | P value |
|---|---|---|
| Preterm baby | 1.50 ± 0.27 | > 0.10 |
| term Neonates | 2.77 ± 0.34 | > 0.10 |
| Total 100 Study baby | 2.14 ± 0.71 | > 0.10 |

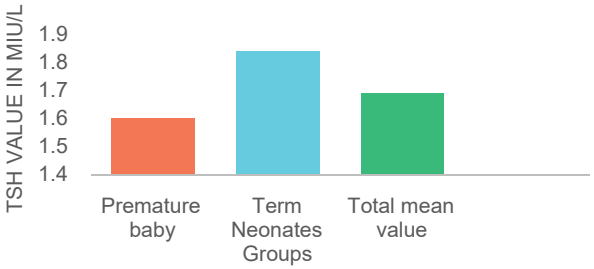


**Figure – 3:** Mean TSH Value of the Study neonates

## Discussion

In this study estimation of Thyroid Stimulating Hormone was done in 100neonates, fifty premature baby, age less than 35 weeks and body weight less than 2000mg and 50 clinically suspected term baby were included for screening the TSH level for diagnosis of congenital hypothyroidism. The mean age of the neonates was 35.58 ±4.29 weeks (Mean ± SD) among them 62 male and 38 female . Male and female ratio about 2:1 The Mean body weight were 2.14±0.71 kg and MeanTSH level was 1.69 ±0.78,IU/L which is consistent with other studies done in our country in verity studies.[5,6,22] But it was done by others.[7,13,24-27] Out of 100 mothers Mean age was 22.98 ±4.02 years. Regarding literacy of the mother-13 passed HSC, 45 passed SSC, 20 studies in secondary school, 15 in primary level and 7 illiterates. Regarding age-48% from Ultrasonography report (USG) and 15% from maternal history of last menstrual period (LMP) and 37% from maternal history (LMP) which correlates with Ultrasonography and these finding are similar with other study done.[5,6] Among 100 mothers, 45% from Dhaka district, 11% from Gazipur, 4% from Mymensingh, 10% from Faridpur, 5% from Madaripur, 10% Narshingdi, 12% from Narayangong and 3% from Pirojpur. No mother was from endemic iodine deficiency zone and none of them had goiter and these finding are similar with other study done[5]differs from other study.[4,25] Out of 50 premature neonates, Mean age was 31.57±1.94 weeks and 30 male and 20 female. Mean body weight 1.50±0.27kg (p>0.10). Mean maternal age 22.70±3.39 years and Mean TSH level 1.60±084mIU/L (p>0.10) and these finding issimilar with other study done by in our country.[5]

Out of 50 clinically hypothyroidism suspected term babies, sign symptoms of congenital hypothyroidism were respiratory distress 10(20%), prolonged jaundice 7(14%), hoarse cry 3(6%), abdominal distension 6(12%), poor feeding 8(16%), decreased heart rate 4(8%), delayed passes of meconium 2(4%), cool body 4(8%), diminished reflexes 4(8%) and umbilical hernia 2(4%), Mean age was 39.37±1.34 weeks, 32(64%) male and 18(36%) female, Mean body weight was 2.77±0.34kg (p>0.10) and Mean TSH level 1.84±0.83mIU/L (p>0.10). Thyroid Stimulating Hormone value of more than 20mIU/L was regarded to be abnormal.In this study Thyroid Stimulating Hormone (TSH) value of the preterm and suspected term neonates were within normal level and did not show significant difference (p>0.10) and these findings are similar with the study done.[5,6,16-18] Approximately one infant in every 3500 to 4500 has congenital hypothyroidism.[20-22] Sample of this study was very small for TSH assay in comparison with the incidence of congenital hypothyroidism and none of the sample was from anyendemic zone for iodine deficiency and all mothers gave history of ingestion anduse of iodinated salt in their routine daily practice. Therefore, failure of detection of congenital hypothyroidism in the present study is not unusual. Further study with large sample including samples from goiterous zone specially Rongpur, Dinajpur, Takurgone (North Bangal) for iodine deficiency, using different samples and appropriate

screening method for estimating TSH are required for detection of congenital hypothyroidism[23], similar to studies conducted by others.[24,25]

**Conclusion:**

There was no significant difference of TSH level of the study babies. It is not possible to make any comment regarding congenital hypothyroidism with this small sample size study. A Nationwide mass screening urgently needed to detect the incidence of congenital hypothyroidism and appropriate measure should be taken to overcome the serious complication like mental handicap resulting from congenital hypothyroidism.

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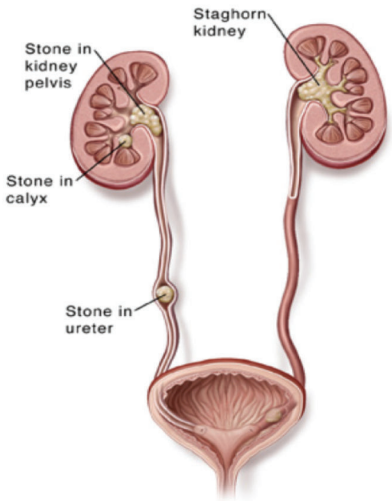
Review Article

# Urolithiasis and Nephrolithiasis: The Two Wings of Analogous Urinary Tract Injuries

M Afiquor Rahman[1], Md. Hasanuzzaman[2], Meherunessa Neela[3], Suriya Hasna Suha[4], Merina Tanzil[5]

**Introduction:**

Commonly known as 'Kidney stones', are termed as 'renal calculi' in medical science. These calculi/stones are of hard masses of different sizes made of mineral crystals clumped together inside the kidneys.[1] Although they start to originate in the kidney, these hard masses (calculi) increase in size inside ureter or bladder. According to the anatomical location of these calculi, they are often termed as: 'kidney stone', 'ureteral stone', or 'bladder stone'. The stone forming procedure is known as 'urolithiasis', 'renal lithiasis', or 'nephrolithiasis'[2] which abstractly implicates 'The two wings of analogous urinary tract injuries'.[1,2]




Urinary stones in different locations of urinary tract[3]

1. Head, Dept of Urology, and Ex-Principal, AWMC
2. Assoc. Prof. Dept of Urology, AWMC
3. Ex-Research Officer, Medical Research Unit, AWMC
4. Ex-Research Officer, Medical Research Unit, AWMC
5. Ex-Research Officer, Medical Research Unit, AWMC

**Correspondence:** Prof. M Afiquor Rahman, Head, Dept. of Urology, and Principal, AWMC, Dhaka. E-mail: afiquor@gmail.com

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**Brief History:**

Looking back to the history, urinary stones (both bladder and kidney stones) were found in Egyptian mummies from 4800 BC. Being an age-old public health issue, the pathogenesis of urolithiasis had been studying**,** yet, its treatment strategies are being refined.[4] In 1901, Elliott Smith discovered a bladder calculus in the pelvis of an Egyptian mummy. The calculus has a uric acid nucleus with concentric laminations of calcium oxalate and ammonium magnesium phosphate.[5] An ancient Indian physician Sushruta, described urolithiasis in 6$^{th}$ century. During the 10$^{th}$ century an Arabian physician Abukasis prescribed the method of smashing stones in the urethra with an instrument devised by his own.[6] During the medieval period in Europe (1096–1438) there was little activity regarding the management of stone disease.[7,8]

**Epidemiology in brief:**

Both urolithiasis (urinary tract calculi or stones) and nephrolithiasis (kidney calculi or stones) are well-reported common occurrences among US population.[13] Recent epidemiological studies have suggested an increased frequency of kidney stone disease in all age groups during the last decades.[9]

In the western world about 0.5% people are diagnosed with urolithiasis, and it is the 3$^{rd}$ most common urological diseases affecting both the males and females worldwide.[10]

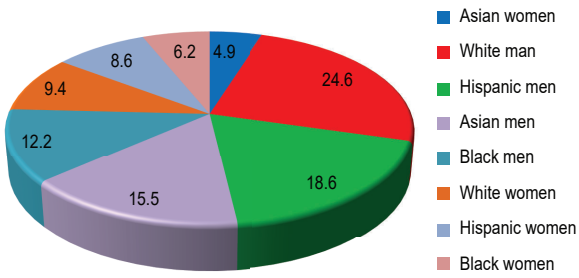


**Figure:** Prevalence of stone disease, specific to gender and race[28]

According to a report, almost 1 of 1,000 adults gets hospitalized in USA every year due to urolithiasis as this disease is more common in adults.[2]

**Demographics:**

In USA

Men: 140.6 per 100,000 population[11]

Women: 65.8 per 100,000 population[11]

In 2019, 115 552 140 incident cases were detected globally.[12]

**Kidney stone belt on the globe:**

In this zone the climatic and social conditions are favorable for stone formation. Some stones are associated with poverty, while others with affluence. In Europe and the USA, there has been a sharp, almost exclusively affluence-related rise in the occurrence of calcium oxalate and uric acid stones. Climate simulations for the USA show that the stone belt is likely to move northwards in the coming two decades.[13]

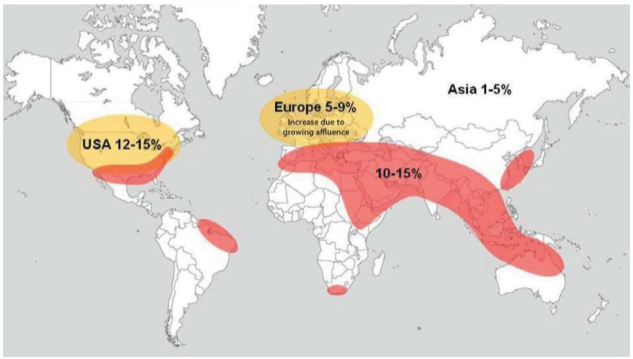


**Figure:** The kidney stone belt (red) extends all the way around the world and shows urinary stone prevalence of 10 to 15%.[14]

**Urolithiasis vs Nephrolithiasis:**

Urolithiasis is the process of forming stones anywhere in the urinary tract (kidney, bladder, and/or urethra),[15] whereas, nephrolithiasis specially refers to stone in the kidney.[16]

**Etiology of and risk factors:**

Though the etiology of renal stone is not well understood[10], multifactorial etiology has been related to this disease0.[9] Both genetic and environmental factors contribute to stone formation.[17]

The major causes of urolithiasis are:[18]

1) Hyperoxaluria, hypercalciuria, hypocitraturia, hyperuricemia, renal tubular acidosis, hypophosphatemia, cystinuria, etc.
2) Other causes include: urinary infections, impaired drainage (i.e. obstruction), post-bariatric surgery, foreign bodies, drugs, etc.

**Types of urolithiasis:**

The five commonest types of urinary stones are calcium oxalate (>50%), Ca Phosphate (10% to 20%), uric acid (8%), struvite (15%), cystine (3%).[19]


**Figure:** Major types of urinary stones

Generally, any physician, who is even not a urologist, can advise **preventive therapeutic measures** for reducing risk of recurrent kidney stones once anyone is at a risk of developing renal calculi.

**Drug Induced Urolithiasis:**

**Indinavir calculi:** Several drugs have been reported to cause urolithiasis. Among them, most frequent is indinavir, especially when administered with ritonavir.[5] Indinavir sulfate is currently one of the most widely used protease inhibitors used against HIV. The incidence of urolithiasis in patients taking indinavir is almost 20%.[20]

A study was conducted in 24 patients of urolithiasis who were taking protease inhibitors. Among them 14 were taking indinavir, three ritonavir, two nelfinavir, and five other drugs. Only in 4 patients of the 14 taking indinavir, the drug was found in kidney stone. Rest of the 10 patients underwent 24-hour urine collection and metabolic abnormalities were found in 80% of them. Five of them had hypocitraturia, four hyperoxaluria, four hypomagnesuria, three hypercalciuria, three supersaturation of calcium oxalate, and two hyperuricosuria. From this study, the authors deduced that metabolic abnormalities are more responsible for the formation of kidney stones than protease inhibitors.[21]

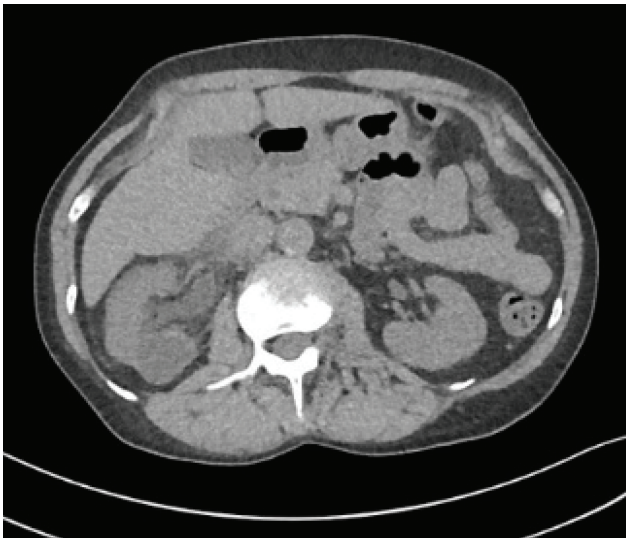

**Figure:** Pure indinavir calculi are radiolucent.[15]

**Drug-Induced Nephrolithiasis:**

**1) Ephedrine calculi:** Ephedrine and its metabolites (norephedrine, pseudoephedrine, and norpseudo-ephedrine) are sympathomimetic agents used for the treatment of enuresis, myasthenia gravis, narcolepsy, and rhinorrhea.[22] Ephedrine calculi are radiolucent.[23]

**2) Indinavir calculi.**

**3) Guaifenesin calculi:**

Guaifenesin is a widely used expectorant that has recently been reported to be associated with nephrolithiasis. They are found in patients consuming guaifenesin in excessive amount.[24]

**4) Xanthine calculi.** These stones occur due to a hereditary disorder called xanthinuria. The deficiency in xanthine oxidoreductase enzyme results in decreased levels of serum and urinary uric acid and high concentration of urinary xanthine that leads to formation of xanthine stone.[25] Patients who receive allopurinol treatment, also develop xanthine oxidoreductase deficiency, eventually causing iatrogenic xanthiuria that results in xanthine crystal formation.[25]

**Summary table of drugs causing kidney stone disease:**

| Drug Induced Urolithiasis | Drug-Induced Nephrolithiasis |
|---|---|
| Indinavir | Ephedrine<br>Guaifenesin |
| Ritonavir | Indinavir<br>Xanthine |

**Size of urinary tract stones:**

**Associated Symptoms of Urolithiasis:**

**1) Pain:**

While tiny urinary tract stone(s) may not cause any symptoms[1,2], larger calculi may cause sharp pain in the side and back, below the ribs, radiating to the lower abdomen and groin[1]

**Renal colic:** Stones obstructing the ureter or renal pelvis or any of kidney's drainage tubes may cause back pain or renal colic characterized by an excruciating intermittent pain, usually in the area between the ribs and hip on one side, radiating across the abdomen and often extends to the genital area. The pain is likely to come in waves, gradually increasing to a peak intensity, then fading, over about 20 to 60 minutes.[2]

**2) Urine:**

The colour may be pink, red or brown, may look cloudy or foul-smelling and the patient feels persistent need to void urine more often than usual but in small amounts.[1]

**3) Other signs & symptoms:**

Other symptoms include nausea vomiting, restlessness, sweating, passing out stone/piece of stone with the urine. Fever and chills also can present if there is an infection.[1]

**Diagnosis of Urinary tract Stone:**

Physicians usually suspect stones in people with renal colic, tenderness over the back/pain or in genital area without any obvious cause. Presence of blood in urine support the diagnosis. Sometimes symptoms and physical examination remain so distinctive that additional tests are often not needed, especially for the patients with a history of urinary tract stones.[2]

**Imaging techniques used to diagnose urolithiasis:**

**1) Non-contrast computed tomography:** Non-contrast CT or CT-KUB are most often used in patients with nephrolithiasis. CT creates a 3D image of the stone and the surrounding tissues, which can be reconstructed into multiple viewing planes. The sensitivity of CT for detecting kidney stones is the highest among all the imaging techniques (~95%). Limitations of CT: radiation exposure, double cost compared to USG.[26]

**2) Ultrasonography:** Ultrasonography is a low-cost imaging modality that does not depend on ionizing radiation, thus sparing the patients from the risk of radiation exposure. Although ultrasonography is less

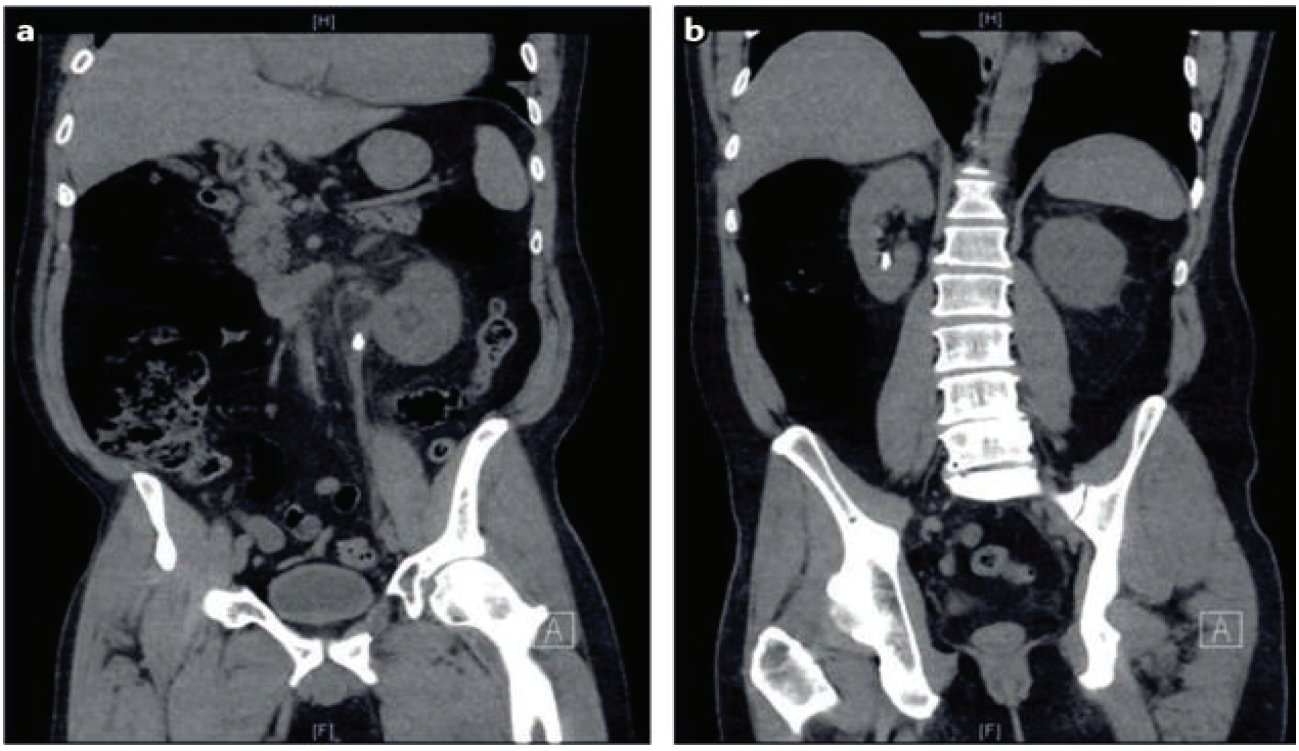


**Figure:** A coronal demonstration of bilateral 8 mm nephrolithiasis on non-contrast CT[26]

sensitive and specific than CT imaging for detecting and sizing of stones, it has good diagnostic ability, and can effectively detect hydronephrosis. USG is recommended as the first-line imaging modality for pregnant and paediatric patients (<14 years old). Modern ultrasonography has a sensitivity of about 80%.[26]

**3) KUB radiography:** KUB radiography allows relatively low ionizing radiation exposure to patients compared with CT (0.15 mSv) and it is cost effective. Nevertheless, as radiography views stones only at one angle, accuracy is decreased that causes reduced sensitivity and specificity and, therefore, limiting its usefulness. Many stone types can be visualized using KUB radiography; although cystine and struvite stones often are poorly visible on KUB radiography, and uric acid and matrix stones are not visible at all.[26]

**4) MRI:** The sensitivity of MRI (82%) is higher than that of USG and KUB radiography but less than that of CT.[26]

**5) Excretory urography:** (previously known as intravenous urography or intravenous pyelography) It is a series of x-rays taken after a radiopaque contrast agent is injected intravenously. This test can detect stones and accurately determine the degree to of blockage of the urinary tract caused by the stone. But it is time-consuming and involves the risks of exposure to the contrast agent.[2]

**Complications of Urolithiasis:**[27]

1) Acute renal failure secondary to obstruction.
2) Anuria.
3) Urinary tract infection with renal obstruction.
4) Sepsis.

**Hydronephrosis:**

It is a clinical condition where the kidney becomes distended due to obstruction in urine outflow. Urine flows back behind the obstruction and remains trapped inside renal pelvis, eventually causing a distended kidney.[28]

**Etiology of Hydronephrosis:**

- In children: Structural abnormalities - congenital anomalies such as posterior urethral valves and other constrictions that narrow or block the ureter or urethra
- In young adults: Large stones in a kidney or in ureter or elsewhere in the urinary tract.
- In older adults: Benign prostatic hyperplasia (BPH) or prostate cancer, tumors, and stones.

As BPH is so common in older males, obstruction is more common among men. Another common cause of obstruction might be stricture (narrowing caused by scar tissue) of the ureter or urethra that can develop after radiation therapy, surgery, or any procedure done on the urinary tract.[28]

A urinary tract infection may result when micro-organisms, especially bacteria become trapped in urine around a blockage by any/few stone(s), for a longer period of time when urine backs up in the tubes inside the kidney, causes hydronephrosis.[29]

**Management of Urolithiasis:**

**Conservative management:** Small stones that are not causing any symptom, blockage or infection of urinary tract are likely to pass with urine. Larger stones (over 5 mm) and those that are closer to the kidney are less likely to pass on their own. Several medications (tamsulosin or calcium channel blockers) may increase the likelihood of spontaneous stone passage.[2]

**History of urinary tract stone removal:** In 1561, Pierre Franco performed the first suprapubic lithotomy. In 1874, a lithotrite was developed by Bigelow, which was which was introduced into the bladder under anaesthesia (called as ***"litholopaxy"***). Young was first reported to use ureteroscopy in 1929. With the introduction of intracorporeal lithotripsy techniques, ureteroscopy became the treatment of choice for ureteric stones. In 1976, Fernstrom and Johannson initiated percutaneous access to remove a renal stone. However, with the introduction of the first extracorporeal shock wave machine in 1980 stone management was changed significantly.[30]

**Nowadays, the following methods[31] are used for urinary stone removal:**

**1) Shock wave lithotripsy** can be used to break up a stone in the renal pelvis or uppermost part of the ureter that is ½ inch (1centimeter) or less in diameter.

**2)** A **ureteroscope** (a kind of endoscope) can be inserted into the urethra, through the bladder and up the ureter to remove small stones in the lower part of the ureter.

**3) Percutaneous nephrolithotomy** is used to remove some larger kidney stones.

**Recurrence of urolithiasis:**

On average, around 30% to 50% of patients, show a probability of recurrence or another stone attack within 3 to 5 years.[31]

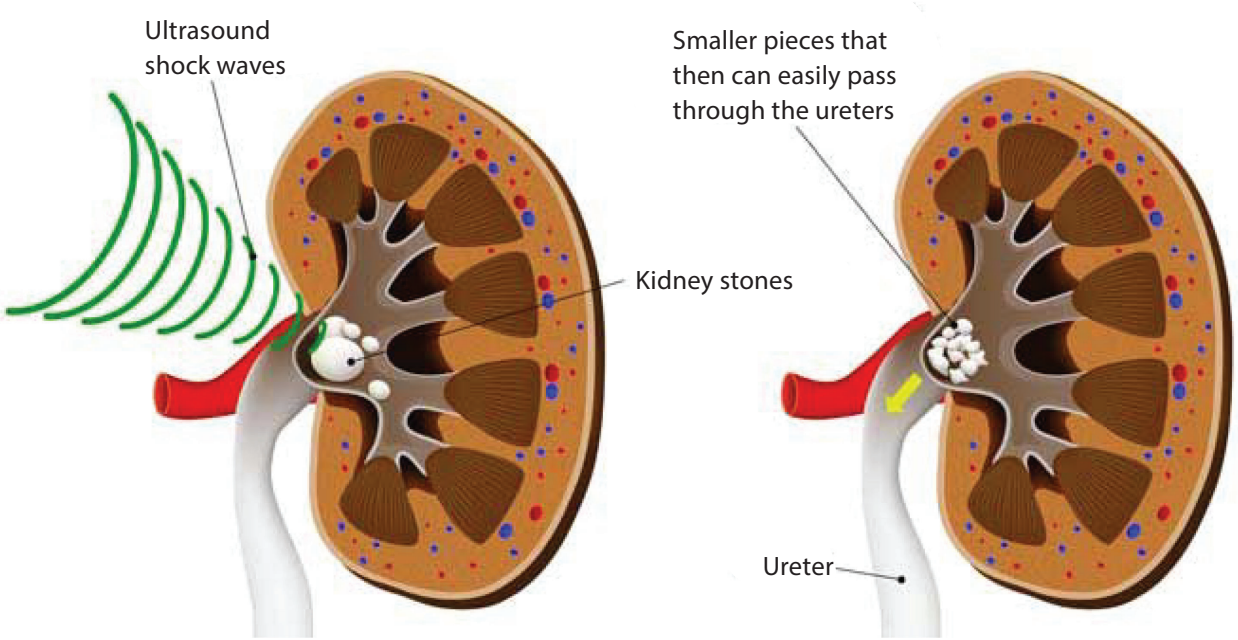


**Figure:** Shockwave lithotripsy[31]

**Prevention of Urolithiasis:**[32]

1) Drinking plenty of water.
2) Avoiding excessive intake of salt.
3) Increasing consumption various types of nuts.
4) Limiting the intake of animal protein.
5) Eating citrus fruits.

**Kampo Medicine in Urinary Stone Disease**

Kampo medicine originated in China had been used for the cure and the prevention of urinary calculi for years, but the effect and the mechanism of this use of kampo medicine are unclear.[16]

**Urolithiasis in pregnancy:**

Urolithiasis in pregnancy is an important health issue. It is one of the most common causes of non-obstetrical abdominal pain and hospital admission during pregnancy.[33]

1 in 3300 pregnancies show symptomatic urolithiasis that complicates pregnancy.[34]

Urolithiasis during pregnancy requires multi-disciplinary approach concerning both department of urology and department of obstetrics and gynaecology.[35]

The first choice of treatment is conservative management and trial of passage with hydration and analgesia. NSAIDs are generally avoided during pregnancy and narcotics are usually administered. This pathway requires a solitary stone <1cm, no infection, adequate oral pain control ability to tolerate food and fluid. The success rate is 70%–80% and 50% of those without spontaneous passage during pregnancy are likely to pass their stones after delivery.[36-42]

**Childhood Urolithiasis - A Paradigms Shift:**

Despite being recognized in children for centuries, the clinical features, evaluation and management of urolithiasis are still evolving. Approximately 7% of all stones occur in children younger than 16 years.

In the past, urolithiasis used to be characterized by bladder stone in children in developing countries; with the incidence of upper tract calculi mostly occurring in industrialized areas, being much lower in children than adults.Nowadays, the incidence of upper tract calculi in children is experiencing a rise globally**,** and the patterns are also changing**.** Smaller endoscopic instruments and the refinement of extracorporeal shock wave lithotripsy (**ESWL**) technology have made treatment of **pediatric stone disease** easier.[4]

However, pediatric urolithiasis is being studied in Ad-Din Women's Medical College, Dhaka, Bangladesh by Prof. ARML Kabir.

**Salient features:**

- Although all the urinary tract stones start to originate inside the kidneys, they are named as per their location, thus urolithiasis and nephrolithiasis remain two analogous terms for urinary calculi.
- Multiple factors facilitate the stone forming process.
- CT scan is the choice of diagnosis option.
- Conservative management is the major approach to treat smaller stones, but larger stones require surgical management.
- There is a chance of recurrence in 30-50% patients.

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## Review Article

# Testicular Tumour

Sardar Rezaul Islam[1], Maruf Raza[2], Debabrata Paul[3], Shah Alam Sarkar[3], Sardar Saminul Islam[4], Md. Hanif[5]

**Abstract**

Testicular cancer is rare, but it is the commonest cause of malignancy in young men. Painless scrotal masses must be investigated with ultrasound imaging and tumour marker assay before being treated with radical inguinal orchidectomy. For unknown reasons, the incidence of this cancer increased in Caucasian population. The incidence of testicular germ cell tumour has doubled in past 40 years. An annual increase of 3-6% is reported in Caucasian population. But the mortality rate has been stable or decreasing due to improvement in treatment.

In the past, metastatic testicular cancer was usually fatal, but recent advances in treatment, including high-dose chemotherapy and stem cell rescue, have considerably improved the prognosis. Indeed, testicular cancer is a bright spot in the oncological landscape and are now considered the model for the treatment of solid tumors. We looked into the epidemiology, presentation, classification, work up, staging, various treatment modalities and prognosis of testicular tumour in this article.

**Key words:** Testicular tumour.

### Introduction

Primary testicular tumors are the most common solid malignant tumor in men between the ages of 20 and 35 years . For unknown reasons, the incidence of this cancer has increased during the last century.[1] Most testicular tumours are derived from the germ cells of the testis, although about 5% of testicular tumours may be derived from other cells, including Leydig cells and lymphocytes (lymphoma).[2] The cause of testicular tumours is unknown, but several predisposing factors are recognized.

The classification of testicular germ cell tumours is confusing because different systems and non-seminomatous components. The USA and WHO classifications focus particularly on the range and degree of differentiation of tissue types within the tumours, whereas the British classification regards all mixed tumours as teratomas (because of their capacity to produce tissues from all three germ layers) and then bases secondary classification on the degree of differentiation.

1. Professor, Department of Surgery, Ad-din Women's Medical College (AWMCH), Dhaka
2. Professor, Department of Pathology, Jahurul Islam Medical College Hoispital (JIMCH), Bajitpur, Kishoreganj.
3. Assistant Professor, Department of Surgery, JIMCH, Bajitpur, Kishoreganj.
4. Registrar, Department of Emergency, AWMCH, Dhaka
5. Registrar, Deptartment of Surgery, ASMCH, Jessore

**Correspondence:** Prof. Dr. Sardar Rezaul Islam, Professor, Department of Surgery, AWMCH, Dhaka. E-mail: islamreza91@gmail.com

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Any solid, firm mass within the testis should be considered testicular cancer until proven otherwise. Prompt diagnosis and early treatment are required for cure. Testicular cancer may be painless, in which case they are sometimes ignored by the patient. In patients with scrotal pain, testicular cancer must be differentiated form epididymitis. The clinician should consider the full differential diagnosis of a testicular mass, which includes not only epididymitis but epididymo-orchitis, testicular torsion hydrocele, hernia, hematoma, spermatocele, varicocele, and syphiliticgumma.[3]

Testicular cancers are highly curable, even in patients with metastatic disease at diagnosis. The prognosis depends upon the histologic type of cancer (seminoma versus nonseminoma), stage, and other features such as tumor marker and type of metastatic disease. Cure rates for good-risk disease are nearly 90-95%.

Various risk factors have been associated with testicular tumors, but the specific etiology is not known. Cryptorchidism, genetic predisposition, family history and prior testicular cancer are important etiological factor. Undescended testis has forty times more chance of developing testicular cancer. (Fig-10 &11)

Painless swelling or nodule of one testicle is the most common presenting symptom. On the physical exam this mass or nodule cannot be separated from the testis. Dull ache or heavy sensation in the lower abdomen could be presenting symptom. Patients who experience a hematoma with trauma should undergo evaluation to rule out testicular cancer.

Disseminated disease have symptoms of lymphatic or hematogenous spread. Presenting symptom could be neck mass in supraclavicular lymph node, anorexia, nausea and other gastrointestinal symptom. Bulky retroperitoneal disease could present as back pain. Cough, chest pain, hemoptysis and shortness of breath could be presenting symptom of mediastinal adenopathy or lung metastatic disease.

**Classification**

Approximately 95% of testicular tumors are germ cell tumors. These are divided into two types: pure seminoma (no non-seminomatous element) & non-seminomatous germ cell tumors.[4]

A. Germ cell tumour
   1. Seminoma
   2. Non seminma (Teratoma)

B. Mixed germ cell tumour

C. Yok sac tumour

D. Sex cord stromal tumor (Sertoli cell, Lyedig cell tumor)

E. Lymphoma

**Seminoma**

In addition to pure seminomas, which constitute roughly 50% of pure germ cell tumors, a seminomatous component is present in 20% of mixed germ cell tumors. Serum tumor markers are usually at normal levels, but if syncytiotropho-blastic giant cells are present, beta-hCG may be elevated. Seminoma looks like clear or vacuolated cells with well-defined cell margin. Most tumours have variable degree of lymphocytic infiltrate. (Fig-7). Classical seminoma has three variant-cribriform, pseudo-glandular and tubular.

**Non-seminoma**

- Embryonal carcinomas constitute about 2% of all testicular germ cell tumors but are histological type in 85% of mixed germ cell tumors. They have large pleomorphic cells with different architectural patterns (fig-6).
- Teratomas are part of the mixed germ cell tumor and are generally benign but have the potential for metastasis. They have elements from all three germ layers: ectoderm, endoderm, and mesoderm. In patients with residual disease after chemotherapy, teratoma is found in approximately 45% of resected specimens.
- Choriocarcinomas are the least common type of non-seminoma but are very aggressive. Widespread hematological metastasis can occur very early in the disease course; the retroperitoneum may be spared. Choriocarcinomas are associated with increased levels of beta-hCG.
- Yolk cell tumors, also called endodermal sinus tumor, are the most common testicular tumor in infants and young children. In adults, pure yolk cell tumors are rare, but yolk cell elements are found in approximately 40% of mixed germ cell tumors. Yolk cell tumors are associated with elevated alpha fetoprotein levels but they do not produce beta-hCG.

Mixed germ cell tumors (those containing two or more germ cell types) constitute approximately one third of testicular cancer (fig-2). Mixed germ cell tumor behaves like non-seminomas. The average age at diagnosis is older than 30 years. (fig-1)

**Diagnostic workup**

Ultrasonogram of the scrotum- USG has 100% accuracy for diagnosis of testicular malignancy. Seminoma has hypoechoic homogenous appearance. Non-seminoma appears as complex cystic and solid masses (fig-5)

USG of the abdomen and Chest x-ray are minimum requirement for staging of the tumour.

USG or CT Scan of the abdomen is necessary to see the enlargement of the para-aortic lymph nodes in the abdomen. (fig-4)

Chest x-ray shows pulmonary metastasis in the form of multiple cannon ball especially in non-seminoma. (fig-3)

**Tumour markers**

Blood must be taken for marker evaluation before surgical removal of the testis. This is important for staging and also for postoperative follow up and to know the response of the treatment and surveillance.

α-fetoprotein (AFP) is produced by the yolk sac elements and is elevated in 50-70% of NSGCT. It is not usually elevated in pure seminoma. Its half-life is about 5 days.

β-human chorionic gonadotrophin (β-HCG) is produced by trophoblastic elements in the tumour. It is raised in 40-60% of NSGCTs & in up to 30% of pure seminomas. It has a half-life of 1 day.

Lactate dehydrogenase (LDH) is less specific, but is more common in seminoma.

Overall, 90% of NSGCTs elaborate at least one tumour marker, while markers are elevated in <40% of seminomas.[5]

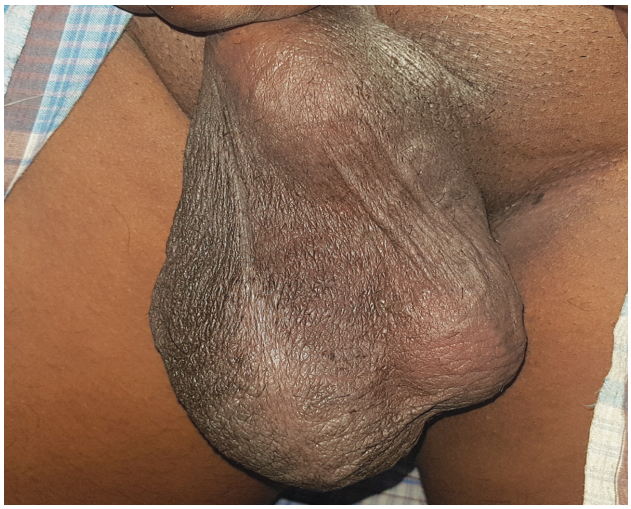


**Fig-1**

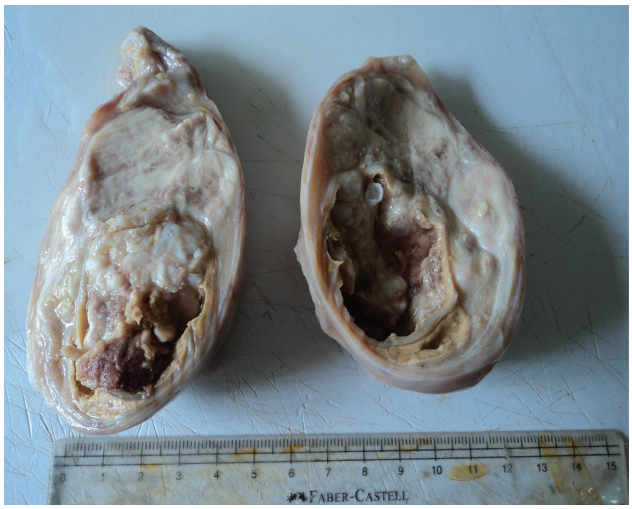
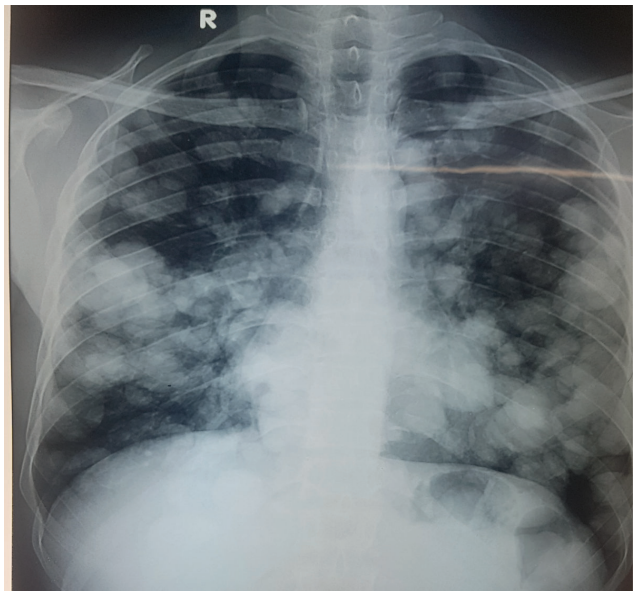



**Fig-2**

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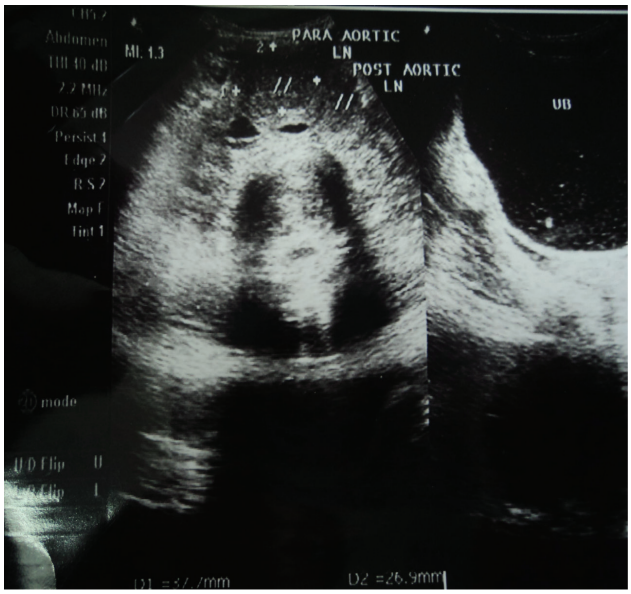


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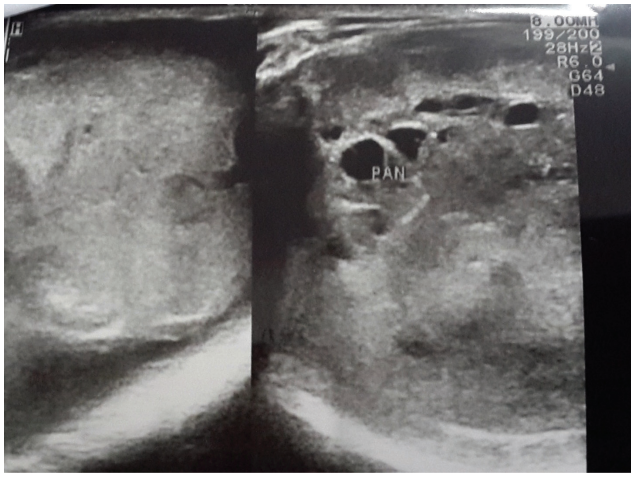


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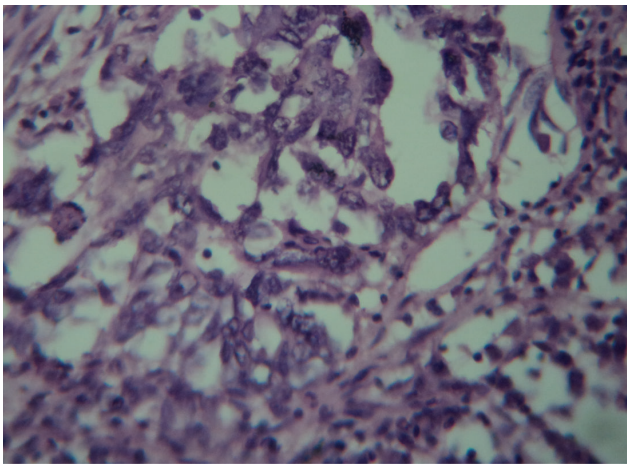

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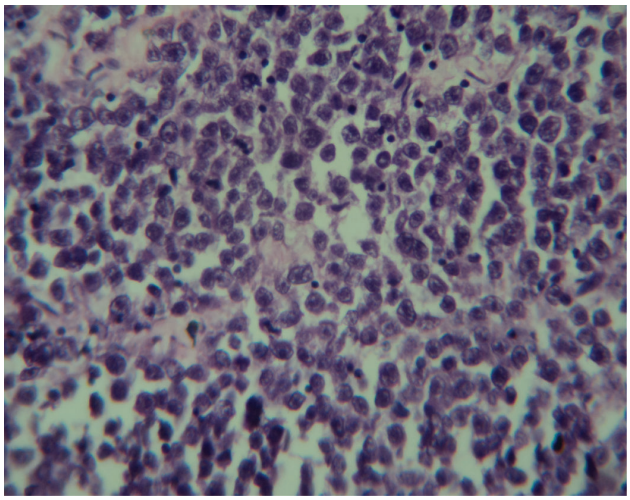

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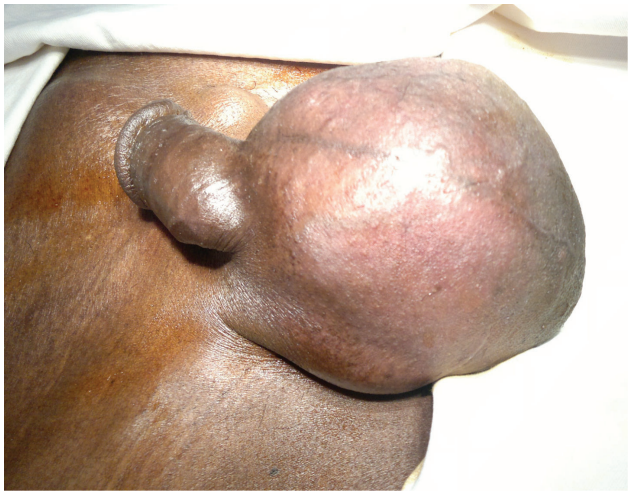

**Fig-8**

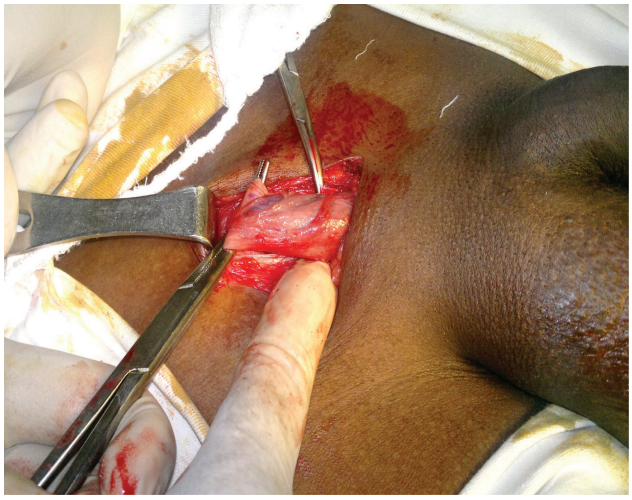

**Fig-9**

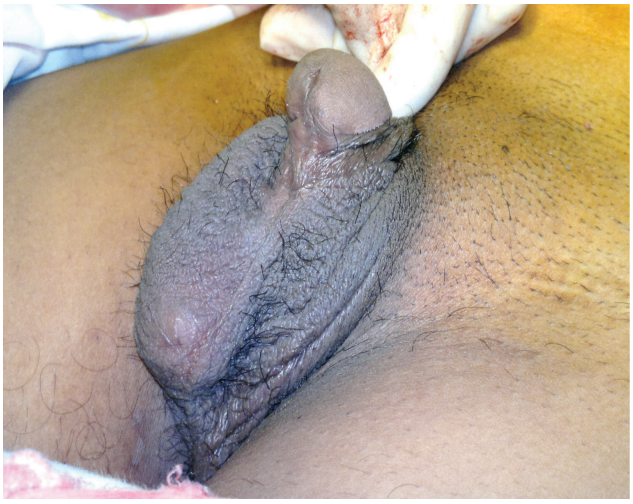

**Fig-10**

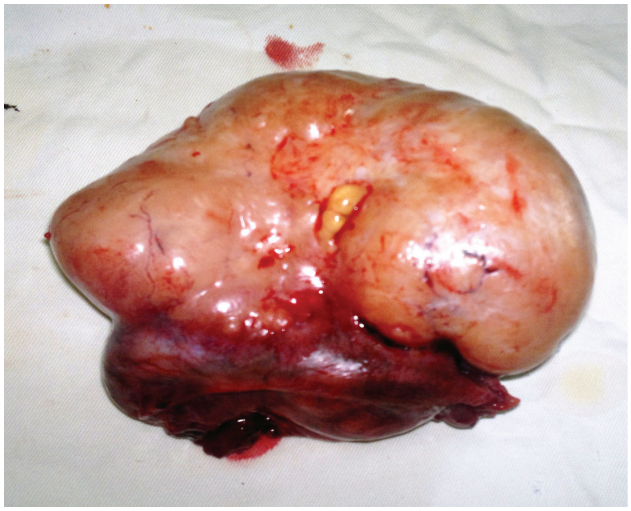

**Fig-11**

**Table-I:** Pattern of pathology of testicular tumour in JIMCH in last 4 years

| Age in years | Pathology |
|---|---|
| 35 | Mixed emryonalcarcinoma+ Choriocarcinoma |
| 32 | Seminoma ((Intra-abdominal) |
| 35 | Seminoma (Intra-abdominal |
| 45 | Seminoma |
| 33 | Seminoma |
| 18 | Teratocarcinoma |
| 24 | Teratoma |
| 20 | Mixed embryonal carcinoma+ Seminoma |
| 25 | Yolk sac tumour |

**Table-II:** Distribution of Pathological subtypes of GCT, based on 649 patient of Royal Marsden Hospital, UK.[6]

| Type of pathology | Percentage |
|---|---|
| Pure seminoma | 360(55%) |
| Embryonal carcinoma | 87(13.5%) |
| Teratocarcinoma | 75(11.5%) |
| Choriocarcinoma | 12(1.8%) |
| Yolk sac tumour | 8(1.2%) |
| Teratoma | 24(3.7%) |
| Mixed Embryonal carcinoma Seminoma | 58(8.9%) |
| Mixed Teratocarcinoma Seminoma | 17(2.6%) |
| Mixed Choriocarcinoma Seminoma | 1(0.2%) |
| Mixed Teratoma seminoma | 8(1.2%) |

According to Table II, more than half of the GCT was pure Seminoma (55%), followed by Embryonal Carcinoma (13.5%) and Teratocarcinoma (11.5%). The next were Mixed Embryonal Carcinoma Seminoma (8.9%) and Mixed teratocarcinoma Seminoma (2.6%).

**Staging**

American Joint Committee on Cancer (AJCC) groupings recommend use both TNM and Serum tumor markers for staging.[7]

**TNM classification**

Primary tumor (T)

TX- Tumor cannot be assessed

T0- No evidence of primary tumor (after radical orchiectomy)

T1-Tumor is limited to the testis and epidemic without vascular invasion

T2-Tumor limited to the testis and epidemic with vascular invasion or to the tunica vaginalis

T3- Tumor invaded the spermatic cord

T4- Tumor invades the scrotum

Lymph nodes (N)

NX- Regional lymph nodes cannot be assessed

N0- No regional lymph nodes

N1- Enlarged regional lymph nodes <2 cm in dimension

N2-Lymph nodes 2-5cmm in dimension

N3- Lymph nodes > 5 cm in dimension

Distant metastasis (M)

MX-Distant metastasis cannot be assessed

M0- No distant metastasis

M1-Non-regional nodal or pulmonary metastasis

Staging according to the level of Tumour marker

Sx indicates tumor markers unavailable or not done. S0 indicates tumor markers are within normal limit. Following table indicates other S categories.[8]

**Final staging**

- Stage I-pT1–4, N0, M0, SX
- Stage IA-pT1, N0, M0, S0
- Stage IB-T2-4, N0, M0, S0
- Stage IS-Any pT/Tx, N0, M0, S1–3
- Stage II-Any pT/Tx, N1–3, M0, SX
- Stage IIA-Any pT/Tx, N1, M0, S0-1
- Stage IIB-Any pT/Tx, N2, M0, S0-1
- Stage IIC-Any pT/Tx, N3, M0, S0-1
- Stage III-Any pT/Tx, any N, M1, SX
- Stage IIIA-Any pT/Tx, any N, M1, S0-1
- Stage IIIB-Any pT/Tx, N1–3, M0-1, S2
- Stage IIIC-Any pT/Tx, N1–3, M0-1, S3

**Risk classification**

Good- risk non-seminoma

- Testicular or retroperitoneal primary tumor, and
- Non-pulmonary visceral metastases, and
- Good markers-S1

**Table-III**

| Stages | LDH | AFP (ng/ml) | HCG (mIU/ml) |
|---|---|---|---|
| S1 | <1.5 times than normal | < 1000 | < 5 000 |
| S2 | 1.5-10 times than normal | 1 000-10 000 | 5 000-50 000 |
| S3 | >10 times than normal | >10 000 | >50 000 |

Table III is presenting the stages of tumor and the level of tumor markers present in each stage.

Intermediate- risk non-seminoma

- Testicular or retroperitoneal primary tumor, and
- Non- pulmonary visceral metastases,
- Intermediate marker-S2

Poor risk non-seminoma

- Mediastinal primary, or
- No pulmonary visceral metastases, or
- Poor markers-S3:

Good-risk seminoma

- Any primary site, and
- No non-pulmonary visceral metastases, and
- Marker-S1

Intermediate-risk seminoma

- Any primary site, and
- Non-pulmonary visceral metastases, and
- Marker-S1

Poor-risk seminoma

No patients are classified as poor prognosis9

**Treatment**

Initial therapy is selected according to AJCC stage group; risk stratification (good, intermediate, or poor risk), as per the guidelines of the International Germ Cell Cancer Collaborative Group 7; and histology (seminoma versus nonseminoma). [8,9]

Current guidelines from the National Comprehensive Cancer Network (NCCN) and the National Cancer Institute recommend treatment approach keyed to AJCC staging. These treatment groups are as follows:

Initial therapy consists of radical orchiectomy via inguinal approach. (Fig-9)

**Seminoma management**

**Stage I**

Clinical stage I seminoma have a very high cure rate. Cure can sometimes be achieved by radical inguinal orchiectomy alone. Options after orchiectomy include active surveillance, adjuvant chemotherapy, and adjuvant radiation therapy. Median time to relapse in patients who do not receive adjuvant treatment is 12 months but relapse can occur even beyond 5 years.

Active surveillance is recommended for patients with horseshoe or pelvic kidney or inflammatory bowel disease and for those who have received prior radiotherapy. Surveillance can also be offered to selected patients with T1 or T2 disease. Surveillance consists of a history and physical exam and measurement of AFP and hCG every 3 to 4 months for the first 3 years, every 6 months for years 4 to 7, then annually up to year 10. A CT scan of the abdomen and pelvis is recommended at each visit and a chest x-ray at alternate visits. It is essential that patients maintain strict adherence to the surveillance program for at least 10 years.

Adjuvant radiation therapy consists of delivery of 20-30 Gy to the infradiaphragmatic area, including th para-aortic lymph nodes and in some cases the ipsilateral ileoinguinal nodes. According to surveillance data, the overall incidence of disease failure without radiation therapy is 15% to 27%, with median of 20%. With radiation therapy, failure rates were 2% to 5%, with a median of 3%.

Adjuvant chemotherapy with a single dose of carboplatin is currently recommended as an alternative to radiation therapy. In a randomized study in 1,477 patients, after a median follow-up of 4 years there was no difference in relapse-free survival between patients receiving single-dose carboplatin and those receiving radiation therapy.[10] Five-year follow-up in 1,148 patients from this trial showed relapse-free rates of 96% for the radiation arm and 94.7% for the carboplatin arm. However, there were 15 new germ cell tumors in the radiation therapy arm versus two in the carboplatin arm, giving a hazard radio of 0.22 (95% CI 0.05, 0.95 p = 0.33). Acute toxicity such as lethargy and days missed from work is less with carboplatin than with radiation therapy.

**Stage-II**

Active surveillance is not an option. These patients receive adjuvant chemotherapy or radiation therapy

Radiation therapy: 35-40Gy is administered to the infradiaphragmatic area, including the para-aortic and ipsilateral iliac lymph nodes. Mediastinal radiation is not recommended.

Adjuvant chemotherapy: Four courses of chemotherapy with etoposide and cisplatin (EP) may be given.11

**Stage-III**

Seminoma stage IIC and III, good risk: Either four cycles of EP or three cycles of bleomycin, etoposide, and cisplatin (BEP)

- Seminoma stage IIC and III, intermediate risk: Four cycles of BEP

**Nonseminoma management**

**Stage I**

After radical inguinal orchiectomy, treatment options are active surveillance or chemotherapy. Retroperitoneal lymph node dissection (RPLND) is used to guide chemotherapy; the number of positive nodes present in the sample determines the number of chemotherapy cycles given. Open nerve-sparing RPLND is preferred over laparoscopic RPLND. RPLND has multiple complications of with (RPLND), including retrograde ejaculation.

**Stage-II**

Recommended treatment varies according to the results of tumor marker assays and CT scan.

Nonseminoma with normal tumor markers: open nerve-sparing RPLND or chemotherapy, either EP for 4 cycles or BEP for 3 cycles.

**Stage-III**

- Nonseminoma stage IIIA good risk: 95% of patients are cured with chemotherapy, either EP for 4 cycles or BEP for 3 cycles.
- Nonseminoma stage IIIB intermediate risk: BEP for 4 cycles is given; the cure rate is 70%.
- Nonseminoma stage IIIB poor risk: Enrollment in clinical trials is preferred. Chemotherapy with 4 cycles with BEP can be considered but fewer than 50% of patients will experience a durable complete response. In patients who cannot tolerate BEP because of pneumonitis from the bleomycin component, VIP (etoposide [VePesid], ifosfamide, mesna, cisplatin [Platinol-AQ]) is recommended.

**Surgery for residual disease**

Surgical resection is recommended for patients with residual disease after chemotherapy.[12] Laparoscopic LN dissection is recommended for stage 1 and 2 disease.[13] Retroperitoneal lymph node dissection (RPLND) should clear the region of residual disease. Open nerve-sparing RPLND is preferred over laparoscopic RPLND. Patients in whom RPLND reveals viable cancer, post chemotherapy residual masses are treated with subsequent chemotherapy.[13,14] Open nerve-sparing RPLND has multiple complications, including retrograde ejaculation.

**Prognosis**

Good-prognosis nonseminoma): 5-year survival is 90%.[15]

Good-prognosis seminoma (90% of seminomas): 5-year survival is 85%

Intermediate-prognosis nonseminoma: 5-year survival is 80%

Intermediate-prognosis seminoma: 5-year survival is 70%

Poor-prognosis nonseminoma: 5-year survival is 70%

Poor-prognosis s seminoma: No seminoma patients are classified as poor prognosis.

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Case Report

# Co-occurrence of Rickets and Scurvy- A case report

Sudipta Roy [1], ARM Luthful Kabir[2], Rahat Bin Habib[3], Masuma Khan[4], Armin Sultana[5], Ashraf Uddin Ahmed[6]

**Abstract:**

Now a days, rickets and scurvy are considered rare except in certain situations. Co-occurrence of rickets and scurvy develops in a child when the risk factors of both conditions present simultaneously. Infant and young children are more vulnerable to micronutrient deficiencies. Appearance of clinical and radiological features of both conditions are dependent on the order and length of first disease prior to the development of the 2nd illness. One of the two diseases will dominate over the other and mask the features of the non-dominant disease. Screening for rickets should be considered for children with poor growth/ development, seizure activity/tetany, and children with chronic malabsorptive states and scurvy should be evaluated to exclude co-occurrence. Here, we report a 14 months old female child presented with cough and respiratory distress for 2 months and poor weight gain since early infancy. She was a preterm, low birth weight baby on exclusive breast feeding without any vitamin supplementation. She had history of inadequate sunlight exposure. Her current diet was lack of fruits and vegetables. Incidentally, she was found having rachitic changes. Her radiology showed features of both rickets and scurvy. So, finally she was diagnosed as a case of persistent pneumonia, co-occurrence of scurvy and rickets with failure to thrive. She was treated with vitamin D, Calcium, Vitamin C, antibiotics and other supportive measures.

**Key words:** Rickets, Scurvy, Micronutrient deficiency, Preterm, Low birth weight (LBW)

## Introduction:

Scurvy and rickets occur simultaneously when a culture or society has risk factors for both conditions. Diet is one of the most important factors for developing a co-occurrence of both conditions. [1] Scurvy is the disease caused by a deficiency in vitamin C. Vitamin C, is a water soluble vitamin and an essential nutrient requiring to obtain from the diet. Vitamin C rich foods include fresh fruits, vegetables, and human breast milk, with small amounts in raw liver. A deficiency of this vitamin is caused by a deficient diet and/or poor nutrient absorption.[2] Vitamin D deficiency (called nutritional rickets) is the most common and typically due to a lack of ultraviolet B (UVB) ray exposure, dietary deficiency and malabsorption in the gut of vitamin D.[3] Infants and young children are more vulnerable to micronutrient deficiencies for rapid growth and use up their stores.[4] Now a days, rickets and scurvy are considered rare which develop only in peculiar situations. Lewis et al recently reported cases of scurvy and rickets have been documented in refugee and ethnic minority populations.[5] Moreover, infants and children are immunocompromised with increased risk of acquiring infectious or diarrhoeal diseases which can causes malabsorption of important nutrients. [4] Socioeconomic status is another risk factor as it may limit access to vitamin C and D by restricting diet and by adopting particular behaviours. After weaning, a diet rich in vitamin C and vitamin D is essential, with adequate sunlight exposure.[6] Here, we report a rare case of 14 months old girl who presented with persistent pneumonia and poor weight gain ,incidentally, she was found having rachitic changes and his radiological findings were correspond with both rickets and scurvy.

1. Associate Professor, Department of Paediatrics, Ad-din Womem,s Medical College Hospital (AWMCH), Dhaka, Bangladesh
2. Professor &Head, Department of Paediatrics, AWMCH
3. Assistant Professor, Department of Paediatrics, Saheed Sayed Nazrul Islam Medical College, Kishoreganj.
4. Assistant Professor, Department of Paediatrics, AWMCH
5. Registrar, Department of Paediatrics, AWMCH
6. Professor, Department of Paediatrics, AWMCH

**Correspondence:** Dr. Sudipta Roy, Mailing address: sudipta.paediatrics@gmail.com
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## Case summary

Mitul, a baby girl of 14 months, 3rd issue of non-consanguineous parents, immunized, belongs to a lower class family was admitted on March,2020 in pediatric ward in AWMCH hospital with the complaints of cough and respiratory distress for 2 months and poor weight gain from early infancy. Previously she was hospitalized and diagnosed as a case of persistent pneumonia. She was being treated with several antibiotics with no cure, hence was referred to our hospital for better evaluation.

She was delivered normally at preterm with low birth weight (1500 gm) with uneventful perinatal period but

she did not receive any vitamin and iron supplementation. She was given exclusive breast milk for 6 months. Then complementary feeding was started but poor in quality. She was never offered fresh vegetables and fruits, meat, fish ,egg etc. On query, her exposure to sunlight was also inadequate, most of the time she used to spend in indoor. Her milestone of development was age appropriate.

She had no h/O recurrent loose motion, skin lesions, easy fatiguability, limb pain, gum bleeding or contact with tuberculosis patients. All other family members were in good health.

**Physical examination**: Mitul had distressed look with intercostal recession, respiratory rate-50/min, Pulse-180/min, Temperature $98^0$F, SPO2-86%, incidentally she was found having rachitic changes such as box shaped head with wide open anterior fontanelle, widening of costochondral junctions of ribs, widening of both wrists and ankles without sign of inflammation. Her weight 6.4 Kg, height 68cm, upper segment- 40 cm, lower segment-28 cm, U:L – 1.4(normal), occipitofrontal circumference (OFC) -46 cm ($50^{th}$ centile), mid upper arm circumference (MUAC) – 115 mm, WAZ -3.8 (severe wasting), HAZ- 2.7 (moderate stunting). On auscultation, there were crepitations on left lung field but no murmur. Other systemic examination revealed no abnormalities.

**Investigations:**

Complete blood count : Hb- 12.0g/dl, white blood cell-16,000/cmm, neutrophil-65%- neutrophilic leukocytosis reflected infection S. calcium- 10mg/dl(normal), S.Phosphate - 2.20mg/dl (normal) , alkaline phosphatase - 610u/L( ↑), parathyroid hormone(PTH) 132 p g /ml (↑) suggestive of rickets, however parents refused to perform vitamin D level which is a costly investigation.

X ray of wrist joints and lower limbs revealed features of both rickets and scurvy-

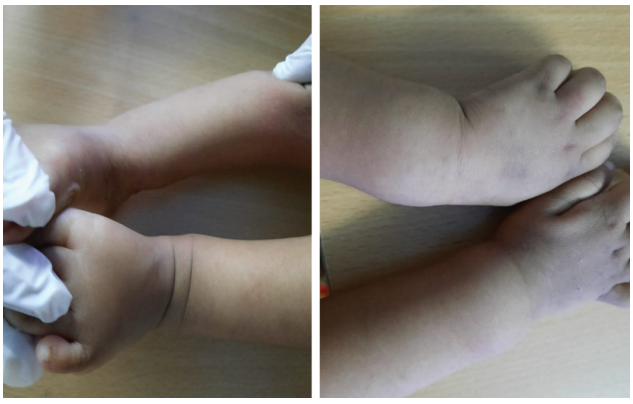

Fig.-1: *Widening of both wrists and ankles*

**Fig. 2:** Picture of the child

Rickets- Fraying, widening and cupping of metaphysis of lower ends of radius, ulna and femur and upper and lower ends of tibia and fibula (Fig-3,4)

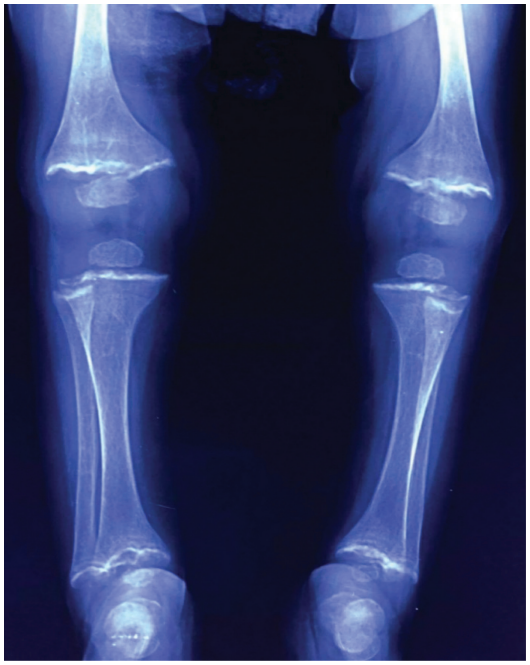

**Fig 3.** Rickets- Fraying, widening, cupping of metaphysis of long bones, Scurvy- White line of Frankel, scurvy line, wimberger ring sign of epiphysis and tarsal bones and osteopenia

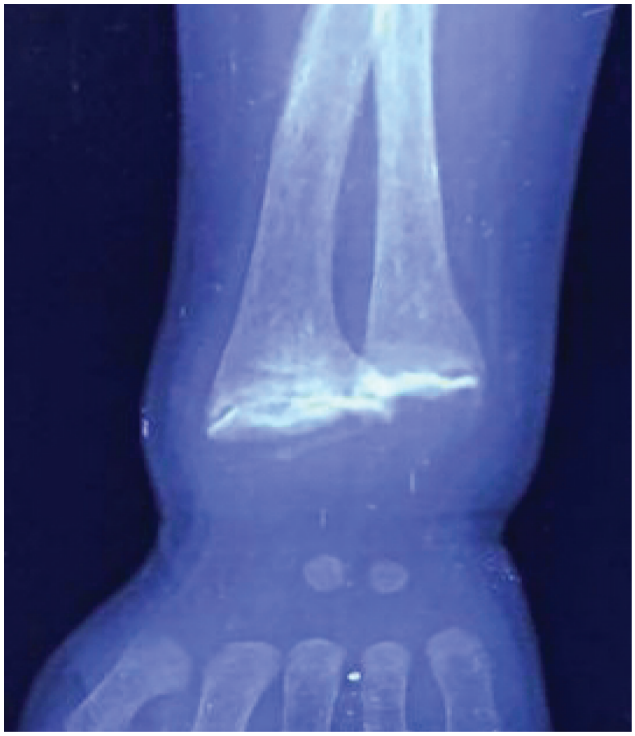

**Fig 4.** Rickets- Cupping, fraying and widening of radius and ulna, Scurvy- White line of Frankel, scurvy line

Scurvy- White line of Frankel, scurvy line, wimberger ring sign of epiphysis and tarsal bones also (just started), osteopenia(Fig-3,4)

CXR- patchy opacity in left mid zone (pneumonia)

To find out the cause of persistent pneumonia, we have excluded tuberculosis by mantoux test (MT test) and gastric lavage for acid fast bacilli (AFB) and GeneXpert and congenital heart disease by echocardiography ECHO). However her S. Antibody levels showed S.IgM-12.20 gm/l↑(range 0.40-1.43gm/l), IgA 1.21gm/l ↑(0.27-0.66 gm/l), S.IgE 117.70IU/L↑(<60.00 Iu/ml) and IgG 8.41gm/L (N).

To exclude other than nutritional rickets we have done liver function test, renal function test, s. electrolyte, arterial blood gas analysis. All the reports were within normal range.

**Diagnosis:**

Therefore, finally on the basis of clinical, biochemical and radiological parameters she was diagnosed as a case of rickets and scurvy with persistent pneumonia with failure to thrive.

**Treatment:**

After counselling she received oral vitamin D (4000IU) daily for 6 weeks with Calcium(250mg), vitamin C (1000 mg) daily , oral antibiotics and other supportive measures. When her respiratory symptoms settled down after 10 days, she was discharged with advice and requested to come for follow up after 1 month.

**Discussion:**

Rickets is an example of extreme vitamin D deficiency, with peak incidence in infancy.[7] Nutritional rickets is present more commonly in Africa, the Indian subcontinent, Asia, Latin America and the Middle East.[8]

Vitamin D is essential for absorption of calcium from the intestines. There are two well-known sources: exposure to sunlight and dietary intake, which accounts for less than 10%. Besides poor dietary intake and inadequate sunlight exposure, further increased risk is associated with dark-skinned individuals, solely breastfed infants and prematurity.[9] Most women in developing countries have vitamin D deficiency which is an important risk factor for congenital rickets .[10] Screening should be considered for children with poor growth/ development, seizure activity/tetany, and children with chronic malabsorptive states.[9]

On the other hand, scurvy, a disease of vitamin C deficiency, has been increasingly reported in recent years.[11] Musculoskeletal manifestations are present in 80% of patients with scurvy and are prominent in pediatric population.[12] Scurvy occurs in disadvantaged populations with poor intake of fresh fruit or vegetables. As mentioned earlier, co-occurrence of rickets and scurvy develops when the society has risk factors for both conditions. This baby was born at preterm with low birth weight and was on exclusive breast feeding without any multivitamin supplementation. She also had lack of exposure to sunlight and fresh fruit and vegetables.

The presentation of co-occurrence is highly variable, and co-occurrence is not associated with any pathognomonic features. Appearance of clinical features of both conditions are dependent on the order and length of first disease prior to the development of the 2nd illness.[13]

In rickets there is defective mineralization of osteoid tissue whereas scurvy produces pathological osteoid and thereby reduces osteoblastic activity but mineralization is unaffected. The majority of classic rachitic features require some normal osteoid tissue for remodeling (e.g., softening and bending of the limbs, flaring of the metaphysis) that is inhibited by the co-occurrence of scurvy. When rickets is dominant it could mask the classic scurvy feature of new bone formation due to poor mineralization, and reduce the clinical symptoms of pain and tenderness caused by

scurvy.[14] Our patient presented with poor growth and rickets seemed to be the dominant one as she had box shaped head, wide open anterior fontanelle , widening of the wrist and ankle joints and growth failure but none of the clinical features of scurvy(limb pain, gum hypertrophy ,etc) was present, but only few radiological changes of scurvy.

Like clinical presentation, bony features of either disease are not clearly visible unless one condition occurred first and had some period to develop bony changes prior to the appearance of the secondary disease.[13] In cases of co-occurrence, classic features associated with either severe rickets (e.g., bending) or severe scurvy (e.g., line of Fraenkel, scurvy line, corner signs, elevated periosteum) are often absent and 'masked' by the processes of counter action of either disease.[14]

In some cases, one of the two diseases will dominate over the other and mask the features of the non-dominant disease. Valentini et al. reported cases of dominant scurvy and where radiographs did not provide any evidence of concomitant rickets unless rickets was well developed.[15] Fouron and Chichoine presented four cases where rickets was the dominant disease. In these cases, radiological features of rickets were predominant and white dense line of Frankel and one example of a Wimberger's ring were present in scurvy cases. However, advanced features of neither disease were present, for example bending of rickets and periosteum elevation of scurvy. [14] Similarly, in this case fraying and widening of metaphysis of long bones, white line of frankel, scurvy line, wimbergers ring were present without advanced features of either disease as we know scurvy and rickets are always counteracting. Cases of scurvy and rickets co-occurrence have been mentioned in a number of recent articles.[16,6]

The diagnosis of any forms of vitamin D deficiency rickets is usually established by clinical, biochemical, and radiographic criteria.[17] In our case in addition to clinical and radiological criteria biochemical parameters such as high alkaline phosphatase and parathormone level with normal calcium and phosphate levels were also suggestive of nutritional rickets. A level of 25 hydroxy vitamin D (25-OH D) less than 12.5 nmol/L (5 ng/mL) is suggested for the diagnosis of rickets.[9] However, we could not perform vitamin D level for financial constraint of the parents.

Though serum Vitamin C concentration in the plasma is specific for the diagnosis of scurvy; vitamin C <11micro-mole/l suggests scurvy, actually it is not necessary to confirm the diagnosis as a meticulous dietary history, physical examination and radiological findings are enough to reach a correct diagnosis easily.[18] In case of co-occurrence, radiographs appear to be the most helpful. Radiographic methods can produce a high percentage of accuracy in identifying co-occurrence of rickets and scurvy.[19]

There are two strategies for administration of vitamin D. In stoss therapy, vitamin D(300,000-600,000IU) is administered orally or intramuscularly as 2-4 doses over one day. The alternative strategy is daily vitamin D with a minimum dose of 2000IU/ day for a minimum of 3 months. Either strategy should be followed by daily vitamin D intake of 400IU/day if < 1 year, or 600IU/day if >1 year along with adequate dietary calcium and phosphorus intake. Vitamin D3 is preferable to D2 because of longer half life. Hypocalcemia should also be treated with parenteral administration of calcium gluconate in case of manifest tetany or convulsions followed by oral calcium supplements.[7] In case of scurvy, supplementation with 100-200mg/day oral or parental vitamin C is the usual treatment. Clinical improvement is seen within 1 week in most cases, however treatment should be continued for up to 3 months.[20]

For adequate exposure to sunlight, a fully clothed child would have to spend two hours outside weekly and darker skinned individuals may require exposures up to 6–10 times this amount. Breast milk is the ideal nutrition for infant, however, it only contains 15 – 50 IU/L of vitamin D. So, American Academy of Pediatrics (AAP) has recommended vitamin D supplementation in all breastfed infants from the first day of life and continue through childhood/adolescence.[9]

**Conclusion:**

In the existing literature of Bangladesh, co-occurrence of scurvy and rickets has not been found being reported. Nutritional assessment is very crucial for every child. When children present with growth failure and developmental delay, rickets should be evaluated and coexistence of other nutrient deficiencies such as scurvy must be ruled out. Several risk factors are associated with co-occurrence. Education on proper nutrition during pregnancy, lactation, infancy and supplementation of vitamin D during pregnancy, lactation period, in preterm &low birth weight babies, in all exclusive breastfeeding babies and adequate exposure to sunlight necessary to prevent its growing resurgence. Proper childhood maintenance visits with growth and development screenings are critical for early detection of these easily treatable condition.

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## Short Communication

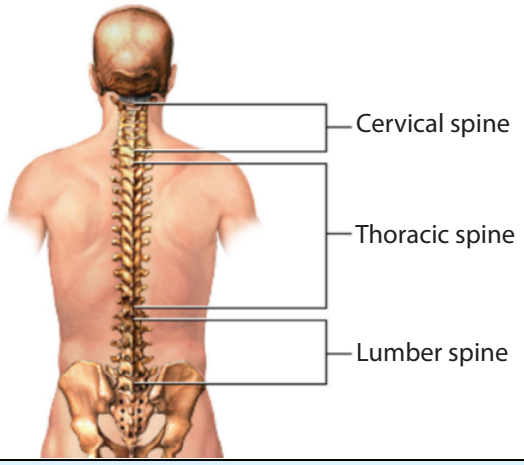
# Spine Surgery- Scopes, Expectation and Drives in Bangladesh: On How to Alleviate Patients' Sufferings

Shahidul Islam[1], Merina Tanzil[2], Najiba Kabir[3], Chowdhury Md. Saleh Al-Nayem[4], Sabikun Nahar Tonima[5]

Globally, 'spine surgery' remains a sophisticated procedure, thus, a bit difficult and "complex" as some spine/neuro-surgeons referred.[1]

This surgery requires more concentration and advanced long curve training. Inappropriate training may lead to unexpected PO and future complications. Though this rate is merely high, but it may cause longstanding disability. Nevertheless, this very message of 'complications' spreads too faster than fairies that often generates superfluous fear and/or needles anxiety among the patients who need surgery.

**Posterior Spinal Anatomy**




1. Prof. & Head, Dept. of Orthopaedics and Spine Surgery, AWMCH
2. Research Officer, Medical Research Unit, AWMCH
3. Intern, AWMCH
4. Medical Officer, Dept. of Orthopaedics and Spine Surgery, AWMCH
5. Honorary Medical Officer, Dept. of Orthopaedics and Spine Surgery, AWMCH

**Correspondence:** Prof. Shahidul Islam, Professor & Head, Department of Orthopaedics and Spine Surgery, AWMCH, Dhaka. E-mail: drshahid34rmc@gmail.com

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There are so many expectations among patients. Multiple demographic, psychological and clinical characteristics are associated with expectations, where disability due to pain is the most consistently associated variable.[2]

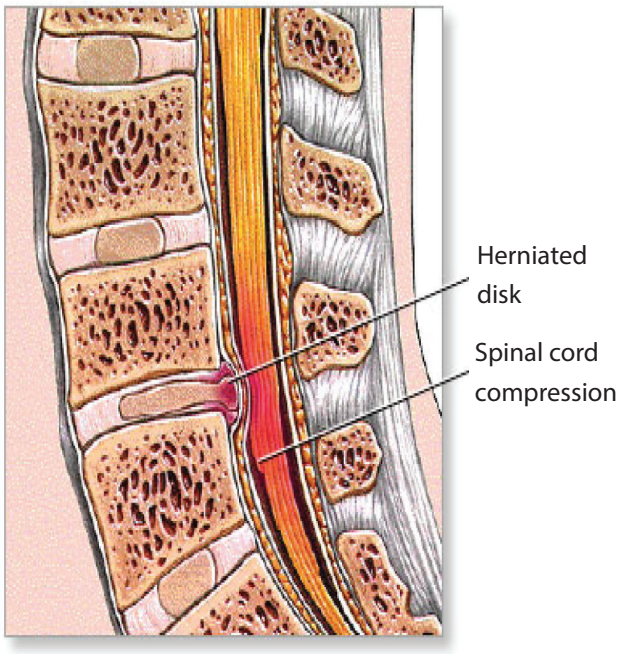
Managing patients' expectations is crucial while providing treatment to the patients undergoing spinal surgery. Patients' satisfaction is associated with improved clinical outcomes and can be upgraded when patients' and surgeons' expectation are aligned and patient preferences are met.

As Spine surgery is largely elective, patients often seek treatment to improve quality of life and relieve subjective symptoms. Understanding patients' expectation is critical to ensure that patients and physicians are working towards similar goals.[3]

In the perspective of Bangladesh, patients say, "Is there any treatment without surgery? We are afraid of surgery because after surgery neurological conditions maybe deteriorated or patient maybe permanently disabled or paralyzed or can't give guaranty whether I'll be fully cured by surgery or not.".

**Indications of surgery in spine disorders are**

- Disc Prolapsed in Lumber, Cervical or dorsal regions which is not cured by conservative procedures.
- Cauda Equina syndrome due to huge disc prolapse.
- Degenerative disc disease with unstable spine.
- Spinal Canal stenosis or spinal cord compression
- Spinal space occupying lesions or spinal tumor usually primary or single level secondary tumor.




- Spinal infections-pyogenic abscess or tubercular infection which causes neurological deficit and does not improve by conservative treatment.
- Spinal injury- unstable or stable.

**Scope of spinal surgery in Bangladesh**

- Institutes or Hospitals where spine surgeries are done mostly present in Capital City in Government level such as **BSM Medical University**, **Dhaka Medical College Hospital** and **Sir Salimullah Medical College Hospital**, **NITOR** (National Institute of Traumatology Orthopedics Rehabilitation), **NINS** (National Institute of Neuro Science) and some well-known private hospitals.
- Outside Dhaka some medical colleges have facilities for spine surgeries and some private hospitals have limited scope also but there is no scope for spine surgeries in District Sadar Hospital. Surgeons who perform better surgeries are also limited. Spine surgeons come from both the Orthopedic & Neurosurgery disciplines. All orthopedic surgeons are not spine surgeons, because many senior orthopedic surgeons were not used to do spine surgeries rather than trauma and routine orthopedic operations. Young orthopedic generation are very much interested about spine surgery. In Bangladesh, usually most of the neurosurgeon performs spine surgeries but expertization is always demanding and limited. So good spine surgeons are less in number. It is not true for not only Bangladesh, but also for other countries in the world. Spine surgeries are costly in all over the world as well as in Bangladesh, especially in private hospitals or private institutes. In Government medical college surgeries are done almost free of cost but scope for bed and surgeons are very limited than its demand. Very limited number of patients get the scope for admission. One can get the chance for admission after visiting outdoor department for several visit. Duration for waiting is almost one month and after admission patient get serial for operation after another month. So, in Government hospital patient can get scope for surgery one and average after two months.

Patient from all over Bangladesh moves towards Dhaka as facilities for surgery are limited only in some large medical colleges.

**Sufferings of Patients**

- They have to travel long distance (Periphery to Capital City)
- They have to maintain long que for admission.
- Long waiting time for operation.
- More money in good private hospital and less hospitality and more complications like infection in Government hospital.

**Hope**

There are hope for future improve facilities. Government institutes and hospital are trying for creating more accommodation as well as private sectors also growing. Our societies for spine surgeons like Bangladesh Spine society and Neuro Spine Society both are trying to improve the skill of surgeons by arranging training program in home and abroad. So, we are waiting for a golden future.

Pictorial evidences of most important Govt. ran hospitals/Institutes where spine surgeries are performed regularly in Dhaka- the Capital of Bangladesh:

- **BSM Medical University**, Dhaka and
- **Sir Salimullah Medical College**,
- **NITOR** (National Institute of Traumatology Orthopedics Rehabilitation),

- **NINS** (National Institute of Neuro Science) and some well-known private hospitals.

And, some of the private &/or corporate level specialized hospitals do also perform spine surgery:

1. Ad-din Women's Medical College Hospital, Moghbazar, Dhaka.
2. Ibn Sina Hospital, Dhanmondi, Dhaka.
3. Islami Bank Central Hospital, Kakrail, Dhaka.
4. Northern International Medical College and Hospital, Dhanmondi, Dhaka.

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Short Communication

# DCR Operation in Rural Area of Bangladesh: A Study of 346 Cases

Md Abdul Matin[1], Mahbubur Rahman Sahin[2], Tasnia Nawreen[3]

**Abstract:**

**Objective:** To observe the result of surgical outcome of DCR operation without intubation.

A total number of 346 DCR surgeries were performed in septic operation theatre of Pangsha Eye Hospital, south western rural area in Bangladesh from January 2004 to December 2014. 346 cases were included in this study.

On the basis of distribution of age, 43.41% of cases were between 14-40 years of age.

Female cases were 83% and male were 17.63%. Only 10 cases were intubated and 336 were not intubated.

**Key words:** Dacryocystorhinostomy (DCR), Intubation (silicone), Nasolacrimal duct obstruction, Lacrimal drainage, Lacrimal passage.

## Introduction:

Three hundred and forty-six operations of dacryocystorhinostomy were performed in the period of 11 years from January 2004 to December 2014 by the author in Pangsha Eye Hospital, Pangsha, Rajbari, a south-western region in Bangladesh.

Total operations were done in a septic operation theatre of this hospital. Dacryocystitis is an acute or chronic inflammation of the lacrimal sac. The surgical procedure of choice is dacryocystorhinostomy in which a connection is established between the lacrimal sac and nose.

Epiphora a condition in which there is tearing because of impaired drainage of tears through the lacrimal passage. Chronic dacryocystitis is the commonest cause of epiphora and also is responsible for cosmetic disfiguration. Obstruction in the lacrimal drainage system and its inflammation is annoying and sometimes is a vision threatening problem.[1]

External dacryocystorhinostomy (DCR) was first described by Toti in 1904, since then it has gone through several modifications.[2]

Acquired lacrimal drainage obstruction may be secondary to trauma, infection, inflammation, rarely neoplasm or mechanical causes.

1. Associate Professor, Ad-din Women's Medical College, Dhaka.
2. Assistant Professor, President Abdul Hamid Medical College, Kisorgon.
3. Medical Officer, Dhaka Medical College.

**Correspondence:** Dr. Md Abdul Matin, Associate Professor, Ad-din Women's Medical College, Dhaka. E-mail: mdabdulmatin096@gmail.com
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Dacryocystitis is a disease of adult group with female preponderance. Prevalence of dacryocystitis is high in those who use bath water like pond water.[3]

## Materials and methods:

The operations were carried out in a septic OT in Pangsha Eye Hospital from January 2004 to December 2014. The operations (346 cases) were done without intubation (only 10 cases were intubated), the age of those patients was from 14 to 70 years. There were 285(83%) females and 61(18%) males. All patients were properly evaluated in the indoor including necessary laboratory investigations for smooth post-operative management and to avoid any short of complication due other systemic diseases.

Type of study:
Retrospective chart analytic study.
Cohort analytic study

Preoperative medication- Inj. Diclofenac 1amp (50mg) and Inj. Diazepam 1amp (10mg) were given 10-15 minutes prior to beginning of the operation for relief of the pain and tension during operation.

## Anesthesia:

Infiltration anesthesia using Xylocaine 2% 5ml and Bupivacaine (.5%) 5ml with 0.15 to 0.2ml of adrenaline 1:1000making its solution to 1:150000/1:200000.

## Surgical procedure:

All the steps of the conventional DCR were done without intubation and only 10 cases were intubated.

**Table-1:** Yearly operations registered:

| Year | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 | 2014 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Female | 11 | 20 | 20 | 17 | 34 | 28 | 33 | 37 | 36 | 29 | 20 |
| Male | 02 | 10 | 09 | 04 | 05 | 13 | 03 | 05 | 03 | 04 | 03 |
| Total | 13 | 30 | 29 | 21 | 41 | 41 | 36 | 42 | 39 | 33 | 23 |

Table I shows the highest operation was performed during 2011 and the total number was 42 out of which 37 were female and 5 were male. During 2008-2009 total 41 DCR operations was done. But the lowest number of operations were 13 in 2004.

**Table-II:** Age of the patients:

| Age in years | No of patients | Percentage |
|---|---|---|
| 14-20 | 06 | 1.79% |
| 21-30 | 63 | 18.21% |
| 31-40 | 81 | 23.41% |
| 41-50 | 89 | 25.72% |
| 51-60 | 64 | 18.50% |
| 61-70 | 43 | 12.43% |
| Total | 346 | 100% |

In table II the highest age group was from 41-50 years which was 25.72%, followed by the age group of 31-40 years (23.41%). The minimum number of people belonged to age group 14-20 years, in percentage it was 1.79%.

**Table-III:** Distribution of sex

| Sex | No of patients | Percentage |
|---|---|---|
| Female | 285 | 82.37% |
| Male | 61 | 17.63% |
| Total | 346 | 100% |

The Table III shows out of total 346 patients, the majority were female (82.37%) and rest were male (17.63%)

**Table-IV:** Side of the eye

| Side | No of patients | Percentage |
|---|---|---|
| R/E | 148 | 42.77% |
| L/E | 198 | 57.23% |
| Total | 346 | 100% |

Table IV presents that 148 operations were done in the right eye 42.77%) and 198 were done in the left eye (57.23).

**Table-V:** With or without intubation

| Intubation | No of patients | Percentage |
|---|---|---|
| Without | 336 | 97.12% |
| With intubation | 10 | 2.89% |
| Total | 346 | 100% |

Table V shows intubation was needed in 336 patients and without intubation 10 operations were done.

**Result:**

Almost all cases were followed up postoperatively for 3 to 6 months. In case of intubations, tube was removed after 2months

**Discussion:**

This study has shown that 41.62% cases belong to 21-40 years age group. Age between 41-50 years group were 25.27% and age 51 to 70 years 30.93%. In one study by Choudhury et al (1994) 58% cases belong to 20-40 years age.[4] Gender wise, about 83% were female and 17% were male. In one study, women is consistent with a study of Kabir et al 80% were females and 20% were male.[5] In another study, 64.11% were female and 35.89% were male[6], another study found 68.85% were female and 31.45% were male out of retrospective study on 459 cases.[7] In another study female were 69.6% and 30.4%.[8] However, most of the female patients in our study came from villages and majority were house wife, using pond water for bathing.

The only successful treatment of chronic dacryocystitis is dacryocystorhinostomy (DCR).

Preferably with single flap rather than double flaps. Untreated dacryocystitis never undergoes spontaneous resolution.[9]

Regarding intubation, we performed 10 cases with intubation and 336 were without intubation. In intubated cases silicon tube remained for 2 months. After 2 months tube was removed.

Syringing was done on 2nd post operative day. In those cases which were intubated 2 of them were failed and re-DCR was done but failed due to soft tissue growth and for that obliterated the osteum and other case failed due to abnormal anatomical structure. In cases without intubation, 5 of them came with partial block after 2 to 4 months and 4 of them were relieved by probing and syringing.

Dacryocystorhinostomy (DCR) is an acceptable method in treatment of epiphora due to acquired naso-lacrimal duct obstruction and success rate of the procedure is also very good.

**Conclusion:**

The female who undergone DCR operation suffered from common cold for long period. They had also upper respiratory tract infection in most of the cases. Hormone factor may contribute for more affection of female.

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Ad-din
Women's Medical College
2 Bara Moghbazar, Dhaka-1217, Phone: +880-2-9362921, 9362926
Fax: 8317307, E-mail: awmc@ad-din.org, Website: www.ad-din.org