Available online at **www.ijmrhs.com**

ISSN No: 2319-5886

International Journal of Medical Research & Health Sciences, 2016, 5, 6:102-121

Study of cases of still births at tertiary maternity care hospital (ReCoDe)

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ABSTRACT

To study the characteristics of pregnancies which end in antepartum still births using the classification system-ReCoDe (Relevant Condition at Death) and to suggest measures to reduce incidence of still births in our study population. The study is a retrospective study of cases of stillbirths. In the present study 100 cases with still births admitted to Deptt. Of Obs & Gynecology Govt Medical College, Sir.T.Hospital, Bhavnagar fulfilling the inclusion criteria was included. All these cases were subjected to routine investigations and the placenta with cord was sent for HPE in all the cases and followed up to one week in the postpartum period. All the cases were classified according to relevant condition at death classification of still births (ReCoDe). On classifying the aetiology of stillbirths in all cases according to the ReCoDe classification we were able to classify 90% of cases and only 10% remain unclassified. In the present study 28% stillbirths were attributed IUGR as a cause, 22% cases mother had hypertensive disorder, 21% with antepartum haemorrhage as a cause and 15% with congenital fetal anomaly. Of 100 cases 53 had a positive correlation in placental and cord on histopathological examination. The present study helped to classify and study the aetiology of stillbirths in study population in simplified way on condition relevant at death. This study lets us know that most of the causes can be taken care of by instituting appropriate measures at right time. The importance of antenatal care, nutrition, counseling early detection and medical help, timely referral to tertiary care hospital, awareness among the patients, doctors and the society can be emphasized in reducing the incidence of stillbirths.

Keywords: stillbirths; ReCoDe classification; IUGR; antepartum haemorrhage; histopathological examination.

INTRODUCTION

The ICD 10 defines fetal death as death prior to complete expulsion of fetal from its mother irrespective of gestational age.[1]

But WHO defines still births as baby born with no signs of life at or after 28 weeks of gestation or if weight is >1000 gm when gestation age is not available.

Perinatal mortality includes still birth and death of the neonate (7 days of life). The antepartum still births are a major contributor to perinatal mortality. So to reduce the perinatal mortality ,reduction of still births is necessary and for this better understanding of aetiology of antepartum still births is important.

The lowest rate of still births has been reported in Finland and Singapore (2 per 1000 births).

98% of still births occur in low and middle income countries. Almost half of the still births occur when women is in labour.

It is estimated that half of the still births in the world occur in: India, Pakistan, Bangladesh, Nigeria and China alone.[2]

In India, rates vary from 20-66 per 1000 births in different states.

Still birth rates are commonly used as indicators for quality of care in individual units but do not address any methods for improving deficiencies in care that may be able to prevent occurrence of still birth.

The value of any death classification system is closely aligned with its ability to identify the causes of death and the key factor which started the chain of events leading to death.[3]

The new ReCoDe classification system derived by a study in midlands region over period of seven years from 1997-2003 [4] By this classification most common cause was fetal growth restriction (43%) and only 15.2 % still births remain unexplained.

Need to "do the best we can" with available information. It is noteworthy that the optimal evaluation of stillbirth is controversial and is influenced by several medical and non-medical factors. First, the true proportion of stillbirths due to any single etiology is largely unknown due to a relative lack of comprehensive, population based studies and debate regarding the definitive attribution of a stillbirth to a particular etiology. Second, many causes of stillbirth remain to be elucidated and investigation into previously unrecognized causes of stillbirth has been limited. Third, stillbirth is an emotionally charged event and different families and cultures will have varied levels of comfort with procedures such as autopsy or genetic testing. Finally, in most settings, cost of testing must be considered prior to initiating a comprehensive evaluation for potential causes of stillbirth.

When deciding which tests are most useful, two principles seem appropriate to consider. First, it is most cost effective to test for the most common causes of stillbirth. Second, it is desirable to identify conditions that predispose couples to recurrent stillbirth as opposed to sporadic pregnancy loss. Identifying a sporadic cause of stillbirth helps bring emotional closure to couples and provides reassurance in future pregnancies. The identification of a sporadic cause of stillbirth also may allow couples to avoid unnecessary tests and interventions in subsequent pregnancies.[5]

Histologic evaluation of the placenta, membranes, and umbilical cord also is quite valuable. This can provide insight into many different potential etiologies of stillbirth including infection, genetic abnormalities, anemia, and thrombophilias. As with autopsy, trained pathologists and a scientific, systematic evaluation are critical. It is noteworthy that placental evaluation is increasingly advised for medico-legal purposes in all cases of adverse perinatal outcome (e.g. preterm birth) including stillbirth. It is rare for families to refuse placental evaluation.[5]
The most convincing proof of an infectious etiology for a stillbirth is a carefully histologic evaluation of the placenta, membranes and umbilical cord. The pathologist may then proceed with appropriate cultures and nucleic acid specimens (for bacteria or viruses) taken for organisms suspected based on histology. Serology for intrauterine viral and protozoal agents including toxoplasmosis, rubella, CMV, herpes (so-called TORCH organisms) is of questionable utility. Although traditionally advised in the evaluation of stillbirth, these titers are rarely clinically useful in the United States (anecdotal experience). Similarly, although routine bacterial culture of the placenta may prove useful, it can be problematic.[5]

It is important to avoid vaginal wall contamination by culturing in between the membranes. It may be useful to culture for ureaplasmas and mycoplasmas in addition to aerobic and anaerobic cultures. However, the incidence of positive cultures in live-born pregnancies is unknown.

Routine testing for antiphospholipid syndrome and/or heritable thrombophilias is controversial. Testing cases characterized by placental insufficiency seems appropriate given the apparent pathophysiology of the conditions. There is limited evidence that treatment in subsequent pregnancies may improve outcome in women with prior stillbirth and these conditions. Accordingly, enthusiasm is considerable for identifying patients with these disorders. There is evidence to support limiting thrombophilia testing to cases with 1) evidence of placental insufficiency such as IUGR or placental infarction, 2) recurrent fetal loss, or 3) personal or family history of thrombosis.[5]

Clinically overt DM and thyroid disease should be excluded. However, screening for subclinical disease is of unproven benefit.

Illicit drug use accounts for a meaningful proportion of stillbirths. Thus, routine toxicology screen is appropriate in most centers. The importance of a good clinical evaluation should not be overlooked when ordering diagnostic tests. Hence this study has been undertaken to know aetiology of stillbirths and classify it accoding to ReCoDe classification and to suggest the measures to reduce its incidence in general population.

AIMS OF THE STUDY

1. To study the characteristics of pregnancies which end in antepartum still births using the classification system-ReCoDe (Relevant Condition at Death)

2. To suggest measures to reduce incidence of still births in our study population.

MATERIAL AND METHODS

Present study was conducted in Department of Obstetrics and Gynaecology at Sir Takhtsinhji Hospital, Govt Medical College Bhavnagar during the period May 2014 to April 2015. 100 cases of pregnancy with still births are included in the present study that fulfilled inclusion criteria

Case Selection: All antepartum stillbirths with gestational age > 28 weeks who presented in the emergency at Deptt of Obstetrics & Gynecology during the study period were included in the study. To know the gestational age at the time of presentation cases were divided in two groups:

1) Gestation age confirmation by known LMP or first trimester ultrasound.
2) Unknown weeks of gestation, the gestational age was rounded off to the nearest gestational age in weeks.

Data Collection: Data relating to sociodemographic information, past history, past obstetric history, associated medical conditions, index pregnancy characteristics were collected for each case.

Still births were classified according to the ReCoDe classification. This system of classification establishes a probable cause for stillbirths.

On admission to the ward detailed history of patient was recorded. According to the scheme laid in the adjacent proforma (Annexure 1) direct questions were asked regarding origin, duration and progress of disease. Detailed obstetric and menstrual history was noted. Clinical examination e.g. general examination, local examination and internal pelvic examination was done in detail.

Routine investigations such as screening, chest X-ray, complete blood count, blood grouping, coagulation profile done. Placenta and its membranes including umbilical cord were sent for histopathological examination and correlation in all the cases. Wherever possible, USG and chest x-ray and ABG analysis of the stillborn baby was done. Data was analysed.

RESULTS AND DISCUSSION

The study was conducted at obstetrics and Gynaecology department, GovtMedicalCollege&, Sir Takhtsingji Hospital, BHAVNAGAR from 1ST MAY 2014 TO 1ST APRIL 2015.There were total 150 stillbirths over this study period and 3746 total births. Out Of these 100 cases of antepartum still births were included in the present study that fulfilled the inclusion criteria.

TABLE 1: Distribution of gender in cases of still births

TOTAL CASES	MALE	FEMALE	UNIDENTIFIED SEX
100	53	46	O1

Table 1 shows distribution of sex in cases of stillbirths. There were total 100 cases of which 53 were males i.e. 53% and 46 were females i.e. 46% and 1 % had un identified sex.(.Amorphous foetus)

In a case control study Dr Kumbhare *et al* (2012) also showed the male sex predominance was seen.

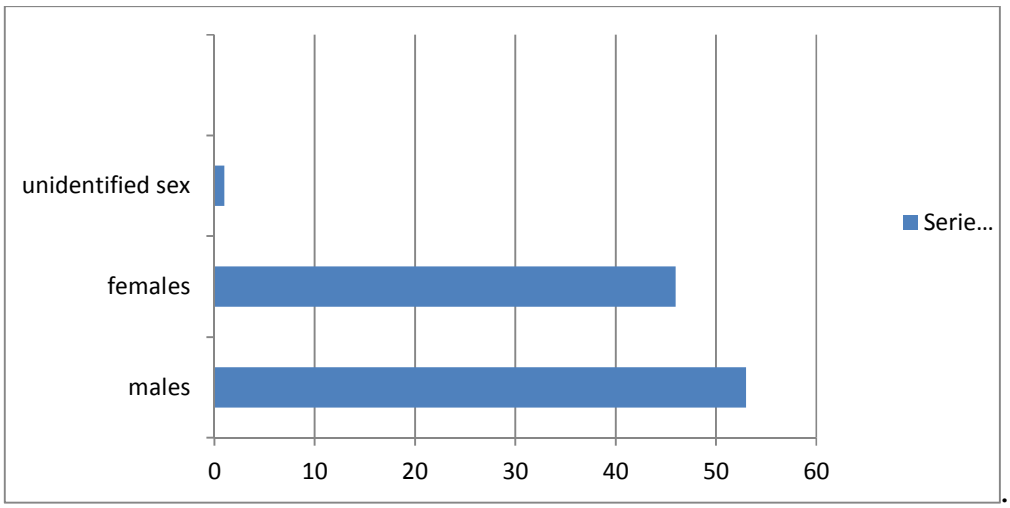

FIG 1: HORIZONTAL BAR GRAPH FOR DISTRIBUTION OF GENDER IN CASES OF STILL BIRTHS

Mondal D, Galloway TS, Bailey, Mathews F *et al* (2014) studied the risk of male fetus in still births. Risk of stillbirth in males is elevated by about 10%. The population-attributable risk is comparable to smoking and equates to approximately 100,000 stillbirths per year globally. The pattern is consistent across countries of varying incomes. Given current difficulties in reducing stillbirth rates, work to understand the causes of excess male risk is warranted. We recommend that stillbirths are routinely recorded by sex. This will also assist in exposing prenatal sex selection as elevated or equal risks of stillbirth in females would be readily apparent and could therefore be used to trigger investigation.

Smith *et al* (2000) studied the risk of gender in causation of stillbirths. He concluded that 1) the association between the male sex and stillbirths diminishes with increasing birth weights and, 2) there was a fall in the proportion of stillbirths in Scotland between 1980-1996, which may have been due to fall in proportion of small babies over the same period. The relative risk for male stillbirths was found to be 1.19 which varied according to birth weight quintile. The main finding of the study was that the increased risk of stillbirth associated with male gender progressively diminishes with birth weight.

TABLE 2: DISRIBUTION OF BIRTHWEIGHTS IN CASES OF STILL BIRTHS

BIRTH WEIGHT IN gms	**1000-1499**	**1500-1999**	**2000-2499**	**>2500**
Cases	17	26	21	36

This table shows that 26% where of 1500-1999 gms birth weight and 36% where normal birth weights >2500gms at term. Hence birth weight was not a significant factor in causing still birth in my study.

Outi Hovatta *et al* (1983) described that predominant feature of the stillborn infants were short gestation and low births weight for gestation. There were no significant differences between the two groups with respect to maternal age, parity distribution, the number of earlier stillbirths and spontaneous and induced abortion.

Alessandri *et al* (1992) performed a matched case control study to analyse the risk factors of unexplained antepartum stillbirths. Infants were matched for year of births, plurality, gender and birth weight of infants and race of mother(Controls). Matching for birth weight was carried out because a number of known risk factors are thoughts to be mediated through low birth weight. Conditional logistic regression was applied and data was dichotomized on the basis of LBW (<2500 grms) and normal birth weight (2500 grms or more). The result was that a large proportion of the unexplained antepartum stillbirths were of LBW (54.6%) making this an important factor in case control comparison.

Gardosi J, Mul T, Mongelli, Fagan D.et al (1998) published a similar study where unexplained antepartum stillbirths amounted to68%, using the Wigglesworth classification. A majority of these were because of LBW. Hence, birth weight was found to be an important risk factor.

Forssas *et al* (1999) discussed that the excess perinatal mortality due to maternal risk factors or to the causal factors behind these risk factors is mainly attributable to their tendency to cause low births weight. Similarly, in a study by McCormic *et al* (1985) on neonatal mortality in United States, controlling for births weight sharply reduced or eliminated the differences associated with the number of maternal factors, but in contradiction the risk posed by advanced maternal age and prior history of fetal loss did not vanish.

Other studies by Neutra et al (1975) , Gruenwald (1969) also have suggested similar analyses where birth weight was found to be a better predictor than gestational age in causing stillbirth.

TABLE 3: DISTRIBUTION OF CASES ACCORDING TO THE GESTATIONAL AGE

GESTATIONAL AGE (weeks)	28-31	32-35	36-39	40 and above
Cases	10	38	43	09

In the present study, 43 % stillbirths in 36-39weeks gestational age . Maximum stillbirths were seen in 36-39 gestational weeks and in 32-35weeks gestational age.

In a case control study DrKumbhare *et al* (2012) observed maximum stillbirths in the gestational age group i.e 36-39 weeks [39].

Onwudeet al (2006) derived that differences in birth weight and gestational age among cases and controls were not statistically significant (P=0.06)

Outi Hovatta et al (1983) described that low gestational age is an important factor for causing intrauterine fetal death.

TABLE 4: DISTRIBUTION OF MATERNAL AGE IN CASES OF STILL BIRTHS

Maternal age in years	20-24	25-29	30 and above
Cases	45	33	21

In the present study the percentage of stillbirths was higher in the age group 20-24 years. This shows that maternal age is not a significant cause for stillbirths.

Recent studies have been done to find association between maternal age and stillbirths.

A Khalil *et al* (2013) has done a cohort study to find association of maternal age and stillbirths. The results of this study were n=55772 in <35 years age group and still births were 195. And n=16325 in 35-40 years age group stillbirth were 75 in that group, and there were 4061 cases in >40 years age group with 20 stillbirths in that group.

In this study they did not find maternal age as significant factor in causing stillbirths.

Similar case control study was done simultaneously by Udibo *et al* (2013) with 824 cases and 1648 controls which showed increased risk of IUGR and stillbirths with increasing maternal age.

Huang *et al* (2008) found that greater maternal was significantly associated with increased risk of stillbirth; relative risk varied from 1.20-4.83 for older versus younger women. The most commonly used definition of advanced maternal age in these studies was 35years or more. A still birth risk among older women was 1.26-1.92 times higher than the risk among women less than 35 years of age. They identified 37 studies, more than 80% of which demonstrated a statistically significant increase in stillbirths with increased maternal age, which was identified as an independent risk factor on multivariate analysis. The biological mechanism of the increase in stillbirth risk with advanced maternal age is uncertain. This would probably be related to low uteroplacental perfusion caused by poor uterine vasculature in older women. The increased risk could also be attributed to the associated between older age

and certain risk factors for stillbirths, such as chronic diseases and medical or obstetric complications. Older women have a higher risk of experiencing pregnancy induced hypertension or gestational diabetes.[25]

Onwude et al (2006) derived that the mean maternal age at delivery was not significantly related to the occurrence of unexplained stillbirths.[26]

Bateman *et al* derived that with the use of ICD-9 codes, the rate of stillbirth was determined as a function of maternal age. The unadjusted rate of stillbirth was elevated from teenagers (OR 1.11; 95% CI 1.08-1.14), and for women 35-39 yrsold(OR 1.28; 95% CI 1.24-1.32) and for women 40 yrs and above (OR 1.72; 95% CI 1.63-1.81).[27]

TABLE 5: DISTRIBUTION OF CASES ACCORDING TO PARITY

Parity	1	2	3	4	5 or more
Cases	39	15	29	12	05

In the present study maximum stillbirths are seen in primipara and 3rd para patients. So the parity doesn't seem to be significant in causing stillbirths.

Huang *et al* (2008) concluded that maternal age and parity are two closely related demographic factors. Few researchers have used multivariate regression analysis to address the interaction between parity and maternal age in terms of its effect on the risk of stillbirths. Further studies may explore this interaction in detail.

Sarkalisonkova *et al* (2010) there was a study to examine the effect of parity on the association between older maternal age and adverse birth outcomes, specifically stillbirth, neonatal death, preterm birth, small for gestational age, and neonatal intensive care unit admission. They conducted a retrospective cohort study of singleton births in British Columbia between 1999 and 2004. In the cohort, 69 023 women were aged 20 to 29, 25 058 were aged 35 to 39, and 4816 were aged 40 and over. Perinatal risk factors, obstetric history, and birth outcomes were abstracted from the British Columbia Perinatal Database Registry. Logistic regression was used to calculate adjusted odds ratios (aOR) and 95% confidence intervals for adverse outcomes in the two older age groups.

Conclusion: Older women were at elevated risk of stillbirth, preterm birth, and NICU admission regardless of parity. Parity modified the effect of maternal age on preterm birth and SGA. Older primiparas were at elevated risk for SGA, but no association between age and SGA was found in multiparas. Older primiparas were at higher risk of preterm birth than older multiparas compared with younger women.

Table 6: Recurrence of stillbirths

Recurrence of SB	**p/h/o stillbirths**
Cases	08

In present study 8% of cases had past history of stillbirths in previous pregnancy. Of these 8 cases with p/h/o stillbirths,3 cases had PIH in previous pregnancy, 2 of them had congenital anomaly and 1 case had anti phospholipid antibodies syndrome and 3 cases no cause could be found.

Puza Sharma *et al* (2007) restricted to a selected group of relatively low-risk mothers. The results confirm the association between prior stillbirth and elevated risk for subsequent fetal demise as reported in the literature. In this study, we observed that stillbirths were more likely to occur after 28 weeks and during the antepartum period. We found that overall, relatively low-risk women who had had a stillbirth in their first pregnancy were about five times more likely to experience stillbirth in their second pregnancy than those with no history of stillbirth. The categorisation of stillbirth subtypes into early and late, as well as antepartum and intrapartum, shed more light on patterns of risks in the second pregnancy in women with a history of stillbirth in the first pregnancy. Compared with late stillbirth, the risk elevation among women with a history of early stillbirth in the first pregnancy was increased by more than fourfold – estimates that were obtained after controlling for potential confounding factors. In a similar fashion, stillbirth recurrence risk varied in magnitude for antepartum and intrapartum stillbirth, with the latter being greater[29]

Onwude *et al* performed a matched case control study in which 75 women who delivered stillbirths were matched with 75 controls. All cases and controls were similar for live birth. Multivariate analysis was performed by Conditional logistic regression while controlling for sets. Cases (women with past stillbirths) were significantly delivered earlier than controls ($p< 0.0001$), with a corresponding smaller baby, although the differences in birth weight were not statistically significant ($p=0.06$). There were no stillbirths in cases and controls during the follow up period of 3 to 7 yrs. Hence it was concluded that a women who has had an unexplained stillbirth at term has no greater risk of recurrence than a matched control.[26]

Bhattacharya *et al* (2010) showed that after adjusting for confounding factors, the odds of recurrence of stillbirths in a second pregnancy was found to be 1.94 (99% CI 1.29-2.92) compared with those who had a previous live birth in the first pregnancy. The adjusted odds ratio for stillbirth in second pregnancy in the presence of placental abruption was 1.96 (99% CI 1.60-2.41) whereas the adjusted odds ratio for preterm deliveries and low birth weight were 7.45(99% CI 5.61-9.39) and 6.69 (99% CI 5.31-8.41) respectively.**[30]**

Kari Klungsoyr Melve *et al* (2010)
Recurrence of Stillbirth in Siblings: Population-based Cohort Study

Among women with a stillbirth in their first pregnancy, 222 (37.0 per thousand) experienced a recurrent second stillbirth compared with 3,507 (6.2 per thousand) with a first live birth. The overall relative odds (i.e., odds ratio) associated with stillbirth recurrence were thus 6.2 (95% confidence interval (CI): 5.4, 7.1). The relative odds associated with early (20–27 weeks) stillbirth recurrence, odds ratio = 27.9 (95% CI: 21.9, 35.6), was considerably higher than that associated with mid/late (≥28 weeks) stillbirth recurrence, odds ratio = 4.2 (95% CI: 3.2, 5.5)

Table 7: ASSOCIATED RISK FACTORS IN CASES OF STILLBIRTHS

CASES	PRESENT
APH	21
IUGR	22
PREGNANCY INDUCED HYPERTENSION	28
POSTDATISM	07

In present study 18% had abruptio placenta and 3% cases had placenta praevia.

This study shows that there was 22% cases of stillbirths had presence of IUGR. IUGR is a contributing factor in causing stillbirths.

In this study only 7% cases of still birth had postdatism.this shows that postdatism is not significant cause of stillbirth.

Gardosiet al (1998) found that many stillborn babies were small for gestational age. In the absence of significant differences in physiological pregnancy characteristics this is unlikely to be constitutional smallness, but represents a preponderance of intrauterine growth restriction. Overall, 61 of the 149 stillbirths (41%) were small for gestational age, defined as $<10^{th}$ centile which is more likely to reflect a pathological association i.e intrauterine growth restriction.

STILLBIRTHS, PLACENTAL DYSFUNCTION AND ABRUPTION

In the UK, about one in 200 infants is stillborn, and rates of stillbirth have recently slightly increased. This recent rise might reflect increasing frequency of some important maternal risk factors for stillbirth, including nulliparity, advanced age, and obesity. Most stillbirths are related to placental dysfunction, which in many women is evident from the first half of pregnancy and is associated with fetal growth restriction. There is no effective screening test that has clearly shown a reduction in stillbirth rates in the general population. However, assessments of novel screening methods have generally failed to distinguish between effective identification of high-risk women and successful intervention for such women. Future research into stillbirth will probably focus on understanding the pathophysiology of impaired placentation to establish screening tests for stillbirth, and assessment of interventions to prevent stillbirth in women who screen positive. [32]

Study on hypertensive disorder in pregnancy and stillbirths 250 173 women and their 255 931 infants were included in the study. Overall, 24 517 women (9.8%) had a hypertensive disorder in pregnancy, including 1411 (0.6%) with chronic hypertension, 10 379 (4.2%) with pre-eclampsia, 731 (0.3%) with chronic hypertension with superimposed pre-eclampsia, and 10 864 (4.3%) with gestational hypertension. Women with, and infants exposed to, hypertension were more likely to suffer death or major morbidity than those without hypertension. Infants of mothers with hypertension were more likely to be too born preterm and small for gestational age. Just over half the women with major morbidity or mortality delivered in hospitals with a high level of medical care. In contrast, most infants with major morbidity or mortality were delivered in hospitals with neonatal intensive care units. Conclusions: Hypertension is a common complication of pregnancy, and adverse outcomes are increased among hypertensive women and their babies. Clinicians appear to be better at identifying and seeking an appropriate level of care for pregnancies where the infant is at risk of a poor outcome than when the mother is at risk.More specific antenatal indicators of poor maternal outcome would help guide the referral of MJA 2005.

Merialdi *et al* (2005) concluded that preterm delivery and hypertensive disorder were the most common obstetric events leading to perinatal deaths (28.7% and 23.6%, respectively). They also studied the preventive role of calcium supplementation in pre-eclampsia leading to stillbirths, and concluded that the calcium group had lower number of stillbirths as compared to placebo group.

Martinek *et al* (2006) founded the pre-eclampsia accounted for 5.6% of the stillbirths among a total of 106 cases in a retrospective study.

E Bergel, AJD Barros *et al* (2007) carried out a similar study in Canada.

Data from 10 randomized controlled trials were included in this review. Pooled analysis showed that calcium supplementation during pregnancy was associated with a significant reduction of 45% in risk of gestational hypertension [Relative risk (RR) 0.55; 95 % confidence interval (CI) 0.36-0.85] and 59% in the risk of pre-eclampsia [RR 0.41; 95 % CI 0.24-0.69] in developing countries. Calcium supplementation during pregnancy was also associated with a significant reduction in neonatal mortality [RR 0.70; 95 % CI 0.56-0.88] and risk of pre-term birth [RR 0.88, 95 % CI 0.78-0.99]. Recommendations for LiST for reduction in maternal mortality were based on risk reduction in gestational hypertensive related severe morbidity/mortality [RR 0.80; 95% CI 0.70-0.91] and that for neonatal mortality were based on risk reduction in all-cause neonatal mortality [RR 0.70; 95% CI 0.56-0.88]

Calcium supplementation during pregnancy is associated with a reduction in risk of gestational hypertension, pre-eclampsia neonatal mortality and pre-term birth in developing countries.

Gardosi *et al* (2005) found that of the 66.2% stillbirths who remained as unclassified according to the Wigglesworth system of classification, most stillbirths were growth restricted. So the largest category of stillbirths was A7 (IUGR) i.e 47%. Only 15.2% cases remained unclassified after applying ReCoDe system (Relevant condition at death). Hence, this system enabled 85% of the cases to be assigned relevant conditions, leaving only 15% as unexplained.

Forssas *et al* (1993) found that among smokers, more low birth weight infants suffer from intrauterine growth retardation rather than from prematurity. Low birth weight may be caused by a short gestation period, intrauterine growth retardation (IUGR) or a combination of both.

Martinek *et al* (2006) concluded that the principal cause was in utero growth retardation (19.8%).

Gardosi *et al* (2011) Intrauterine growth restriction: new concepts in antenatal surveillance, diagnosis, and management

Stillbirth and IUGR Such validation of the principles of the growth potential have allowed IUGR or FGR to be introduced as an additional category when classifying stillbirth and found that after excluding congenital anomalies, more than 50% of stillbirths had preceding IUGR (10th customized centile). As a result, the proportion of unexplained stillbirths drops from 65- 70% using the Wigglesworth classification to 15%. This has since been confirmed in an independent comparative study. While IUGR is usually the result of underlying placental pathology and not in itself the cause of the demise, it is a clinically relevant condition. Awareness of this strong link allows a

renewed focus of attention on the antenatal identification of IUGR as a first step toward prevention of stillbirths. Antenatal awareness that the fetus is not growing well is an essential quality indicator of maternity care. [36]

In a case control study by Dr. Kumbhare *et al* (2012), by applying chi square test (p=0.0157) hypertension was a significant factor in causing stillbirths. Similarly in the same study APH also was a significant factor in causing stillbirths at (P< 0.0001) when chi square test was applied.

TABLE 8: OLIGOHYDRAMNIOS IN CASES OF STILLBIRTHS

Oligohydramnios	Presence
Cases	**20**

In the present study 20% cases of stillbirths had associated oligohydramnios.

TABLE 9: CONGENITAL ANOMALY

Congenital anomaly	Presence
Cases	15

In the present study 15% had presence of congenital anomaly like spina bifida, anencephaly, hydrocephalus, morbid baby, amorphous conjoined twins. All these were fresh stillborn babies and all were female child. All the stillbirths with congenital anomaly were major defects not compatible with life.

In a study by Dr.Kumbhare *et al* (2012) congenital anomaly was present in all cases and not in any controls. Hence it was a very significant factor in causing stillbirths with p<0.001 using the fischers test.

Lawn *et al* (2009) suggested that congenital abnormalities are underestimated even in high income countries because only obvious external abnormalities are detected and important internal structural and metabolic disorders are missed.In the global data around 5-15% of stillbirths are attributed to a congenital cause.

Martinek *et al* (2006) concluded that foetal congenital and chromosomal anomalies accounted for 18.9% of stillbirths.

A study published in Phillppe De Wals et al (2011) to study the neural tube defects in the study population and its prevention with folic acid supplementation. A total of 2446 subjects with neural-tube defects were recorded among 1.9 million births. The prevalence of neural-tube defects decreased from 1.58 per 1000 births before fortification to 0.86 per 1000 births during the full-fortification period, a 46% reduction (95% confidence interval, 40 to 51). The magnitude of the decrease was proportional to the prefortification baseline rate in each province, and geographical differences almost disappeared after fortification began. The observed reduction in rate was greater for spina bifida (a decrease of 53%) than for anencephaly and encephalocele (decreases of 38% and 31%, respectively).

TABLE 10: TYPES OF STILLBIRTHS

CATEGORY	FRESH	MACERATED
CASES	60	40

In present study 60% were fresh and 40 % were macerated stillbirths. Of these 60 fresh stillbirths, all of these cases were referred from periphery and urban slums with with diagnosed as antepartum fetal death.

TABLE 11: CLASSIFICATION OF FRESH AND MACERATED STILLBIRTHS ACCORDING TO CAUSE

CASES	Number of fresh stillbirths	Number of macerated stillbirths
SEVERE PIH AND ECLAMPSIA	13	05
CONGENITAL ANOMALY	15	-
APH	14	07
HEV ENCEPHALOPATHY	03	04
CORD PROLAPSE	02	-
DIABETES MELLITUS	01 (GDM)	01 (dm type 1)
RUPTURE UTERUS	01	01(h/o fall down)
POSTDATE THICL MSL	01	05
DRUG MISUSE	01	-
TRUE KNOT	01	-
PLACENTAL INSUFFICIENCY IN HPE OF PLACENTA	01	02
2^{ND} TWIN	01	-
HEART DISEASE NYHA GRADE 3	01	-
LOOPS OF CORD AROUND NECK	-	03
CHORIOAMNIONITIS AND CMV INFECTION IN HPE OF PLACENTA	-	02
HYPERTHYROIDISM	-	01
NON IMMUNE FETAL HYDROPS	-	03
UNCLASSIFIED	04	06

In the present study the 18 cases of severe pre-eclampsia and eclampsia with diagnosed cases of intra uterine fetal death. Out of that 4 cases of eclampsia were cases of antepartum.

In congenital anomaly as discussed earlier the 15 cases were fresh stillborn with major anomalies most of them anencephaly, spina bifida and hydrocephalus which were incompatible with life.

In antepartum haemorrhage there were total 21 cases of which 14 had fresh stillbirths and 7 had macerated. 10 of these cases from periphery who were trial were refereed on deterioration of maternal condition for further management at our institution which on admission were diagnosed to be having APH with intrauterine fetal death. while rest of the patients were emergency admissions with irregular and infrequent antenatal visits and taking no medications for severe hypertension.

In the present study HEV hepatitis and encephalopathy caused 7 still births. Of these 6 patients were our admitted patients who did not take regular antenatal visits and seeked medical help very late in late phase of the disease. Of these 3 had nonimmune hydrops diagnosed in antepartum ultrasonography. There was a epidemic of HEV hepatitis in the year 2014.

Of the 2 cases of cord prolapse , 1 was referred as case of premature rupture of membranes since 6hours and fetal distress in 3^{rd} gravida. At the time of admission patient was in 1^{st} stage of labour with cord prolapsed in vagina with absent fetal cord pulsations. The 2^{nd} patient was primigravida with premature rupture of membranes since 3 hours and patient was from rural area directly from home as emergency patient. At the time of admission there was cord prolapse in vagina with absent cord pulsations.

In present study the patients with diabetes were 1 case with gestational diabetes mellitus was a emergency patient with absent fetal movement in 3^{rd} gravid at 32 weeks of gestation which turned out to be intra uterine fetal death. And other case was a 2^{nd} gravid with DM type-1 with uncontrolled diabetes with mixtard insulin at 35 weeks of gestation and was reffered as a case of intra uterine fetal death.

Two cases of rupture uterus ,1 was 3^{rd} gravida with prolong trial at resulting in rupture uterus and IUFD. And 2^{nd} patient was primigravida with h/o accidental fall at home leading to rupture uterus and intrauterine fetal death.

In a case of true knot in cord which had associated long cord of approximately 60 cm and polyhydramnios resulting in fresh stillborn.

In drug misuse patient was a 3^{rd} gravida with chronic hypertension taking since 2 years tab telmisartan.0, enalapril and tab furesemide. These drugs are from diuretics and angiotensin receptor blockers which are contraindicated in

pregnancy as they cause uteropacental insufficiency and teratogenicity. The patient was taking antenatal visits from a specialist from rural area though patient has been very irregular in antenatal visits only 2 visits till 34 weeks pregnancy. Same patient also had previous stillbirth due to hypertension.

In the present study with one case was primigravida with premature rupture of membranes with fever with intrauterine fetal death. HPE of placenta showed chorioamnonitis due to cytomegalovirus.

In twins pregnancy patient was referred late and lack of availability of transport patient had ambulance delivery of 1st twin and prolong interval between delivery of both twin the 2nd twin was a fresh stillborn.
The patient was a primigravida with hyperthyroidism with methimazole 15 mg TDS through out pregnancy had stillbirth due to uncontrolled thyrotoxicosis and placental insufficiency in HPE.

As evident from the association and stillbirth, most of the causes can be detected early by careful screening at antenatal clinic and institution of timely management during pregnancy and labour can reduce the development of stillbirth.

TABLE- 12 HISTO-PATHOGY OF PLACENTA ITS MEMBRANES AND UMBILICAL CORD

CASES	Gross appearance of placenta	Microscopic appearance
Placental abruption (18)	Placenta is small and thin and blackish clots seen in the placenta.	Membranes normal, cord normal with two arteries and veins. Areas of necrosis and haemorrhage seen, increased synctial knots, perivillous fibrin deposition. Intrachorionic and retroplacentalhaemorrhage seen suggestive of placental abruption.
Hypertensive disorder (22)	Placenta is small and thin and blackish clots seen in the placenta.	Membranes normal, cord with two arteries and veins which are thickened.perivascular fibrosis seen. Areas of necrosis and haemorrhageseen,ghost villi seen, increased synctial knots, perivillous and decidual fibrin deposition. Suggestive of maternal vascular disease and placental infarction.
HEV hepatitis (06)	Placenta is of normal size with membranes ,cord and placental tissue shows yellow tinge.umbulical cord is normal with 3 vessels.	Placental sections shows avascular villi and villi with empty vessels. Areas of decidualfibrosis ,perivillous fibrin deposition,necrosis, and obliterative end arteritis seen. Perivascular fibrosis seen. Findings suggestive of vascular disease due to hepatitis E.
Diabetes mellitus type 1 (01)	Placenta is large and edematous.	Hyperplacentosis seen with edematous villi with neutrophilic infiltration .cord shows single umbilical artery.
Antiphospholipid syndrome (01)	Small size placenta with greyish membrane over it. Visible calcifications seen in placental tissue.	Cord section shows two arteries and veins which are thrombosed. Avascular villi and fibrin deposition seen, areas of necrosis and infarction seen. Thrombosisin vessels is due to anti phospholipid antibodies.
Drug misuse (01)	Placenta is small and thin and blackish clots seen in the placenta.	Membranes normal, cord with two arteries and veins which are thickened.perivascular fibrosis seen. Areas of necrosis and haemorrhageseen,ghost villi seen, increased synctial knots, perivillous and decidual fibrin deposition.Suggestive of maternal vascular disease and placental infarction.
Chorioamnionitis (01)	Normal sized placenta seen with greyish film over membranes.	Neutrophils infiltrating maternal deciduas and chorionic plate suggestive of chorioamnionitis. Most commonly findings suggests cytomegalovirus infection.
Non immune hydrops (03)	Placenta is large and edematous.	Hyperplacentosis seen with edematous villi with neutrophilic infiltration seen. Increased avascular villi and obliterative endarteritis.

53 had positive histopathological examination.

Lawn *et al* (2009) concluded that distinction between macerated (antepartum) and fresh (intrapartum) stillbirths is important for stillbirth preventions strategies. Examination of fetal remains for signs of skin deterioration, skin or umbilical cord staining due to darkened amniotic fluid, or skull softening can determine whether fetus is macerated or fresh. Rates of fresh stillbirths are assumed to reflect the quality of intrapartum care, while rates of macerated stillbirths are assumed to reflect the quality of fetal growth and of care during the antenatal period.

TABLE 12: DISTRIBUTION OF CASES ACCORDING TO ReCoDe CLASSIFICATION IN PRESENT STUDY

CONDITION	NUMBER
GROUP A: FETUS	
CONGENITAL ANOMALY	15
IUGR	22
NON IMMUNE HYDROPS	06
FETOMATERNAL HAEMORRHAGE	-
GROUP B: UMBILICAL CORD	
CORD PROLAPSE	02
TRUE KNOT	01
LOOP OF CORD AROUND NECK	05
GROUP C:	
PLACENTAL ABRUPTION	18
PLACENTA PRAEVIA	03
PLACENTAL INSUFFICIENCY	11
GROUP D:	
POLYHYDRAMNIOS	06
OLIGOHYDRAMNIOS	20
GROUP E:UTERUS	
RUPTURED UTERUS	02
GROUP F: MOTHER	
HYPERTENSIVE DISORDER	28
HEV HEPATITIS	07
DRUG MISUSE	01
ANTI PHOSPHOLIPID SYNDROME	01
DIABETIS MELLITUS 1	01
CARDIAC DISEASE	01
GROUP G: INTRAPARTUM ASPHYXIA	21
GROUP H: TRAUMA	-
GROUP I: UNCLASSIFIED	10

As it is evident from above most of the stillbirths were falling into various groups of ReCode s classification suggesting multifactorial cause of death of fetus.

In present study most common maternal condition was hypertension (28),and antepartum hemorrhage(21) ,followed by Fetal condition was Fetal growth restriction (22)and congenital anomaly(15).

By the ReCoDe classification, the most common condition was fetal growth restrictions (43.0%), and only 15.2% of stillbirths remained unexplained. ReCoDe identified 57.7% of the Wigglesworth unexplained stillbirths as growth restricted. The size of the category for intrapartum asphyxia was reduced from 11.7% (Wigglesworth) to 3.4% (ReCoDe). Hence, the new ReCoDe classification system reduces the predominance of stillbirths currently categories as unexplained. This classification system seeks to identify the relevant conditions at the time of death in utero.

Gardosi *et al* (2005) published that by the conventional Wigglesworth classification, 66.25 of the stillbirths (1738 of 2625) were unexplained. The proportion of stillbirths that were unexplained was high regardless of whether a post-mortem examination had been carried out or not (67% and 65%; P=0.3).

Robalo *et al* (2013) examined the etiological factors contributing to late fetal death over a 10 year period through a retrospective cohort study. The cause of death was classified according to the ReCoDe system (Gardosi et al) similar to our study. Unexplained stillbirths contributed to 24.5% cases consistant with previous studies. The percentage contributions of other factors like fetal pathology (28.4%), placental factors (26.9%), and maternal conditions (21.2%), amniotic fluid disorders (10.6%) and umbilical cord events (9.6%) was drawn. Poor antenatal care, advances maternal age and gestational age had no statistically significant association with unexplained.

LIMITATION OF MY STUDY

- Due to small of small sample size facility based retrospective study. So the exact etiology could not be commented.

- Large population of community based on longitudinal and cross sectional studies with maternal age, sex of fetus, parity, and gestational age matched case control study are needed to know the exact cause of stillbirth in the given population.

CONCLUSION

Inmy study there were total 100 cases of stillbirths. These 100 cases of still births were then classified according to THE ReCoDe CLASSIFICATION which was selected according to the inclusion criteria over a period of 1 year from May 2014 to April 2015.

- The ReCoDe classification system of stillbirths could be able to classify 90% of my cases to relevant condition at death and only 10% remained as unclassified.
- There were total 3746 live births during study period in SIR T HOSPITAL Bhavnagar, obstetrics and gynaecology department. And total stillbirths were 150 in this study period..

- There were 28% cases with hypertensive disorders of pregnancy resulting in to stillbirths.
- There were 22% cases of stillbirth showing intra uterine growth restriction as an important factor in causing stillbirth in my cases.

- Among 100 cases 8% had past history of stillbirth This shows that recurrence of stillbirth is significant factor in causation of stillbirths.

- In this study 18% had placenta abruption, 3 % had placenta praevia and 11% other causes of placental insufficiency.

- 15% cases had major congenital anomaly like anencephaly, spina bifida , amorphous conjoined twin, hydrocephalus, etc

- Also drug misuse of telmisartan and tricyclic antidepressants resulted in placental insufficiency and IUGR and stillbirth in 1 case out of 100.

- HEV hepatitis and jaundice resulted in placental insufficiency and still births in 6% cases in this study.

- Anti phosholipid syndrome resulted in placental thrombosis and infarctions and IUFD in 1 case out of 100.

- Also cardiac disease (RHD WITH MS WITH MR WITH MITRAL VALVOTOMY) in mother was present in a case.

- 2% cases of still birth were due to rupture uterus and others like 2% cord prolapsed, 5% with loops of cord around neck and 1% true knot in cord, and 6% with non immune hydrops.

- Only 10% remained unclassified with no relevant condition on death.

- 53% cases of stillbirths were male fetus and 47% were female fetus.

- 60% of still births were fresh stillborn and 40% were macerated.

- In 53 cases histopathological report of placenta iits membranes and cord had positive correlation**.**

- As evident from the association and stillbirth, most of the causes can be detected early by careful screening at antenatal clinic and institution of timely management during pregnancy and labour can reduce the development of stillbirth.

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