Obesity has become “a silent epidemic, a major public health issue and is likely to remain so for the foreseeable future. It has become a worldwide phenomenon cutting across regional and economic barriers. It contributes to the development of several chronic diseases including type 2 diabetes mellitus, hypertension, coronary heart disease, and stroke. As per WHO, it is a “killer disease” at par with HIV and malnutrition. The rate of obesity in the general population is increasing drastically. The WHO estimated that in 2016, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 650 million were obese [1]. In developing countries like India, obesity often co-exists with under nutrition leading to double burden [2]. Body mass index (BMI) is the most commonly used parameter for measuring obesity at population level. WHO defines obesity as BMI > 30 for the world population [1]. However, this value could be misleading when comparing the Western countries to Asian Pakistani population. This is because of the difference in the phenotype and general body structure of the two diverse set of people in East and West. Pakistani people are obese at a lower BMI than specified for Western people. Recent studies have also shown that Asian Pakistani have more pre-disposition for truncal obesity and the risk of complications for Asians is
well below the cut-off values of BMI recommended by WHO, and thus for the Pakistani population, BMI>25 is defined as obesity [3]. Many studies have observed that maternal obesity can result in adverse outcomes for both women and fetuses like increase in the risk of miscarriage, gestational diabetes mellitus (GDM), gestational hypertension and preeclampsia [4]. It has also been associated with prolonged pregnancies, prolonged labor, two-fold increased risk for a caesarean delivery, increased incidence of post-natal infections with prolonged hospital stay [5, 6]. Obesity is often associated with a high risk of adverse neonatal outcomes including stillbirth, birth defects like neural tube defects, abdominal wall defects etc., neonatal intensive care admissions and perinatal mortality rates [7, 8]. Furthermore, long term studies demonstrate that having an obese mother increases the risk of child growing up to be obese themselves, thereby possibly inducing a transgenerational effect [9]. With alarmingly increasing prevalence of obesity in India, the need to determine its effect on maternal and fetal outcome is increasing. This study aimed to highlight the impact of maternal obesity on the outcome of singleton pregnancy in otherwise uncomplicated singleton women” in Pakistani population.

M E T H O D S

The study was conducted on 320 pregnant women who visited the hospital from October 2020 to September 2021. The participants enrolled were in their first trimester with viable singleton pregnancy. Women with pre-existing hypertension, diabetes, heart disease, thyroid disorders or any other chronic illness; bad obstetrics history or prior caesarean second were excluded from the study. They were categorized into two groups: obese group comprising of 160 women with BMI > 25 and non-obese group comprising of 160 women with BMI <25. They were followed up for feto-maternal outcome. Routine antenatal care was given as per hospital protocol. Maternal outcome variables included were antepartum complications (miscarriages, GDM, pre-eclampsia, eclampsia), onset of labor (spontaneous, induced), mode of delivery (vaginal, caesarean, instrumental) and postpartum complications (postpartum hemorrhage, wound sepsis, prolonged hospital stay). Perinatal outcome variables included were birth weight, intrauterine deaths (IUDs), stillbirth, macrosomia and NICU admissions. All results were analyzed statistically with the help of parametric and non-parametric tests, wherever applicable. A p-value of <0.05 was considered as statistically significant.

R E S U L T S

Table 1 shows the mean age was slightly higher in obese group (26.7 years) as compared to non-obese group (24.55 years), p-value 0.001. Obesity may cause reduced fertility that may be a probable cause of obese women being older than non-obese ones. Parity was similar in both the groups (p-value 0.223).

| Parameters            | Non-obese group | Obese group | p-value |
|-----------------------|-----------------|-------------|---------|
| BMI (Mean + SD)       | 22.4 +1.45      | 30.9+14.8   |         |
| Mean age              | 24.55 years     | 26.7 years  |         |
| Primary               | 35%             | 30%         |         |
| Multiparity           | 65%             | 70%         | 0.001   |
| Non Vegetarian        | 83.8%           | 67.5%       | 0.223   |
| Vegetarian            | 16.2%           | 12.5%       | 0.499   |
| Lipid Profile Deranged| 2.5%            | 10%         | 0.05    |

Table 2 show that there was increased incidence of antepartum complications in obese group as compared to non-obese group. The occurrence of gestational hypertension and preeclampsia was significantly more in the obese group (p-value <0.001). The proportion of miscarriage (p-value 0.246), APH (p-value 0.225) and gestational diabetes mellitus (p-value 0.426) were more in the obese group; though it was not statistically significant.

| Parameters                          | Non-obese group | Obese group | p-value |
|-------------------------------------|-----------------|-------------|---------|
| Spontaneous abortion                | 4(2.5%)         | 10(6.2%)    | 0.246   |
| Preeclampsia                        | 22(14.1%)       | 64(42.1%)   | <0.001  |
| GDM                                 | 18(10.3%)       | 22(14.5%)   | 0.426   |
| Intrauterine growth restriction (IUGR)| 10(6.4%)     | 8(5.3%)     | 0.777   |
| Antepartum hemorrhage (APH)         | 4(2.6%)         | 10(6.7%)    | 0.225   |

Table 3 show the difference in the onset of labor as well as mode of delivery, between the two groups was significant. Proportion of pregnant women having induced labor was more in the obese group as compared to non-obese group. The p-value was 0.007 making this correlation significant. Also, the rate of caesarean second was significantly higher in the obese group (37.5%) when compared to the non-obese group (13.8%), p-value < 0.001. The complication of shoulder dystocia was observed significantly more in obese group (18.4%) than in non-obese group (3.8%), p-value 0.004. Postpartum complications, like postpartum hemorrhage was more in obese group (16%) than in non-obese group (9%). But it was statistically not significant; p-value = 0.188. The wound sepsis was significantly higher in obese group (28%) than in non-obese group (9.5%), p-value 0.009. Thus, prolonging hospital stay in the obese group. It was found that the obese group had significantly longer duration of stay in the hospital (mean stay 3.34 + 2.04) than the non-obese group (2.44 +1.65), p-value was 0.002.
Non-obese group
Obese group

| Parameters                  | Non-obese group | Obese group | p-value |
|-----------------------------|-----------------|-------------|---------|
| Induction of Labour         | 20(13.3%)       | 44(31.9%)   | 0.007   |
| LSCSS                       | 22(15.8%)       | 60(43.5%)   | 0.006   |
| Wound sepsis                | 08(5.1%)        | 28(18.7%)   | 0.009   |
| Maternal injury             | 18(11.5%)       | 30(20.0%)   | 0.150   |
| Maternal injury             | 18(11.5%)       | 30(20.0%)   | 0.150   |
| Wound sepsis                | 08(5.1%)        | 28(18.7%)   | 0.009   |
| PPH                         | 14(9.0%)        | 24(16.0%)   | 0.188   |
| Mean hospital stay          | 2.44 + 1.65     | 3.34 + 2.04 | 0.002   |

Table 3: Intrapartum complications in both groups

Table 4 shows that in perinatal outcomes, the mean birth weight in the obese group (3.29 + 0.4603 Kg) was significantly more than in the non-obese group, (2.75 + 0.5960) (p<0.001). The proportion of macrosomia babies were observed significantly more in the obese group (p-value <0.001). Thirteen percent neonates, obese group required NICU admission compared to 6.6% neonates in non-obese group, the difference was not statistically significant (p-value 0.19). In obese group, there were three (4%) IUDs while in non-obese group, there were two (2.6%) IUDs. The proportion of IUDs was more in obese group than non-obese group; but p-value was 0.62 making this difference insignificant.

Table 4: Perinatal outcomes in both groups.

**DISCUSSION**

This study demonstrates that maternal obesity can result in adverse outcomes for both mother and fetuses like increase in the risk of miscarriage, gestational diabetes, gestational hypertension, preeclampsia, sudden IUD, macrosomia, shoulder dystocia, and higher cesarean rates. The rate of miscarriage was seemingly more in obese group (6.2% vs 2.5%) though not statistically significantly. Recent evidence indicate that obese women undergoing infertility treatment are at increased risk of spontaneous miscarriage [10]. However, this point is controversial. Roth et al conducted a study in 494 patients to ascertain whether BMI affects first-trimester pregnancy outcome in patients with ininfertility [11]. It is concluded that the likelihood of a spontaneous abortion in singleton gestations in the first trimester, after treatment for infertility, was not affected by BMI. In this study, the number of pregnant women developing gestational hypertension and preeclampsia remained significantly high in obese group (42.1%) as compared to non-obese group (14.1%), p-value <0.001. Similarly, Dasgupta et al., in his prospective cohort study found that the incidence of gestational hypertension and pre-eclampsia/eclampsia was significantly higher in obese (36.9%) compared to normal subjects (16.1%) [12]. Walsh et al., concluded both obesity and preeclampsia are associated with increased markers of inflammation such as C-reactive protein and inflammatory cytokines, tumor necrosis factor-α, interleukin-6, and interleukin-8 [13]. These findings suggest that obesity is a risk factor for pre-eclampsia because of pre-existing inflammation. This study does not show significant correlation between obesity and GDM, but still GDM cases were found more in obese category (14.5%) than non-obese (10.3%), p-value was 0.426. This may be because of smaller sample size and increased number of GDM complicated pregnancies in non-obese group than previous studies. Chu SY et al., in a meta-analyses estimated the risk of GDM in maternal obesity and their findings indicate that high maternal weight is associated with a substantially higher risk of GDM [14]. Obesity is considered to be an insulin resistant state, and thus accentuates the insulin resistance of normal pregnancy. Obese women with GDM are more likely to need insulin to achieve optimum glycemic control, as compared to women with normal BMIs, and the use of insulin in these pregnant women is also associated with better pregnancy outcome. In present study, although the proportion of pregnant women having genital infection remained high in the obese group but the p-value was 0.062 making this correlation insignificant. Sebire et al., also concluded from their study that genital tract infections are more common in obese compared to non-obese pregnant women [15]. Obesity is associated with higher incidence of induction of labor, as seen with many studies conducted earlier. Proportion of pregnant women having induced labor were more in the obese group (31.8%) as compared to non-obese group (13.3%), p-value was 0.007. The indication was mainly hypertension, post-datism and diabetes related complications. Robinson et al., also found increased rates of labor induction in obese group when compared to non-obese groups(32.1% in obese and 19.3% in non-obese) [16]. Similarly, Athukorala et al., in their study found that the overweight and obese women were more likely to be induced than women with a normal BMI (RR: 1.33 [95%CI 1.13, 1.57], p = 0.001 and RR 1.78 [95%CI 1.51, 2.09], p < 0.0001 respectively).17 Singh and colleagues in their study found that with increase in maternal BMI there was a dose dependent increase in number of women having induction of labour [18]. Results of study showed much higher rates of cesarean second in obese women as compared to non-obese (37.5% vs 13.8%). Although, the rates of operative vaginal delivery were lower in the obese groups. This is likely due to the higher cesarean delivery rates in the obese groups and the reluctance to perform operative...
vaginal deliveries in this population because of the increased risk of shoulder dystocia. Similarly, Robinson et al., also found that obese women had a higher rate of caesarean delivery, with the adjusted OR increasing with increased maternal weight (moderate obesity: adjusted OR 1.60, 95% CI 1.66–1.83; severe obesity: adjusted OR 2.46, 95% CI 2.11–2.85) [16]. Obese women were less likely to have operative vaginal deliveries. Also, in a population-based study conducted by Sheiner et al., the association between maternal obesity and caesarean section remained significant [19] In an observational study conducted by Barau et al., it was found that there is a linear association (X2 for linear trend, P < 0.001) between maternal corpulence and risk of caesarean deliveries, the leanest mothers having the best rate of vaginal delivery [20]. The obese women in our study are more than twice likely to deliver by caesarean due to various reasons like labor dystocia, macrosomia and poor myometrium contractility. In this study, the complication of shoulder dystocia was observed significantly more in obese group (18.4%) than in non-obese group (3.8%), p-value < 0.004. Similarly, Majouni et al., concluded in their retrospective study that maternal obesity (OR: 95% CI: 3.6: 2.1–6.3) was a predictive factor of shoulder dystocia [21]. Also, Usha Kiran et al., found that the women in obese group were four times more likely to have shoulder dystocia [22]. The macrosomia associated with obesity being the main contributing factor. It has also been suggested that obesity leads to an increase in maternal so issue inside the pelvis, which narrows the birth canal. Regarding maternal injuries, we did not find any significant association of high BMI with maternal injuries in our study. Dasgupta et al., also found no significant increase in perineal tears, C-section angle extensions and other maternal injuries among obese women in their study (p > 0.05) [12]. Similarly, Beyer et al., 2011 showed that there was no difference between the obese and non-obese group in the rate of injuries during delivery though foetal birth weight increased significantly with higher BMI [23]. The high rate of caesarean delivery may be a contributory factor in obese group as the vaginal delivery is avoided with the resultant less likelihood of injuries. Obesity was also associated with higher incidence of wound sepsis and episiotomy infection and had significantly longer duration of stay in the hospital. Hence, increasing BMI was associated with an increased risk of wound complications. Similarly, Athukorala et al., concluded in their study that obese women were more likely than women with a normal BMI to require antibiotics for a wound infection (RR 2.77 [95% CI 1.11, 6.96], p = 0.03) [17]. Fetal complications are also observed to be increased in obese pregnant women. The mean birthweight was significantly increased for the obese group compared to the non-obese group. Similarly, Mamun et al., 2011, [24] found that women who were obese prior to pregnancy and women who gained excess weight during pregnancy were at greater risk for higher birth weight difference. In this study, the proportion of macromomcic babies was observed more in the obese group (p-value < 0.001). Dasgupta et al., also found that there was a significant association between macromomcia and morbid obesity [12]. Sheiner et al., concluded that after having adjusted for diabetes mellitus, no significant association was found between macromomcia and obesity alone [19]. In this study, there was a significant association between macromomcia and obesity. Also many women who delivered macromomcic babies; had developed GDM. Incidences of perinatal mortality were relatively high in the obese group as compared to non-obese group, though statistically insignificant. Sebire et al., [15] found that maternal obesity was associated with a higher foetal death rate. In this study the cause of IUD in the non-obese group was PIH with foetal growth restriction in one case and the other was probably because of post-datism with meconium aspiration. In the obese group, the probable causes of IUDs were preeclampsia, GDM, deranged Doppler, etc. The cause of stillbirth was birth asphyxia in two cases and one case had unexplained aetiology. From this study, we can't conclude that obesity is an independent risk factor for IUD and stillbirth; but we can say that due to more emphasis of adequate antenatal checkups and routine investigations, the rates of IUD and stillbirth have declined compared to the previous studies.

CONCLUSIONS

Present study supports that obesity is associated with deleterious effect on feto-maternal outcome. In spite of the limitations of this study in terms of small sample size and short span of me it can be concluded that obesity is a risk factor for many antepartum, intrapartum, postpartum and fetal complications. In order to minimize the adverse effects of obesity on both mother and fetus, appropriate multidisciplinary management should be done.

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