IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 3 Ser. 11 (March. 2019), PP 14-26
www.iosrjournals.org

A study of Primary cesarean section in Multiparous women

Dr. Leena Gattam[1]; Dr. P.Hima Bindu[2];
[1]*Dr. Leena Ghattam, Junior Resident, General Medicine, Siddhartha Medical College / Govt.General Hospital, Vijayawada, Krishna Dist, Andhra Pradesh, India.*
[2]*Dr. P.Hima Bindu, Professor, Gynaec & Obst., Siddhartha Medical College / Govt.General Hospital, Vijayawada, Krishna Dist, Andhra Pradesh, India*
Corresponding Author: Dr. Leena Ghattam

Abstract: Aim: *To study the incidence, indications for primary caesarean section in multiparous women and analysis of various related factors, To study maternal and fetal outcome after primary caesarean section in multiparous women and To investigate the association of high hsCRP (> 3 mg/L) levels with ischemic stroke and its subtypes in Indian patients.*
Place and duration of study: *Gynaec & Obst. Department, Siddhartha Medical College / Government General Hospital, Vijayawada,Krishna District, Andhra Pradesh from Jan'2017 to June'2018.*
Methodology: *150 patients of primary cesarean sections in multipara done in Govt.General Hospital, Vijayawada attached to Siddhartha Medical College, Vijayawada were studied and analysed. This study includes the multiparous women who had delivered vaginally in previous pregnancies and are undergoing cesarean section for the first time.*
Results: *Majority (67.33%) of patients were from the age group 21-25yrs. 79.33% patients were booked cases and 20.6% were unbooked. Anemia (57%), antepartum hemorrhage (24%), malpresentations and severe pre-eclampsia (20%) were most frequently encountered antenatal complications in multiparous women. Antepartum hemorrhage (24%) and fetal distress (24%) were the common indications for cesarean section in multiparous women. There were no cases of maternal mortality in our study. Paralytic ileus and puerperal sepsis were more Common post operative morbidity and seen in 3 cases each. 32.66% babies were admitted in NICU. Most common indications for NICU admissions were meconium aspiration syndrome and prematurity. Perinatal mortality in the study was 15.6% and among them Antepartum hemorrhage has the highest perinatal mortality rate of 56.25%.*
Conclusion: *The most common indications for cesarean sections in multipara are antepartum haemorrhage, fetal distress and malpresentations. Cephalopelvic disproportion in multipara can be more significant and dangerous than in primipara because delay in recognition leads to obstructed labour and second stage cesarean sections which carry more maternal and fetal morbidity. Good antenatal and intrapartum care and early referral will reduce the maternal and perinatal morbidity and mortality in multipara. Multipara in labour should be given the same attention as primigravida.*
Keywords: *Cesarean section, Multipara, Primary.*

Date of Submission: 28-02-2019 Date of acceptance:18-03-2019

I. Introduction

The term Cesarean Section (CS) refers to the operation of delivering the baby through incision made on the abdominal wall and on intact uterus after the period of viability. It has an enormous potential for the preservation of life and health, probably greater than that for any other surgical operations. The evolution of cesarean delivery as a safe procedure with extraordinary low maternal and fetal mortality rates is one of the most important developments in modern obstetrics and perinatal medicine. By the early decades of the 20th Century, several important innovations in surgical care has occurred including aseptic technique, reliable anesthesia and the control of hemorrhage by proper suturing of tissue planes and ligation of severed blood vessels. The introduction of the lower segmental incision allowing exclusion of the uterine wound from the peritoneal cavity dramatically decreased the risk of postoperative peritonitis as a complication of puerperal endometritis. The addition of blood transfusion and antibiotic therapy further reduced the mortality and morbidity of cesarean section. This decrease in maternal mortality of cesarean section made the operation a reasonable alternative for delivery of the fetus at increased risk for asphyxia or trauma from labour and vaginal delivery.

Worldwide the rise in cesarean section rate during the last three decades has been alarmingly high and needs an in depth study. Cesarean section is one of the most commonly performed major surgical procedures. In

America By 2004, the overall cesarean rate had risen to 29.1% and the primary cesarean rate to 20.6%, both representing the highest national rates ever reported[1]. According to a study by Indiancouncil of medical research (ICMR), the incidence of cesarean sections is 25.4%for the years 1998-1999[2]. Evidence from research studies shows there is a growing tendency for cesarean deliveries especially during complications confronted at the time of pregnancy and delivery. Increasing maternal age which varies by parity is associated with significantly elevated risks for pregnancy complications and adverse outcomes including increased risks for cesarean section.

As per the latest data (National Family Health Survey 2015-16 (NFHS-4), the cesarean rates at population level in India seem to be 17.2 % and in Andhra Pradesh seems to be 40.1%. The same document goes on to look at Cesarean rates in the private and public sector and whilst the discrepancy in the rates in these two sectors has been commented upon, there is no mention in the commentaries of the fact that the private sector delivers more babies than the public sector in the urban areas and absolutely no indication of morbidity rates either maternal or neonatal in either sector. There is also no acknowledgement of the fact that the lower rates in public sector could simply be a reflection of the paucity of capacity, both infrastructure and human resource.

To reiterate and quote from the WHO working group on cesarean section – *"The time has come to put the debate about the preferable rate of CS on hold.* Let's start to collect data uniformly so that in the near future we will be able to move our focus from CS rates at population level to monitoring and discussing CS rates and outcomes in each group of the Robson classification. Only then will we have the data and evidence that will lead us more clearly to actions to improve care". *(Betran AP, Torloni MR, et al for the WHO Working Group on Caesarean Section. WHO Statement on Caesarean Section Rates. BJOG 2016;123:667–670)*

FOGSI recommends the setting up of a cloud based registry linked to its website which will collect anonymous data at hospital level using the WHO recommended Robson's ten group classification system as the first step in determining the range of cesarean rates.

We would like to emphasise that the hallmark of labor management in the 21st century should be individualized care for the laboring woman with the expectation of a successful and safe vaginal delivery, together with the ability to intervene with a cesarean delivery, if needed, to prevent morbidity and mortality. *(Adapted from Caughey A B BIRTH 41:3 September 2014)*

The indications for performing cesarean section have changed a lot in recent years and keep changing in, for varied circumstances. Several non clinical factors have substantial effect on the rates of cesarean section. Women of higher socio economic status have higher incidence of cesarean section than do women of lower socio economic status. Trends of higher cesarean section rates are found in teaching hospitals and paying hospitals. Age and parity of the women influence the cesarean section rates being more in young and elderly primigravida and grand multipara. The other areas of dispute include the place of cesarean section in breech delivery, fetal distress and placental abruption. Assisted reproductive technology is more widely used than in the past and is associated with greater caesarean delivery rates. Obesity, which is a caesarean delivery risk, has reached epidemic proportions. Primary cesarean deliveries are an important target for reduction, because they lead to an increased risk for a repeat cesarean delivery. Of particular interest are the cesarean deliveries that are elective, although the clinical use and implications of the term elective requires clarification. Elective cesarean deliveries can include medically and obstetrically indicated procedures that generally occur before labour. Elective cesarean deliveries can also include procedures for which there is no clear medical or obstetric indication. There is a growing concern that there is a rising rate of the latter Maternal choiceelective primary cesarean deliveries generate both clinical and ethical controversy and concern[3].

II. Aims And Objectives

- To study the incidence, indications for primary caesarean section in multiparous women and analysis of various related factors
- To study maternal and fetal outcome after primary caesarean section in multiparous women

III. Materials And Methods

Majority (67.33%) of patients were from the age group 21-25yrs. 79.33% patients were booked cases and 20.6% were unbooked. Anemia (57%), antepartum hemorrhage (24%), malpresentations and severe pre-eclampsia (20%) were most frequently encountered antenatal complications in multiparous women. Antepartum hemorrhage (24%) and fetal distress (24%) were the common indications for cesarean section in multiparous women. There were no cases of maternal mortality in our study. Paralytic ileus and puerperal sepsis were more

Common post operative morbidity and seen in 3 cases each. 32.66% babies were admitted in NICU. Most common indications for NICU admissions were meconium aspiration syndrome and prematurity. Perinatal mortality in the study was 15.6% and among them Antepartum hemorrhage has the highest perinatal mortality rate of 56.25%.

3.1Inclusion criteria

- This includes the multiparous women who underwent cesarean section for the first time who have delivered vaginally in previous pregnancies.

3.2 Exclusion criteria

The study does not include women who had

- Delivery of less than 28 weeks.
- Underwent cesarean section in previous pregnancy, previous uterine surgery or hysterotomy.
- Secondary abdominal pregnancy

Information regarding age, socioeconomic status, details about previous conception, antenatal care and booking status was collected. Complete general physical examination, systemic examination and obstetric examination was done.

Routine and relevant investigations such as analysis of urine (albumin, sugar, Microscopy), HB gms/dl, Blood Grouping and Rh typing, VDRL, HIV, HBsAg, RBS were all done. Ultra sound with fetal Doppler study was done whenever found necessary. Cardio Tocographic monitoring was done during labour to assess fetalwell being. Period of gestation was derived from history of LMP and clinical examination and confirmed by ultrasound. Engagement of head during labour, duration of labour, indication for cesarean delivery, colour of liquor, abnormality of III stage, puerperium; weight of baby, maturity, APGAR and congenital malformation are recorded. Maternal complications like post partumhemorrhage, anemia, toxemia, hydraminos, antepartum hemorrhage, intra-uterine growth retardation and neonatal morbidity like prematurity, meconium aspiration syndrome and birth asphyxia were noted.

IV. Observation And Results

The 150 patients admitted in our hospital selected for study.

Table-1 : Agewise distribution

Age Group	No.of Patients	Percentage
16-20	14	9.33%
21-25	101	67.33%
26-30	29	19.33%
31-35	6	4.0%
Total	150	100.00

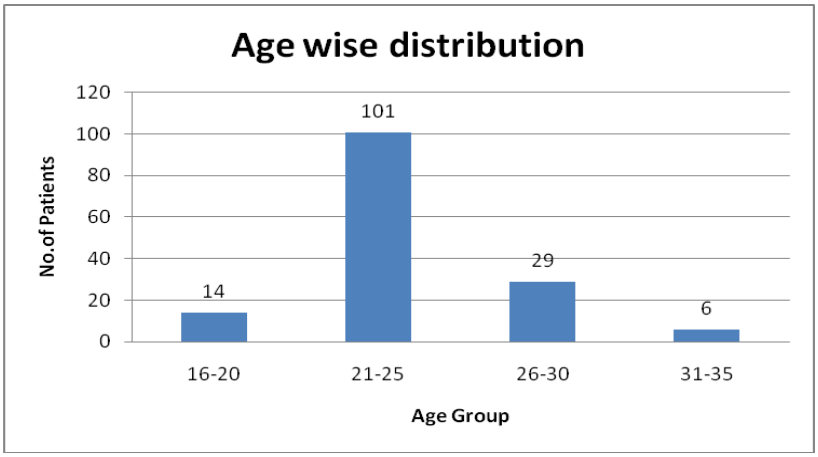


Table-2 : Gravida wise distribution

Gravida	No.of Patients	Percentage
G1	0	0.00%
G2	93	62.00%
G3	43	28.66
G4	10	6.66
G5	4	2.66
Total	150	100%

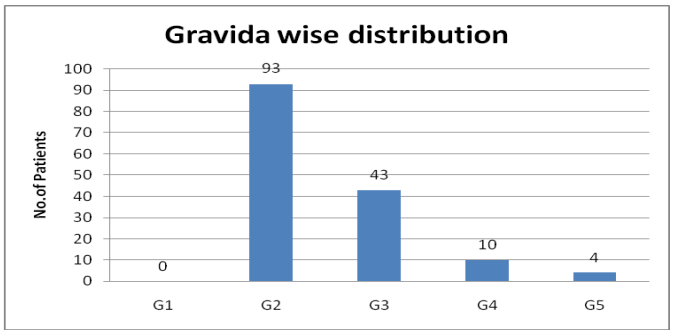


Table-3 : Parity

Parity	No.of Patients	Percentage
P1	119	79.33%
P2	25	16.66%
P3	6	4.0%
Total	150	100%

Table-4 : Antenatal Care

Antenatal Care	No.of Cases	Percentage
Booked	119	79.33%
Unbooked	31	20.6%
Total	150	100%

Table 5: Indications of primary cesarean section in multiparous women

Indications	**Number of patients**	**Percentage**
Breech	18	12
Cephalic	119	79.33
Footling	1	0.66
Oblique lie	2	1.33
Transverse Lie	5	3.33
Twin Cephalic	3	2%
Twin Footling	1	0.66
Twins Oblique	1	0.66

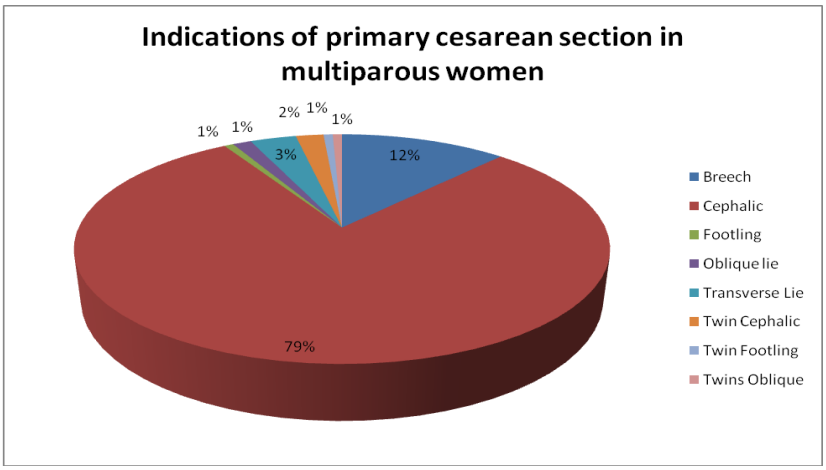


Table- 6 : Duration of Labour before cesarean

Duration (Hrs)	No.of Patients	Percentage
<5	63	42%
6-10	53	35.3%
11-15	2	1.3%
>21	1	0.6%
Not in Labour	31	20.66%
Total	150	100%

Table -7 : Post.op. Complications

Post operative complications	Number of patients
Paralytic ileus	10
Puerperal fever	12
Uneventful	122
Urinary tract infection	6
Total	150

Table – 8: Fetal Outcome

Birth weight in kgs	No of Babies
<1.5	1
1.6-2.0	8
2.1-2.5	23
2.6-3.0	42
3.1-3.5	41
3.6-4.0	17
>4	13
Total	**145**

Table- 9 : Neonatal Outcome

Neonatal outcome	**Number**
Live births	149
Term	138
Preterm	11
<34 weeks	2
>34weeks	9
Stillbirths	1

Table 10 : Causes for stillbirth in the study.

Cause	Number
Placenta previa	1

Table 11: Neonatal morbidity

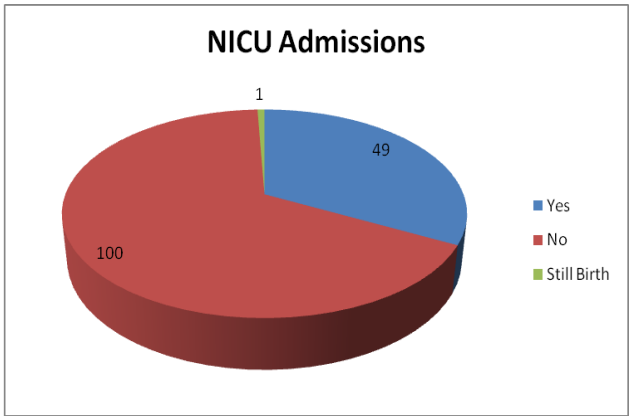
NICU admissions	**Number of babies**	**Percentage**
Preterm care	10	29.41
Meconium aspiration syndrome	11	32.35
Birth asphyxia	3	8.8
Neonatal Jaundice	1	2.94
Sepsis	9	26.47
Total	34	100

Table 12 : Perinatal mortality

Cause	Number
Placenta previa	1

Table-13: NICU Admisions

NICU admissions	**Number of babies**	**Percentage**
Yes	49	32.66%
No	100	68.66%
Still Birth	1	0.66%
Total	150	100.00%



V. Discussion

Cesarean section is not the panacea for all obstetric problems but it is an excellent solution when applied judiciously. 150 cases of primary cesarean sections in multipara done in Siddhartha Medical College / Govt.General Hospital, Vijayawada from October'2016 to September'2018 were analysed.

5.1 Incidence

The frequency of primay cesarean section in multiparous women in Government General Hospital, Vijayawada is 5.7% of total primary cesarean sections and 1.25% of the total number of deliveries during the study period.

Incidence of primary cesarean section in multipara in the present study is 1.25% which is comparable with Jacob and Bharghav study (2.06%) [17].

Not surprisingly the WHO issued a new statement in 2015 with the headline "Every effort should be made to provide caesarean sections to women in need, rather than striving to achieve a specific rate" World Health Organization. WHO Statement on Caesarean Section Rates. Geneva: World Health Organization; 2015 (WHO/ RHR/15.02).

5.2 Indications of Cesarean Section:

The four major indications for cesarean section in multipara in our study were fetal distress, malpresentations, PROM, antepartum hemorrhage, and fetopelvic disproportions. Each of these indications will be discussed individually.

5.3 Foetal Distress

Foetal distress as an indication for lower segment cesarean section in multipara is low owing to the complacent attitude of the patient; and the obstetrician. Incidence of fetal distress as quoted by various authors is given below

AUTHOR	PERCENTAGE
Jacob et al (1972)	8.6
Klein et al (1963)	7.5
Praagh et al (1968)	7.6
O'Sullivan (1963)	10.3
Sikdhar et al (1980)	18.8
Vashista et al (1972)	7.4
Present study	24

The incidence of fetal distress in the present study is slightly more as compared to other studies, this can be attributed to frequent use of cardiotocogram in these recent years as compared to the previous decades. The frequency of fetal distress in the present study is comparable to Sikdhar and Mithra study.

5.4Cesarean section for cephalopelvic disproportion: [16-18, 23, 36]

Author	Percentage
Duckman et al (1968)	22
Klien et al (1963)	14.5
Tancer et al (1959)	17
Jacob et al (1972)	26
Vashista et al (1972)	22
Present study	06

In a study by Duckman et al 22 multipara had primary cesarean section for cephalopelvic disproportion (4.1% of primary cesarean section) and contracted pelvis was found in 11 cases[16]. Deflexion of the head and the size and configuration of vertex have contributed to the relative disproportion in these patients. All patients were in active labour at the time of cesarean section and Oxytocin stimulation was used in thirteen cases. Although contraindications to the use of oxytocin have become greatly diminished in the past decade, its use in the multipara with apparent cephalopelvic disproportion is still hazardous. If used to overcome a dysfunctional labour with a possible and not definite disproportion, a careful scrutiny with a definite cut off time of the oxytocin should be practiced.

In the study by Klein et al, incidence of primary cesarean section in multipara was 14.5% (27 cases). All infants delivered by cesarean section in this series were larger than the largest infant previously delivered through the vagina[18].Klien states that high incidence of forceps deliveries and high mortality as noted in previous obstetric history were significant pointers to suspected possible disproportion between the fetus and maternal pelvis.

From the above studies it is evident that disproportion does occur in multipara, though osteomalacia as an etiological factor may not be encountered in the present day. It is to be stressed that there is a tendency to allow even closely observed patients to go too long in a nonproductive type of labour just because they are multipara.

Klein states that multipara in early labour with foetal head not engaged should receive the same careful investigation for cephalo-pelvic disproportion that a primigravida would receive. The fact that the multipara has had one or more vaginal deliveries should be regarded as an optimistic fact but not diagnostic criteria for spontaneous delivery of the fetus. Reluctance to diagnose this cephalopelvic disproportion leads to a longer labour, with development of excessive moulding and caput formation which makes the observer to believe that progress has been made. Many times, delivery with forceps is attempted and fails[18].

Duckman et al states that cephalopelvic disproportion in a multipara can be more significant and more dangerous than in primi because of the delay in recognition. Earliest recognition of its existence is made possible by more frequent discussion of the problem. Hence the philosophy towards CPD be reevaluated with a more liberal and earlier use of cesarean section. Cesarean section rate may increase slightly but healthier infants and mothers will more than offset the slight change in statistics[16].

5.5 Malpresentations And Malpositions

Malpresentations are more common in a grand multi and are favoured by a pendulous abdomen and lordosis of the lumbar spine. Transverse lie is the most common malpresentation encountered. According to Eastman[6], the causes of transverse lie are:

a) Abnormal relaxation of the abdominal wall
b) Pelvic contraction
c) Placenta previa

Frequency of malpresentations in multipara:

Author	Percentage
Klien et al (1963)	10.2
Sen (1967)	11.7
Jacob et al (1972)	24
Present study	15

The incidence of transverse lie increases with parity occurring 10 times more frequently in patients of parity four or more than in a primigravida. Relaxation of the abdominal wall with a pendulous abdomen allows the uterus to fall forwards deflecting the long axis of the birth canal into an oblique or transverse position. "Pelvic contraction" and placenta previa act similarly by preventing engagement.

The other malpresentations encountered in multipara are breech, compound presentation, brow and face. Some of the neglected cases of transverse lie present as hand prolapse. The malposition commonly encountered in multiparas is occipitoposterior position.

5.6 Uterine Dysfunction

The commonly encountered dysfunction are uterine inertia or inco-ordinate uterine action. Uterine inertia is especially a feature when associated with multiple pregnancy or hydramnios can be treated by judicious use of oxytocin in multipara with intensive monitoring.

Reported incidence of uterine dysfunction:

AUTHOR	**PERCENTAGE**
Jacob et al (1972)	4.0
Kasturilal (1972)	9.8
O'Sulivan (1963)	2.6
Praagh et al (1968)	**3.08**
Sen (1967)	1.1
Present study	2.0

In the present series 2 patients were induced with 25µg of misoprostol vaginally every 6th hourly but even after 48 hours of induction there was no progress of labour and were subjected to cesarean section.

5.7 Bad Obstetric History

Includes previous history of stillbirths or neonatal deaths or consecutive abortions. Most of them undergo elective lower segment cesarean section.

Incidence of BOH

AUTHOR	**PERCENTAGE**
Jacob et al (1972)	5.3
Klein et al (1963)	2.2
Praagh et al (1968)	2.4
O'Sullivan (1963)	1.3
Sen (1967)	6.38
Vashista et al (1972)	12.96
Present study	6.0

The incidence of BOH in the present study is 6.0% which is similar to the incidence in Jacob, Bharghav and also Sameer Sen's series.

5.8 Antenatal care:

Author	Total no of cases	Booked cases	Unbooked cases
Vashista et al (1972)	54	14 (25.93%)	40 (74.07%)
Present study	100	33 (33%)	67 (67%)

The percentage of booked and unbooked cases in the present study is comparable with vashista et al study. Most of the cases were unbooked in both the studies

Maternal Morbidity and Mortality in Multipara

Author	Maternal mortality (%)
Klein et al (1963)	0.5
Sen (1967)	2.12
Jacob et al (1972)	6
Present study	nil

Cesarean section is major operative procedure. There is potential for injuries to ureter, bladder, bowel, blood vessels and lacerations of cervix, vagina and broad ligaments. It also increases the risk of post partum hemorrhage, pulmonary embolism, paralytic ileus, urinary tract infections and other infections. In our study with good intra operative and post operative care there was no maternal mortality.

Yoles and Maschiach (1998) Reviewed all deliveries in Israel between 1984-1992 Maternal mortality rate following cesarean section in multipara is shown below:

Delivery Period	MMR / 1,00,000 Births
Vaginal	3.6
Cesarean Total	21.8
Emergency	30
Elective	2.8

In this series the causes of maternal mortality are renal failure due to mismatched blood, placenta previa, septicemia, obstructed labour, threatened rupture[31].

5.9 Post operative maternal morbidity

AUTHOR	PERCENTAGE
Jacob et al (1972)	18.6
Praagh et al (1968)	10.4
Sen (1967)	20.2
Present study	10

The causes of maternal morbidity were fever, urinary tract infection, lung complications, paralytic ileus, wound infection and puerperal sepsis.

Perinatal Mortality in Multipara

AUTHOR	**PERCENTAGE**
Jacob et al (1972)	25.0%
Klein et al (1963)	11.6%
Kasturilal (1972)	19.6%
Praag et al (1968)	7.1%
Sikdhar et al (1980)	13.5%
Present study	15.6%

Perinatal mortality is very high when cesarean section is performed as an emergency procedure as in placenta previa, accidental hemorrhage, toxaemia, cord prolapse and obstructed labour. Common causes of neonatal deaths are prematurity, fetal asphyxia and septicemia.

5.10 Morbidity in second stage cesarean sections:

Intrapartum and postpartum morbidity is more common in second stage cesarean deliveries than the first stage. According to Alexander James, Leveno, Kenneth et al cesarean deliveries performed in second stage were associated with longer operative time , epidural analgesia, chorioamnionitis and higher birth weights[34]. In our study second stage cesarean section were associated with intraoperative difficulties such as such as uterine incision extension (2 cases), uterine atony requiring cesarean hysterectomy (1 case) and longer operating time. 45.45% of the patients required intraoperative or postoperative blood transfusion. The mean birth weight of these babies delivered by second stage cesarean section was 3.25 kg compared to 2.76 kg for the whole study group. Among the post operative complications 2 patients had wound disruption requiring resuturing. NICU admissions were seen in 54% of cases. Perinatal mortality rate was 18.18%.

Perinatal Mortality In Multipara

AUTHOR	PERCENTAGE
Jacob et al (1972)	25.0%
Klein et al (1963)	11.6%
Kasturilal (1972)	19.6%
Praagh et al (1968)	7.1%
Sikdhar et al (1980)	13.5%

Perinatal mortality is very high when cesarean section is performed as an emergency procedure as in placenta previa, accidental hemorrhage, toxaemia, cord prolapse and obstructed labour. Common causes of neonatal deaths are prematurity, foetal asphyxia and septicemia.

Not only mothers, babies are also vulnerable to unnecessary risks from rising cesarean section rates. The first danger to the baby is the 1% to 9% chance that the surgeon's knife will accidentally lacerate the fetus (6% in nonvertex presentation). A much more serious risk is respiratory distress syndrome (RDS). Cesarean section per se is a potential risk factor for RDS in preterm infants and for other forms of respiratory distress in mature infants. Another distinct hazard is iatrogenic prematurity. Even with repeated ultrasound scans, there may be errors in judging when to do an elective cesarean section. As cesarean section rates rise, so do premature births. While in USA more infants were born in 2004 by cesarean section, more were born prematurely and more were born with a low birth weight in 2004 than in 2003. Both RDS and prematurity are major causes of neonatal mortality and morbidity[32].

The important causes of fetal mortality being antepartum hemorrhage, obstructed labour with intrauterine death and cord prolapsed and associated medical disorders like diabetes mellitus.hazardous. If used to overcome a dysfunctional labour with a possible and not definite disproportion, a careful scrutiny with a definite cut off time of the oxytocin should be practiced.

In the study by Klein et al, incidence of primary cesarean section in multipara was 14.5% (27 cases). All infants delivered by cesarean section in this series were larger than the largest infant previously delivered through the vagina[18].Klien states that high incidence of forceps deliveries and high mortality as noted in previous obstetric history were significant pointers to suspected possible disproportion between the fetus and maternal pelvis.

From the above studies it is evident that disproportion does occur in multipara, though osteomalacia as an etiological factor may not be encountered in the present day. It is to be stressed that there is a tendency to allow even closely observed patients to go too long in a nonproductive type of labour just because they are multipara.

Klein states that multipara in early labour with foetal head not engaged should receive the same careful investigation for cephalo-pelvic disproportion that a primigravida would receive. The fact that the multipara has had one or more vaginal deliveries should be regarded as an optimistic fact but not diagnostic criteria for spontaneous delivery of the fetus. Reluctance to diagnose this cephalopelvic disproportion leads to a longer labour, with development of excessive moulding and caput formation which makes the observer to believe that progress has been made. Many times, delivery with forceps is attempted and fails[18].

Duckman et al states that cephalopelvic disproportion in a multipara can be more significant and more dangerous than in primi because of the delay in recognition. Earliest recognition of its existence is made possible by more frequent discussion of the problem.

VI. Conclusion

Multiparity is a problem associated with poverty, illiteracy, ignorance and lack of knowledge of the available antenatal care and family planning methods. A multipara who has earlier delivered vaginally may still require a cesarean section for safe delivery. Primary cesarean sections in multipara constitute only a small percentage of total deliveries (1.99%) but are associated with high maternal and fetal morbidity.

Anemia, antepartum haemorrhage, malpresentations and severe pre-eclampsia were most common associated preoperative complications. Fetal distress (24%), antepartum haemorrhage (24%), malpresentations (15%) and fetopelvic disproportions (6%) were most common indications for cesarean sections. The highest maternal morbidity (90.9%) in the study was seen in patients undergoing second stage cesarean sections and the highest perinatal mortality (56.25%) was seen in women with antepartum haemorrhage.

Good intrapartum and postpartum care have eliminated maternal deaths in our study. Unrecognized cephalopelvic disproportion leading to obstructed labour (in referred cases) has increased the maternal morbidity. Hence a multiparous women in labour requires the same attention as that of primigravida. Good antenatal and intrapartum care and early referral will reduce the maternal and perinatal morbidity and mortality in multipara.

.

References

[1]. Peter H. Diagnosis and classification of Diabetes mellitus and impairedglucose tolerance. Bennet, Joslin's. 13th edition. pp. 193.
[2]. Ramchandran A, Das AK. API text book of medicine. 7th edition. XVIII-Diachetrlo I. Basic consideration of diabetes mellitus. pp.1097.
[3]. Sahib AKY. Study of ciprofloxacin resistant Escherichia coli (CREC) in type2 diabetic patients with symptomatic urinary tract infections. Iraq J CommMed, 2008; 21(1): 58-63.
[4]. Young KR & Calncy CF. Urinary tract infection complicating diabetesmellitus. Med Din Noth Am, 1955; 39: 1665.
[5]. Ooi BS, Chen B. Prevalence and site of bacteriuria in Diabetes Mellitus.Pool Grad Med J,1974;50:497.
[6]. American diabetes association 2003 position statement Diabetes carecolumn 26, supplement 1, January2003. pp 21.
[7]. Bonadio M, Costarelli S, Morelli G, Tartaglia T. The influence of diabetesmellitus on the spectrum of uropathogens and the antimicrobial resistance inelderly adult patients with urinary tract infection. BMC Infect. Dis. 2006; 6:54.
[8]. Valerius NH, Eff C, Hansen NE et al. Neutrophil and lymphocyte function inpatients with diabetes mellitus. Acta Med Scand, 1982; 211: 463–7.
[9]. Stapleton A. Urinary tract infections in patients with diabetes. Am J Med,2002; 113(1): 80-84.
[10]. Hosking DJ, Bennett T, Hampton JR. Diabetic autonomicneuropathy. Diabetes Care, 1978;27:1043–54.
[11]. Kadri SM, Gash B and Rukhsana A. Antibiotic Sensitivity and ResistanceProfile of the Micro-organisms Responsible for Urinary Tract Infection Observed in Kashmir, India. Indian Journal for the Practicing Doctor, 2004; 1(1): 79-84.
[12]. Orenstein R and Wong ES. Urinary tract infections in adults. Am FamPhysician, 1999; 59(5): 1225-1234.
[13]. Hackett G. Urinary tract infection (UTI). Updated 2005 Oct.
[14]. Baldwin AD, Root HF. Infections of the upper urinary tract in the diabeticpatient. N Engl J Med, 1940;223:244-9.
[15]. Sharkey TP, Root HF. Infections of urinary tract complicating diabetesmellitus. JAMA, 1935;104:2231-5.
[16]. Robbins SC, Tucker AW. The cause of death in diabetes. N Engl J Med,1944;231:865-8.
[17]. Huvos A, Rocha J. Frequency of bacteriuria in patients with diabetesmellitus.N Engl J Med 1959;261:1213-6.
[18]. Rengards RT. Asymptomatic bacteriuria in sixty-eight diabetic patients. AmJ Med Sci, 1960;239:159-64.
[19]. Szucs S, Cserhati, Csapo G, Balazs V. The relation between diabetesmellitus and infections of the urinary tract. Am J Med Sci, 1960;240:186-91.
[20]. Vejlsgaard R. Studies on urinary infections in diabetics. Bacteriuria inpatients with diabetes mellitus and in controls. Acta Med Scand,1966;179:173-82.
[21]. Andriole VT. Asymptomatic bacteriuria in patients with diabetes—enemy orinnocent visitor?. N Engl J Med, 2002;347:1617-8.
[22]. Wisinger DB. Urinary tract infection: current management strategies. Postgrad Med, 1996;100(5): 229-36.
[23]. Manduru M. Urinary Tract Infections. Infectious Diseases Module . Fallsemester, PHPR 6440. Available from www.utoledo.edu/colleges/pharmacy/clinical/utis. html - 31k.
[24]. Mc Rac NS, Linda M and Daiiriki S. Bacterial Infection of the GenitourinaryTract. In Smith General Urology, International edition, Emil, A. and McAninch, W. 15th Ed, Lange Medical Books, 2000; 237-243.
[25]. Colgan R, Williams M. "Diagnosis and treatment of acute uncomplicatedcystitis." American family physician. 2011; 84 (7): 771–6.
[26]. Salvatore S, Cattoni E, Siesto G, Serati M, Sorice P, Torella M. "Urinarytract infections in women.". European journal of obstetrics, gynecology, andreproductive biology. 2011;156 (2): 131–6.
[27]. Nicolle LE. "Uncomplicated urinary tract infection in adults includinguncomplicated pyelonephritis". Urol Clin North Am, 2008; 35 (1): 1–12.
[28]. Mazzulli T, Skulnick M, Small G, Marshall W, Hoban D J, Zhanel GG, Finn Sand Low DE. Susceptibility of community Gram negative urinary 2001.
[29]. Bhat RG, Katy TA, Place FC. "Pediatric urinary tract infections.".Emergencymedicine clinics of North America. 2011 Aug; 29 (3): 637–53.
[30]. Larcombe J. Urinary tract infection in children. BMJ, 1999; 319(7218): 1173-1175.
[31]. Nicolle LE. Resistant pathogens in urinary tract infections., J Am GeriatrSoc, 2002; 50 (Suppl), S230 - S235.
[32]. Smaill F, Vazquez JC. "Antibiotics for asymptomatic bacteriuria inpregnancy." Cochrane Database Syst Rev 2007; (2): CD000490.
[33]. Dielubanza EJ, Schaeffer AJ. "Urinary tract infections in women.". TheMedical clinics of North America. 2011 Jan; 95 (1): 27–41.
[34]. Nicolle LE. "The chronic indwelling catheter and urinary infection in long-term-care facility residents". Infect Control Hosp Epidemiol. 2001; 22 (5):316–21.
[35]. Phipps S, Lim YN, Mc Clinton S, Barry C, Rane A, N'Dow, J. "Short termurinary catheter policies following urogenital surgery in adults". CochraneDatabase of Systematic Reviews, 2006; (2): CD004374.
[36]. Gould CV, Umscheid CA, Agarwal RK, Kuntz G, Pegues DA. "Guideline forprevention of catheter-associated urinary tract infections 2009". InfectControl Hosp Epidemiol. 2010; 31 (4): 319–26.

[37]. Harris, Richard. "Genitourinary infection and barotrauma as complications of'P-valve' use in drysuit divers". Diving and Hyperbaric Medicine. 2009Dec;39(4):210–2.
[38]. Stamm WE . Urinary Tract Infection and Pylonepritis. In Harrisons Principlesof internal medicine. 2005. eds. Kasper DL, Braunwald E, Fauce AS, HauserS, Longo DL and Jameson L J. 16th ed. Mc Graw Medical publishingdivision, New York, 2, 1715-1719.
[39]. Schneeberger C, Geerlings SE, Middleton P, Crowther CA. "Interventionsfor preventing recurrent urinary tract infection during pregnancy." TheCochrane database of systematic reviews. 2012 Nov; 11: CD009279.
[40]. Foxman B. Epidemiology of urinary tract infections: incidence, morbidity,and economic Costs. The American Journal of Medicine, 2002;113(1A): 5S -13S
[41]. Siroky MB. Pathogenesis of bacteriuria and infection in the spinal cordinjured patient., Am J Med, 2002;113 (Suppl 1A): 67S - 79S.
[42]. Faro S and Fenner DE. Urinary tract infections., Clinical Obsterics andGynecology, 1998; 41(3): 744-754.
[43]. Barnett BJ and Stephens DS. Urinary tract infection: an overview. TheAmerican Journal of Medical Science, 1997; 314 (4): 245-249.
[44]. Mullenix T A and Prince RA. Urinary tract infections and prostatitis., InPharma- cotherapy: A Pathophysiologic Approach, 4th ed, eds. DiPiro JT,Talbert RL and Yee GC, Connecticut, Appleton & Lange, Stamford, 1999;1779 - 1794.
[45]. Baron EJ, Peterson R and Fingold M. Microorganism encountered in theurinary tract. in Bailey and Scott.s Diagnostic Microbiology, ninth ed, Mosby,St Louis Boston, Chicago, 1998; 249-267.
[46]. Neill MA, Tarr PI, Taylor DN and Trofa AF. Escherichia coli. in Food borneDisease Handbook, eds. Hui YH, Gorham JR, Murell KD and Cliver DO, Inc.New York, 1994; 169-213.
[47]. Caplenas NR and Kanarek MS. Thermotolerant non-fecal source Klebsiellapneumoniae validity of the fecal coliform test in recreational waters.,American Journal of Public Health, 1984; 74: 1273-1275.
[48]. Young KR & Calncy CF. Urinary tract infection complicating diabetesmellitus, Med Din North Am, 1955; 39; 1665.
[49]. Hall LM, Duke B, Urwin G et al. Epidemiology of Enterococcus faecalisurinary tract infection in a teaching hospital in London. United Kingdom. JClin Microbiol. 1992;30:223.
[50]. Subramanium O, Sivaramam U, Kumar S, Selvaraj S, Shanmugan N.Antibiotic resistance pattern of biofilm-forming uropathogens isolated fromcatheterized patients in Pondicherry, India. AMJ. 2012; 5(7): 344-348.
[51]. Todar, K. Pathogenic E. coli., in Online Textbook of Bacteriology, Universityof Wisconsin - Department of Bacteriology. 2007, pp.11- 30.
[52]. .Johnson J, Kuskowski M, Menard M, Gajewski A, Xercavins M and Garau J.Similarity between human and chicken Escherichia coli isolates in relation tociprofloxacin resistance status., Journal of Infectious Diseases, 2006;194(1): 71-78.
[53]. Salyers AA, Gupta A and Wang Y. Human intestinal bacteria as reservoirsfor antibiotic resistance genes., Trends Microbiol, 2004;12 (9): 412.416.
[54]. Eickhoff TC. 1972. Klebsiella pneumoniae infection: a review with referenceto the water- borne epidemiologic significance of K. pneumoniae presence inthe natural environment, national Council of the Paper Industry for Air andStream Improvement. Inc. Technical Bulletin no. 254, New York.
[55]. Brochert A. 1999. Klebsiella: one potentially nasty bacteria.PersonalMD.com.Availablefromhttp://www.personalmd.com/news/klebsiella_102299. shtml 33k.
[56]. Qarah S. 2005. Pseudomonas aeruginosa infections, [cited 2005 12Dec].
[57]. Gonzalez G and Bronze MS. 2006, Proteus Infection., e-Medicine availablefrom: http://www.emedicine.com/med/TOPIC1929.HTM [Updated [2006 2March]
[58]. Russso TA. Diseases caused by Gram negative bacilli. in HarrisonsPrinciples of Internal medicine, eds. Kasper DL, Braunwald E, Fauce AS,Hauser SL, Longo DL and Jameson LJ. 16th ed, 2005; 1: 878-885.
[59]. Raz R, Colodner R and Kunin CM. 2005, .Who are you- Staphylococcussaprophyticus? 2005; 40: 896-898.
[60]. Hooton TM, Schles P, Hugher JP, Winter C, Roberts PL, Stapleton A andStamm WE. A prospective study of risk factors for symptomatic urinary tractinfection in young women., The New England Journal of Medicine,1996;335(7): 468 - 474.
[61]. Bates JM, Raffi A and Prasadan K. Tamm-Horsfall protein knockout miceare more prone to urinary tract infection., rapid communication, Kidneyinternational, 2004; 65(3): 791-797.
[62]. Mims CA, Playfair JH, Roitt IM, Wakelin D and Williams R. Urinary tractinfection. in Medical Microbiology, 1st ed. Mosby year book Europe Ltd.,London, 1993; 23.1-23.8.
[63]. Johnson CC. Definitions, classification, and clinical presentation of urinarytract Infections. Medical Clinics of North America, 1991; 75(2): 241-252.
[64]. Gupta K. Addressing antibiotic resistance. The American Journal ofMedicine, 2002; 113 (Suppl 1A): 29S - 34S.
[65]. Hooton TM, Besser R, Foxman B, Fritsche TR and Nicolle LE. AcuteUncomplicated Cystitis in an Era of Increasing Antibiotic Resistance: AProposed Approach to Empirical Therapy., Clinical Infectious Diseases,2004; 39: 75.80.
[66]. Zhanel GG, Karlwsky JA, Harding GKM, Carrie A, Mazzuli T and Low DE. ACanadian national surveillance study of urinary tract isolates fromoutpatients: comparison of the activities of trimethoprim - sulfamethoxazole,ampicillin, mecillinam, nitrofurantoin, and ciprofloxacin., The CanadianUrinary isolate Study Group. Antimicrob Agents Chemother, 2000; 44(4):1089-1092.
[67]. Gupta K, Thomas M and Stamm WE. Increasing antimicrobial resistanceand the management of uncomplicated community acquired UTIs. Annals ofinternal medicine, 2001; 135: 41-50.
[68]. Zilevica A. Hospital-acquired and Community- acquired Uropathogens,Modelling of Infection., Bioautomation, 2005; 3: 63 - 67.
[69]. Orrett FA and Davis G. Comparison of Antimicrobial Susceptibility Profile ofUrinary Pathogens for the Years, 1999 and 2003., West Indian Med J, 2006;55 (2): 95.
[70]. Collee JG, Duguid JP, Fraser AG and Marmion BP. Laboratory strategies inthe diagnosis of infectious syndromes. in Practical Medical Microbiology,eds Collee JG, Barrie MP, Fraser AG and Simons A.14th ed. ChurchillLivingstone, New York, 1996; 53 - 93.
[71]. Kass EH. Pregnancy, pyelonephritis and prematurity. Clin Obstet Gynaecol1970; 13: 239-54.
[72]. Toedter N. 2000. Urinary Tract Infection, LADUR Education Article, [on theinternet] Available from http://rxweb.ulm.edu/pharmacy/oore/2000/20Feb-Mar.pdf .
[73]. Goswami R, Bal CS, Tejaswi S. Prevalence of urinary tract infection & renal scars in patients with Diabetes mellitus. Diabetes Res Clin Pract. 2001 Sep;53(3):181-6.
[74]. Harding GK, Zhanel GG, Nicolle LE, Cheang M. Antimicrobialtreatment in diabetic women with asymptomatic bacteriuria. NEngle J Med 2003; 348(10): 957-8.
[75]. Bonadio M, Boldrini E, Forotti G et al. Asymptomatic bacteriuriain women with diabetes: influence of metabolic control. ClinInfect Dis 2004;38:41-5.

[76]. Bonadio M, Costarelli S, Morelli G, Tartaglia T.The influence ofdiabetes mellitus on the spectrum of uropathogens and theantimicrobial resistance in elderly adult patients with urinary tract infection. BMC Infect Dis 2006; 6:54.
[77]. Harding GK, Zhanel GG, Nicolle LE, Cheang M. Antimicrobialtreatment in diabetic women with asymptomatic bacteriuria. NEngle J Med 2003; 348(10): 957-8.
[78]. Choice of Antibacterial drugs. Treatment Guidelines from TheMedical Letter. 2007 May; 5(57).
[79]. Geerlings SE. Urinary tract infections with diabetes mellitus:epidemiology, pathogenesis and treatment. Int J AntimicrobAgents. 2008 Feb; 31(1): S54-7.
[80]. Porpon Rotjanapan, David Dosa. Asymptomatic verusSymptomatic urinary tract infections in Long term care facilityresidents. Geriatrics for the practicing physician. 2009 Nov;92(11): 377-379.
[81]. Shill MC, Moain FB, Karmakar UK. Prevalence of uropathogensin diabetic patients and their corresponding resistant pattern:Results of a survey conducted at Diagnostic centre in Dhaka,Bangladesh. Oman Med J 2010; 25(4): 282-85.
[82]. Mehvish Saleem, Betty Daniel. Prevalence of Urinary Tract Infection among Pateints with Diabetes in Bangalore City. Int. J. Emerg. Sci, 2011 June; 1(2):133-142.
[83]. Raman BV, Chaudhury A. Prevalence of uropathogens in diabetic patients and their resistance pattern at a tertiary care centre in South India. Int J Boil Med Res. 2012; 3(1): 1433-35.
[84]. Hamdan ZH, Eman Kubbara, Amar M Adam, Onab SHassan, Sarah O Suliman and Ishag Adam. Urinary tract infections and antimicrobial sensitivity among diabetic patients at Khartoum, Sudan. Annals of Clinical Microbiology and Antimicrobials. 2015; 14(26).
[85]. Priyadharshini A, Mangaiyarkarasi T, Balasubramaniam R, Senthil Praksh D, Gopal R. Biofilm production and antibiotic resistance among uropathogens causing bacteriuria in diabetic individuals. SJAMS 2014; 2(2A): 568-571.
[86]. Mackey & Mc Cartney Practical Medical Microbiology. 14 th edition. Elsevier publications, Chapter 5 – Specimen collection, cuture containers and media, pp. 95 – 111.
[87]. Betty A Forbes, Daniel F Sahm, Alice S Weissfeld. Laboratory cultivation and isolation of bacteria. Bailey &Scott's Diagnostic Microbiology 2007. 12th ed. Mosby, St Louis, Missouri.
[88]. Howes DS and Henry S. 2008, Urinary Tract Infection Female. [on the internet] e medicine 2008, [updated 2008 Jan], Available from http://www.emedicine.com /EMERG/topic626.htm
[89]. Collee JG, Duguid JP, Fraser AG and Marmion BP. Laboratory strategies in the diagnosis of infectious syndromes. Practical Medical Microbiology, eds Collee JG, Barrie MP, Fraser AG and Simons A.14th ed. 1996. Churchill Livingstone, New York, 53-93.
[90]. Tullu MS. Urinary catheter related nosocomial infections in paediatric intensive care unit., Indian Journal of Medical Microbiology, 1998; 44: 35-39.
[91]. Koneman EW, Allen SD, Janda WM, Schreckenberger PC, Winn JC Jr, editors. Color Atlas and Textbook of Diagnostic Microbiology 2006. 6th ed. 624–662. Philadelphia: Lippincott-Raven.
[92]. Stamm WE. Urinary Tract Infection and Pylonepritis., in Harrisons Principles of internal medicine, eds. Kasper D L, Braunwald E, Fauce AS, Hauser S, Longo DL and Jameson LJ. 2005. 16th ed. Mc Graw Medical publishing division, New York, 2, pp.1715-1719.
[93]. Johnson CC. Definitions, classification, and clinical presentation of urinary tract Infections. Medical Clinics of North America, 1991; 75(2): 241-252.
[94]. Mackie & Mc Cartney Practical Medical Microbiology. 14 th edition. Elsevier publications, Chapter 7 – Tests For Identification of Bacteria, pp. 131 – 150.
[95]. Danaei G, Finucane MM, Lu Y, Singh GM, Cowan MJ, Paciorek CJ et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. Ê Lancet 2011; 378(9785): 31-40.
[96]. World Health Organization (WHO). South Eastern Asia Region: Nepal statistics summary (2002- present). Updated 2011; cited 2013. Available from: http://www .who.int/nmh/countries/npl_en.pdf.
[97]. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes care, 2004; 27:1047-53.
[98]. Najar MS, Saldanha CL, Banday KA. Approach to urinary tract infections. Ind J Nephro 2009;19(4),129-39.
[99]. Sibi G, Devi AP, Fouzia K, Patil BR. Prevalence, Microbiologic Profile of Urinary tract Infection and its treatment with Trimethoprim in Diabetic Patients. Res J Microbiol, 2011; 6: 543- 51.
[100]. Connolly A, Thorp JM, Urinary tract infection in pregnancy. Urol Clin North Am 1999; 26 (4): 779-8.
[101]. Haider G, Zehra N, Munir AA, et al. Risk factors of urinary tract infection in pregnancy. J Pak Med Assoc, 2010;60(3): 213 -216.
[102]. Nicolle LE, Friesen D, Haridng GK, Roos LL. Hospitalization for acute pyelonephritis in Mnaitoba, Canada, during the period from 1989 - 1992; impact of diabetes, pregnancy, and aboriginal origin. Clin Infect Dis, 1996; 22: 1051 - 6.
[103]. Sharma BD, Rohit Bansal, Gupta B. Asymptomatic bacteriuria in diabetics. Journal Indian Academy of Clinical Medicine, 2012 Mar; 13(1): 55 - 59.
[104]. Md Hamza Saber, Lovely Barai, J Ashraful Haq, Md Sharifiul Alam Jilani, Jaheda Begum. The Pattern of organism causing urinary tract infection in Diabetic and Non Diabetic patients in Bangladesh. Bangladesh J Med Microbiol 2010; 4(1): 6-8.
[105]. Acharya D, Bogati B, Shreshtha GT, Gyawali P. Diabetes Mellitus and Urinary tract infection : spectrum of uropathogens and their antibiotic sensitivity pattern. JMMIHS, 2015; 1(4): 24 - 28.
[106]. Jha N, Bapat SK. A study of sensitivity and resistance of pathogenic micro-organisms causing UTI in Kathmandu Valley. Kathmandu Univ Med J 2005; 3(2):123-9.
[107]. Vishal Sharma, Vishal Gupta, Mridula mittal. Prevalence of uropathogens in Diabetic patients and their antimicrobial susceptibility pattern. National Journal of Laboratory Medicine, 2012 June; 1(1): 26-28.
[108]. Yeshitela B, Gebre-Selassie S, Feleke Y. Asymptomatic bacteriuria and symptomatic urinary tract infections (UTI) in patients with diabetes mellitus in Tikur Anbessa Specialized University Hospital, Addis Ababa, Ethiopia. Ethiop Med J. 2012 Jul;50(3):239-49.
[109]. Patterson 1b, Andriole VT. Bacterial urinary tract infections in diabetes. Infect Dis Clin North Am 1997; 11: 735-50.
[110]. Sabrina J Said A, Mambula K, et al Bacteral isolates and drug susceptibility patterns of urinary tract infection among pregnant mothers at Muhimbili National Hospital in Tanzania. Journal of Health Research, 2010; 12: 4 – 14.
[111]. Alemu A, Mogus F, Tefas A et al. bacterial profiles and drug susceptibility patterns of urinary tract infection in pregnant women at university of Gonda teaching hospital North West Ethiopia. BMC Research notes, 2012; 5:197.
[112]. Le J, Briggs FF, Mackeown et al. Urinary tract infection during pregnancy. Anna pharmacotherapy, 2004; 38(10): 1692-701.
[113]. Pozzilli P, Leslie RD. Infections and diabetes: mechanisms and prospects for prevention. Diabet Med 1994; 11: 935-41.

[114]. Joshi N, Caputo GM, Weitekamp MR et al. Infections in patients with diabetes mellitus. N Eng J Med 1999; 341: 1906-12.
[115]. Andriole V. Asymptomatic bacteriuria in patients with diabetes—enemy or innocent visitor?. N Engl J Med, 2002; 347: 1617-8.
[116]. Wheat LJ. Infection and diabetes mellitus. Diabetes Care, 1980; 3: 187-97.
[117]. Keane EM, Boyko EJ, Reller LB, Hamman RF. Prevalence of asymptomatic bacteriuria in subjects with NIDDM in San Luis Valley of Colorado. Diabetes Care,1988; 11: 708-12.
[118]. Geerlings SE, Stolk RP, Camps MJ et al. Asymptomatic bacteriuria may be considered a complication in women with diabetes. The Diabetes Mellitus Women Asymptomatic Bacteriuria Utrecht Study Group. Diabetes Care 2000; 23: 744-9.
[119]. Pometta D, Rees SB, Younger D, Kass EH. Asymptomatic bacteriuria in diabetes mellitus. N Engl J Med, 1967; 276: 118-21.
[120]. Bonadio M, Pulitanò L, Catania B, Marchetti P, Miccoli R, Navalesi R. Urinary tract infection in women with controlled diabetes. In: Losse H, Asscher AW, Lison AE, Andriole VT, editors. Pyelonephritis. Stuttgart, Germany : Georg Thieme Verlag. 1984; 5: 109-13.
[121]. Brauner A, Flodin U, Hylander B, Ostenson CG. Bacteriuria, bacterial virulence and host factors in diabetic patients. Diabet Med, 1993;10:550-4.
[122]. Batalla MA, Balodimos MC, Bradley RF. Bacteriuria in diabetes mellitus. Diabetologia 1971; 7: 297-301.
[123]. Mario Bonadio, Elisabetta Boldrini, Giovanna Forotti, Elena Matteucci, Armando Vigna, Stefano Mori and Ottavio Giampietro. Clinical Infectious Diseases. 38(6): pp. e41-e45.
[124]. Nicolle LE. Asymptomatic bacteriuria in diabetic women. Diabetes Care 2000; 23: 722-3.
[125]. Zhanel GG, Harding GKM, Nicolle LE. Asymptomatic bacteriuria in patients with diabetes mellitus. Rev Infect Dis 1991;13:150-4.
[126]. Mehvish Saleem, Betty Daniel. Prevalence of Urinary tract infection among patients with Diabetes in Bangalore city . Int J Emerg Sci, 2011 June: 1(2): 133-142.
[127]. Kelestimur F, Unal A, Pasaoglu H, Basar E, Kilic H, Doganay M: Asymptomatic bacteriuria in patients with diabetes mellitus. Mikrobiyol Bul 1990, 24(2):126-32.
[128]. Marie E A Bissong, Peter N Fon, Fritz O Tabe - Besong and Theresa N Akenji. Asymptomatic bacteriuria in diabetes mellitus patients in Southwest Cameroon. Afr Health Sci, 2013 Sep; 13(3): 661-666.
[129]. Abhilash KPP, Balaji Veeraraghavan, Abharam OC. Epidemiology and outcome of bacteremia caused by Extended Spectrum Beta - Lactamase producing Escherichia coli and Klebsiella spp in a tertiary care teaching hospital in South India. JAPI, 2010 Dec, 58.
[130]. Bacheller, C. D. and Bernstein, J. M. 1997, .Urinary tract infections., Medical Clinics of North America, 81(3), 719-730.
[131]. Davison, A. M., Cumming, A. D. and Swainson, C. P. 1999, .Diseases of the kidney and urinary system. in Davidson.s Principles and Practice of Medicine, eds. Haslett,H., Silver, E. R., Boon, N. A. and Hunter, J. A. A., 18th ed, Churchill Livingstone, Edinburg , pp. 417-4

Dr. Leena Ghattam. "A study of Primary cesarean section in Multiparous women." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 3, 2019, pp 14-26.