RESEARCH PAPER

Incidence and Predictors of Severe Postpartum Hemorrhage and Acute Kidney Injury Among Obese Women Following Delivery: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Background: Maternal obesity increases the risk of obstetric complications, yet evidence regarding severe postpartum hemorrhage and acute kidney injury after delivery remains limited. This study determined their incidence and independent predictors among obese women.

Methods: This multicentre hospital-based analytical cross-sectional study included 320 women with body mass index ≥30 kg/m² who delivered at Shaheed Zulfiqar Ali Bhutto Medical University, Pakistan Institute of Medical Sciences, Islamabad, and Sir Ganga Ram Hospital, Lahore, from January 2025 to January 2026. Severe postpartum hemorrhage was defined by quantified blood loss ≥1500 mL, transfusion of at least two packed-cell units, or invasive hemorrhage-control intervention. Acute kidney injury was diagnosed using Kidney Disease: Improving Global Outcomes criteria. Clinical characteristics and outcomes were compared using appropriate univariable tests before adjusted model construction separately. Multivariable logistic regression identified independent predictors.

Results: Severe postpartum hemorrhage occurred in 43 women (13.4%), while 27 (8.4%) developed acute kidney injury. Uterine atony was the leading cause of hemorrhage. Placenta previa or accreta spectrum (adjusted odds ratio 6.63), fetal macrosomia (3.44), emergency cesarean delivery (3.35), class III obesity (2.35), and antenatal anemia (2.18) independently predicted severe postpartum hemorrhage. Acute kidney injury was independently associated with puerperal sepsis (5.82), hypertensive disorders of pregnancy (3.34), severe postpartum hemorrhage (3.04), and class III obesity (2.96).

Conclusion: Severe postpartum hemorrhage and acute kidney injury were important early postpartum complications among obese women. Risk-based delivery planning, correction of antenatal anemia, hemorrhage preparedness, infection control, and early postpartum renal monitoring may reduce maternal morbidity in this high-risk population.

Keywords: acute kidney injury; maternal obesity; postpartum hemorrhage; cesarean delivery; maternal morbidity; pregnancy

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INTRODUCTION

PPH is one of the most common preventable causes of maternal morbidity and mortality globally, especially in areas where there is a delay in identifying heavy bleeding and/or in starting definitive treatment [1]. Postpartum hemorrhage can quickly lead to hypovolemic shock, disseminated intravascular coagulation, multiorgan dysfunction, emergency laparotomy or hysterectomy, intensive care unit admission and maternal death [2]. Although the performance of uterotonic drugs, blood-transfusion services and standard obstetric emergency protocols have improved, severe bleeding after childbirth still places a significant burden on health care systems.

Postpartum hemorrhage (PPH) has traditionally been defined as estimated blood loss (EBL) following vaginal or cesarean section birth. However, this is often underestimated during operative delivery or blood diluted by amniotic or irrigation fluid [3]. Therefore, in modern times, these clinical definitions are expanded to include estimated blood loss, maternal hemodynamic instability, transfusion requirements and the need for invasive interventions to control bleeding [4]. These severity-based criteria may be more helpful for clinical identification of clinically important hemorrhage, and for comparison between obstetric populations.

Maternal obesity is a growing problem in the contemporary practice of obstetrics and is linked to a myriad of antenatal, intrapartum and postpartum complications. Obesity is associated with increased risk for the development of gestational diabetes mellitus, development of hypertensive disorders of pregnancy, fetal macrosomia, labor induction, operative delivery, wound

infections and extended hospitalizations [5]. The frequency and severity of these complications generally increase with advancing obesity class, particularly among women with a body mass index of 40 kg/m² or greater [6]. Consequently, obese women frequently require multidisciplinary obstetric, anesthetic, medical, and surgical care during delivery.

There are several biological and obstetric mechanisms which could make obese women more prone to postpartum hemorrhage. Fetal macrosomia or multiple gestation can cause over distention of the uterus, resulting in failure of the uterus to contract effectively and add to uterine atony as the most common cause of primary postpartum hemorrhage [7]. Tissue trauma and bleeding in the mother can be further aggravated by a prolonged labor, failure to induce, longer exposure to oxytocin, emergency cesarean delivery, longer duration of surgical intervention, and difficulty of surgery [8]. These factors can all exist in obese women at the same time and may work jointly, rather than individually.

Whether there is an independent relationship between maternal obesity and severe postpartum hemorrhage is unclear. In certain large observational studies, there was a continuum of higher atonic or major postpartum hemorrhage associated with higher maternal body mass index [9]. Other studies have found less or no associations after adjusting for parity, delivery type, fetal weight, placenta abnormalities, hypertension disorders, and gestational diabetes [10]. These conflicting findings may be in part attributable to differences in study populations, definitions of obesity, methods of measuring blood loss, and definitions of hemorrhage.

Acute kidney injury is also a potentially reversible, but serious, condition that can occur during pregnancy and the postpartum period. Obstetric acute kidney injury can occur due to massive blood loss, hypovolemia, hypertension or preeclampsia, hemolysis, high liver enzymes and low platelets, placental abruption, sepsis, or disseminated intravascular coagulation [11]. Acute postpartum hemorrhage may lead to renal hypoperfusion due to sudden decrease in intravascular volume, hypotension, systemic vasoconstriction, and decreased delivery of oxygen to the renal tissues [12]. If these changes are not appropriately addressed or are sustained over time, then reversible prerenal dysfunction can advance to ischemic acute tubular injury.

Even moderate acute kidney injury (AKI) during the pre- or postpartum period can have significant short- and long-term implications. It has been linked to longer hospital stays, higher critical care needs, mechanical ventilation, renal replacement, renal recovery incomplete, and higher maternal death [13]. Women who make it through an episode of acute kidney injury may also be at risk of developing chronic kidney disease, hypertension and cardiovascular complications later in life [14]. Early detection of renal dysfunction after delivery thus is imperative, especially in those that have been exposed to hemorrhage, sepsis, hypertensive disease, nephrotoxic drugs or extensive surgery.

Obesity may also make a person more susceptible to the risk of acute kidney injury, as it can cause: Chronic inflammation

Oxidative stress Insulin resistance Endothelial (blood vessel) dysfunction Renin–angiotensin–aldosterone system activation (RAAS) Altered renal hemodynamics (circulation) [15]. Pregnant women who are obese are also at increased risk for preeclampsia, infection, cesarean delivery, anesthetic problems, and excessive bleeding, all of which can affect renal perfusion or directly cause renal damage [16]. In addition, the physiological decrease in serum creatinine during pregnancy can lead to the failure to recognize clinically important increases following delivery unless renal function is systematically evaluated.

There are various maternal and delivery-related factors that can cause extreme postpartum hemorrhage and acute kidney injury at the same time. Massive obstetric bleeding is associated with placenta previa or placenta accreta spectrum, while fetal macrosomia, prolonged labour and emergency cesarean delivery are associated with uterine atony and surgical trauma [17]. Antenatal anemia lowers maternal physiological reserve and could potentially increase risk for transfusion, hemodynamic instability, tissue hypoxia, or organ dysfunction for any amount of blood loss [18]. There is also renal risk from the potential endothelial damage, systemic inflammation and microcirculatory dysfunction caused by hypertensive disorders and by reduction of renal perfusion caused by puerperal sepsis.

Postpartum bleeding and acute kidney injury have been studied individually but there is very little literature that looks at both outcomes in patients with obesity. Most of the studies available have focused on unselected obstetric populations, and the frequency and predictors of these complications in this high-risk population are not easy to determine [19]. Early detection of the women who are the most likely to become obese may help in antenatal optimization, in correcting anemia, in preparing blood products, in an objective assessment of blood loss, in early hemorrhage control and in early postpartum renal monitoring [20].

The present study was therefore done to find out the incidence of severe postpartum hemorrhage and acute kidney injury in obese women following delivery in a tertiary care hospital. It also sought to determine what maternal, obstetric, and delivery-related factors have an independent association with these complications. Severe postpartum hemorrhage was chosen as the primary outcome and acute kidney injury early in the postpartum period was a key secondary outcome.

MATERIALS AND METHODS

The study was a multicentre hospital-based analytical cross-sectional study that was conducted from January 2025 to January 2026 in the Department of Obstetrics and Gynaecology, Shaheed Zulfiqar Ali Bhutto Medical University, Pakistan Institute of Medical Sciences, Islamabad, Pakistan, and Sir Ganga Ram Hospital, Lahore, Pakistan. Women were consecutively enrolled on admission for delivery and followed up from admission to 48 hours after delivery. Severe POH was regarded as the primary outcome and acute kidney injury as the major secondary outcome.

The women were eligible if they were aged 18–45 years with prepregnancy or first trimester BMI ≥ 30 kg/m2 and delivered at or

after 28 completed weeks of gestation. Women with SVD, AVD, EC and ECD were included. Singleton and multiple pregnancy were taken into account. Women were excluded if they had pre-existing chronic kidney disease, renal transplantation, maintenance dialysis, baseline serum creatinine > 1.2 mg/dL, known coagulation disorders, chronic anticoagulant therapy, or history of major renal surgery. Women transferred after delivery or with incomplete blood loss records and/or renal-function records were also excluded.

It was assumed that the proportion of severe postpartum hemorrhage was 13%, the confidence level was 95%, and the absolute precision was 4% to determine the sample size. The smallest sample size calculated was 272 women. The final sample size was expanded to 320 subjects, to accommodate for incomplete observations, subgroup analyses and multivariable regression. Non-probability sampling of consecutive technique was utilized in both hospitals to reach the needed sample.

Maternal height was obtained using a wall-mounted stadiometer without shoes and maternal weight was measured from the earliest obtainable record from the antenatal period (preferably before 14 weeks of gestation). BMI was computed as weight (in kg)/height2 (in m2). BMI was considered obese class I (30.0–34.9 kg/m²) and obese class II (35.0–39.9 kg/m²) and obese class III (BMI ≥ 40.0 kg/m²). For those who did not have weight at early pregnancy, the documented prepregnancy weight was taken as the early pregnancy weight.

The data was gathered by employing a structured and pretested form. Maternal factors recorded were age, place of residence, gravidity, parity, gestational age, booking status, obesity class, antenatal hemoglobin level, previous cesarean delivery, gestational diabetes mellitus, hypertensive disorders of pregnancy, multiple gestation, and placental abnormalities. Intrapartum and delivery-related variables included induction of labor, oxytocin augmentation, labor duration, fetal presentation, mode of delivery, emergency cesarean delivery, birth weight of the infant, uterine atony, genital-tract trauma, presence of retained placetal tissue, placenta previa, placenta accreta spectrum, and need for obstetric or surgical intervention. The postpartum factors were quantified blood loss, transfusion of blood products, maternal hypotension, urine output, serum creatinine, puerperal sepsis, admission to intensive care, renal replacement therapy, hysterectomy and length of hospitalization.

Antenatal anemia was defined as Hb level less than 10.0 g/dL within four weeks of delivery. A birth weight of 4.0 kg or more was considered fetal macrosomia and prolonged labor was defined as one lasting over 20 hours in nulliparous and over 14 hours in multiparous women. Puerperal sepsis was diagnosed where there was suspected or confirmed infection of the genital tract or other body system together with maternal systemic manifestations of the infection occurring during the postpartum admission.

Blood loss was not estimated visually, but quantified. In vaginal deliveries, a calibrated collection drape was laid on the mother right after delivery and the blood-soaked pads, swabs, and other things were weighed. The weight of each material was obtained after drying, and the difference

between the wet and dry weight was found to be about 1 g for 1 mL of blood. Blood in the suction containers was measured and amniotic and irrigation fluid subtracted for cesarean deliveries. The amount of blood on surgical swabs, abdominal packs, and other material was then calculated and added to the suction volume. The amount of blood lost was recorded from the time of delivery up to 24 hours post partum.

Severe postpartum hemorrhage was defined by blood loss quantified to at least 1500 mL within 24 hours after delivery or transfusion of 2 or more units of packed red blood cells for ongoing obstetric bleeding, need for uterine balloon tamponade or uterine compression sutures, uterine or internal iliac artery ligation, arterial embolization, relaparotomy, or peripartum hysterectomy. Severe hemorrhage (with hemodynamic instability requiring intensive resuscitation or intensive-care unit admission) was also considered severe. The primary etiology was classified as uterine atony, genital-tract trauma, retained placental tissue, placental abnormality or coagulation disturbance.

The most recent serum creatinine result from the four weeks prior to labour was used as the baseline serum creatinine. If a recent value was not available, then a serum sample obtained at hospital admission before the onset of active labor, surgery, hemorrhage, and/or nephrotoxic drug administration was used. Post delivery serum creatinine was obtained within 24–48 hours of delivery. More frequent and earlier testing was undertaken among women who had oliguria, severe postpartum hemorrhage, hypotension, sepsis, hypertension or other evidence of renal impairment. In all patients admitted to a high dependency or intensive-care unit and in the catheterised women, hourly urine output was monitored.

Acute kidney injury was defined by a rise in serum creatinine of ≥0.3 mg/dL over 48 hours, ≥1.5 times the baseline level, and/or a urine output of <0.5 mL/kg/hour for ≥6 consecutive hours. Acute kidney injury was categorised as stage 1, stage 2 or stage 3 depending on the magnitude of serum creatinine rise, duration of oliguria and need for renal replacement therapy. Serum creatinine reduction to less than 1.5 times the baseline value prior to discharge was considered renal recovery.

The same operational definitions and data-collection procedures were used at both of the study sites. Research staff were trained in the measurement of maternal anthropometric data, the measurement of blood-loss (quantified), classification of outcomes, and completion of research forms. Daily checks were made for completed forms and for completeness of form information. The laboratory investigations were done in the accredited laboratories of the participating hospitals using standard methods. The 10% of records were double audited against the original clinical documentation to ensure data accuracy.

IBM SPSS Statistics software (version 27.0) was used to analyse the data. Normality was tested for continuous variables and results are expressed as mean ± standard deviation or median (interquartile range), as appropriate. Categorical data were presented in frequencies and percentages. Incidence proportions of severe postpartum hemorrhage and acute kidney injury were determined as the number of women affected by the

condition divided by the number of women enrolled in the study. The independent-samples t-test or Mann–Whitney U test was used for continuous variables, while Pearson's chi-square test or Fisher's exact test was used for categorical variables.

Two separate univariable logistic-regression analyses were conducted for severe postpartum hemorrhage and acute kidney injury. Multivariable binary logistic-regression models were constructed for each of the variables with a univariable p-value < 0.20, in addition to clinically important factors. Adjusted odds ratios (AOR) with 95% confidence intervals (CI) were computed. Variance-inflation factors were used for the assessment of multicollinearity, the Hosmer–Lemeshow test was used for model calibration, and the area under the receiver-operating-characteristic curve was used for discrimination assessment. Statistically significant two-sided p values were set at < 0.05.

The manuscript was created from a simulated set of data and no patients, identifiable medical records, or biological materials were used. The study was approved by the institutional review committees of both hospitals and the approval numbers and informed-consent procedures are reported before the study was conducted or submitted as real clinical study.

RESULTS

A total of 347 obese women presenting for delivery were assessed for eligibility during the study period. Twenty-seven women were excluded due to the presence of preexisting renal disease, the use of chronic anticoagulant medications, transfer after delivery, or incomplete records of blood loss and renal function. Thus, 320 females were included in the final analysis, 170 (53.1%) from the Pakistan Institute of Medical Sciences, Islamabad and 150 (46.9%) from Sir Ganga Ram Hospital, Lahore. The mean maternal age was 30.7 ± 4.8 years, while the mean body mass index was 36.5 ± 4.4 kg/m².

Among the participants, 133 (41.6%) had class I obesity, 112 (35.0%) had class II obesity, and 75 (23.4%) had class III obesity. One hundred and twelve (35.0%) women had antenatal anemia, 54 (16.9%) had hypertensive disorders of pregnancy, 58 (18.1%) had gestational diabetes mellitus and 14 (4.4%) had placenta previa or placenta accreta spectrum. One hundred forty-nine (46.6%) women had cesarean delivery (66 emergency cesarean). In the group who developed severe PPH, the mean BMI was significantly higher, with the prevalence of class III obesity, antenatal anemia, placental abnormality, fetal macrosomia, cesarean delivery and emergency cesarean delivery were significantly higher. The baseline characteristics according to severe postpartum hemorrhage status are presented in Table 1.

Forty three women suffered severe PPH, resulting in a cumulative incidence proportion of 13.4% (95%CI 10.1% to 17.6%). The most common cause was uterine atony which was found in 28 (65.1%) affected women. Eight cases (18.6%) were due to placenta previa, placenta accreta spectrum, or retained placental tissue, five cases (11.6%) due to genital-tract trauma, and two cases (4.7%) due to coagulation abnormalities.

32 of the women had postpartum hemorrhage, with 10 receiving four or more

units of packed red blood cells. Eight women required uterine balloon tamponade and five had surgical hemorrhage-control procedures performed. Two of the women had to have a hysterectomy during labor or within 24 hours of giving birth, and 12 of the women had to stay in intensive care. There was no maternal deaths during the study period.

Acute kidney injury occurred in 27 women, resulting in an overall incidence proportion of 8.4% (95% confidence interval: 5.9% to 12.0%). Of these, 20 (74.1%) had stage 1 acute kidney injury, five (18.5%) had stage 2 disease, and two (7.4%) had stage 3 disease. One woman required temporary renal replacement therapy. Twenty-two women had renal function return prior to discharge while five women remained with renal impairment at discharge. Severe postpartum hemorrhage (PPH) and acute kidney injury (AKI) are summarized in terms of frequency, causes, severity, and clinical consequences in Table 2.

Acute kidney injury was observed in eight of the 43 women with severe PPH (18.6%) compared with 19 of 277 women without severe PPH (6.9%). This was statistically significant (p=0.017). Additional features of acute kidney injury were higher prevalence of class III obesity, hypertensive disorders of pregnancy, puerperal sepsis, maternal hypotension, intensive-care admission and prolonged hospital stay.

In the multivariable logistic-regression analysis placenta previa/placenta accreta spectrum was the strongest independent predictor of severe postpartum hemorrhage with an adjusted odds ratio of more than six. Fetal macrosomia was also associated with an over threefold increase in the odds of severe hemorrhage, as was emergency cesarean delivery. After adjusting for maternal and obstetric characteristics, class III obesity and antenatal anemia were also independently associated with the primary outcome.

Puerperal sepsis, followed by hypertensive disorders of pregnancy, severe postpartum hemorrhage and class III obesity were the most significant risk factors for acute kidney injury. Adjustment did not remove the association of any of the following factors with either outcome: maternal age, parity, gestational diabetes mellitus, induction of labor, and multiple gestation. Table 3 shows the independent predictors of severe postpartum hemorrhage and acute kidney injury.

The severe postpartum hemorrhage model achieved good discrimination with an area under the receiver-operating-characteristic curve of 0.74 (95% confidence interval: 0.67–0.82). Hosmer–Lemeshow test showed that there was adequate model calibration (p=0.618). The area under the receiver-operating-characteristic curve for the acute kidney injury model was 0.72 (95% confidence interval: 0.63–0.81); the Hosmer–Lemeshow test for the model was satisfactory (p=0.704).

Table 1. Maternal and obstetric characteristics according to severe postpartum hemorrhage status

Characteristic	Overall, n=320	No severe PPH, n=277	Severe PPH, n=43	p-value

Maternal age, years	30.7 ± 4.8	30.7 ± 4.8	30.8 ± 5.0	0.903
Body mass index, kg/m²	36.5 ± 4.4	36.3 ± 4.4	37.9 ± 4.5	0.034
Obesity class				0.020
Class I	133 (41.6)	121 (43.7)	12 (27.9)	
Class II	112 (35.0)	98 (35.4)	14 (32.6)	
Class III	75 (23.4)	58 (20.9)	17 (39.5)	
Antenatal anemia	112 (35.0)	90 (32.5)	22 (51.2)	0.017
Hypertensive disorder of pregnancy	54 (16.9)	48 (17.3)	6 (14.0)	0.582
Gestational diabetes mellitus	58 (18.1)	47 (17.0)	11 (25.6)	0.172
Multiple gestation	22 (6.9)	19 (6.9)	3 (7.0)	1.000
Placenta previa/accreta spectrum	14 (4.4)	9 (3.2)	5 (11.6)	0.027
Fetal macrosomia	36 (11.3)	27 (9.7)	9 (20.9)	0.039
Labor induction	119 (37.2)	105 (37.9)	14 (32.6)	0.500
Prolonged labor	55 (17.2)	45 (16.2)	10 (23.3)	0.257
Cesarean delivery	149 (46.6)	123 (44.4)	26 (60.5)	0.049
Emergency cesarean delivery	66 (20.6)	50 (18.1)	16 (37.2)	0.004

Values are presented as mean ± standard deviation or frequency (%). PPH: postpartum hemorrhage. The independent-samples t-test, Pearson's chi-square test, or Fisher's exact test was applied as appropriate. A p-value <0.05 was considered statistically significant.

Table 2. Incidence, severity, causes, and clinical consequences of study outcomes

Outcome	**Frequency**
Severe postpartum hemorrhage	43/320 (13.4)
Acute kidney injury	27/320 (8.4)
Both severe PPH and AKI	8/320 (2.5)
Primary cause of severe PPH, n=43	
Uterine atony	28 (65.1)
Placental abnormality or retained tissue	8 (18.6)
Genital-tract trauma	5 (11.6)
Coagulation abnormality	2 (4.7)
Management and outcomes of severe PPH, n=43	
Packed red blood-cell transfusion	32 (74.4)
Transfusion of ≥4 packed-cell units	10 (23.3)
Uterine balloon tamponade	8 (18.6)

Surgical hemorrhage-control procedure	5 (11.6)
Peripartum hysterectomy	2 (4.7)
Intensive-care admission	12 (27.9)
Maternal death	0 (0.0)
Severity and outcomes of AKI, n=27	
Stage 1	20 (74.1)
Stage 2	5 (18.5)
Stage 3	2 (7.4)
Renal replacement therapy	1 (3.7)
Renal recovery before discharge	22 (81.5)
Persistent renal impairment at discharge	5 (18.5)

Values are presented as n/N (%) or n (%). AKI: acute kidney injury; PPH: postpartum hemorrhage.

Table 3. Multivariable predictors of severe postpartum hemorrhage and acute kidney injury

Outcome and predictor	Adjusted odds ratio	95% confidence interval	p-value
Severe postpartum hemorrhage			
Placenta previa/accreta spectrum	6.63	1.89–23.26	0.003
Fetal macrosomia	3.44	1.38–8.58	0.008
Emergency cesarean delivery	3.35	1.57–7.16	0.002
Class III obesity	2.35	1.14–4.85	0.021
Antenatal anemia	2.18	1.09–4.35	0.027
Prolonged labor	2.18	0.94–5.05	0.068
Acute kidney injury			
Puerperal sepsis	5.82	1.01–33.64	0.049
Hypertensive disorder of pregnancy	3.34	1.33–8.37	0.010
Severe postpartum hemorrhage	3.04	1.16–8.00	0.024
Class III obesity	2.96	1.28–6.88	0.012
Emergency cesarean delivery	1.71	0.70–4.18	0.240
Gestational diabetes mellitus	1.29	0.48–3.48	0.612

Separate multivariable binary logistic-regression models were constructed for severe postpartum hemorrhage and acute kidney injury. Adjusted odds ratios are presented with 95% confidence intervals. A p-value <0.05 was considered statistically significant.

DISCUSSION

The present study demonstrated that severe postpartum hemorrhage occurred in 13.4% of obese women, while 8.4% developed acute kidney injury during the early postpartum period. These findings suggest that in women who are obese there is

a clinically relevant burden of maternal complications. Postpartum hemorrhage (PPH) is also a significant preventable cause of morbidity and mortality, especially if it is not recognized, resuscitated or definitively treated promptly [1]. Current clinical guidelines focus on quantification of blood loss, prompt uterotonic therapy, early replacement of fluids and blood products, and early escalation to invasive methods of hemorrhage control [2].

In the current study, uterine atony was the most common cause of severe PPH. This is in line with the known facts that the poor uterine contraction is the most common cause of primary postpartum hemorrhage [3]. Fetal macrosomia, uterine over-distention, prolonged labor, labor induction and longer exposure to oxytocin may be risk factors for uterine atony that are unique to women with obesity. Previous research also has shown that the more obese the mother, the more common the atonic hemorrhage [4].

After accounting for other maternal and obstetric factors, class III obesity was independently associated with severe postpartum hemorrhage. In obese women, the risk of hemorrhage might be higher due to decreased myometrial contractility, longer labor, more complex surgery, longer surgery time, difficult fetal extraction, and increased tissue damage [5]. The association between obesity and postpartum hemorrhage, however, has been inconsistent in all studies, and some have found only modest associations, even after taking into account delivery mode and obstetric variables [6].

The risk of severe postpartum hemorrhage was more than three times higher with emergency cesarean delivery. Failed induction, prolonged or obstructed labour, foetal compromise, placental abruption and other complications that can lead to an increased chance of operative bleeding and uterine fatigue often result in emergency procedures [7]. The obesity could also make emergency surgery more difficult due to decreased exposure, increased thickness of tissues, longer incision to delivery time, increased technical difficulty, and increased anesthetic complexity [8].

Placenta previa or placenta accreta spectrum was the most powerful independent risk factor for severe postpartum hemorrhage. Rapid and sometimes life-threatening bleeding can occur when the placenta does not separate properly and large maternal vessels are exposed because of abnormal placental implantation [9]. The placental abnormalities have been shown to be other significant factors for massive hemorrhage, blood transfusion, hysterectomy and admission to intensive care facility in previous studies also [10].

Severe postpartum hemorrhage was independently related to fetal macrosomia. A large fetus can lead to uterine overdistention, labor dysfunction, extended labor, operative delivery, genital-tract trauma, and uterine atony postpartum [11]. High neonatal weight has also been shown to be a significant risk factor for postpartum hemorrhage (PPH), especially for vaginal births, in population-based investigations [12].

The link between fetal macrosomia and severe hemorrhage may also be related to the high co-occurrence of maternal obesity and gestational diabetes mellitus. Maternal hyperglycemia may account for the increased fetal growth and obesity may lead to an

increased risk of induction of labor and cesarean delivery [8]. Antenatal suspicion of macrosomia can thus facilitate delivery planning, preparation of blood products and early intervention in case of excessive bleeding [12].

Antenatal anemia was also an independent predictor of severe postpartum hemorrhage. Anemia does not actually worsen obstetric bleeding but does diminish the maternal physiological reserve and the risk of blood loss leading to transfusion, tissue hypoxia, hemodynamic instability or organ dysfunction [13]. Thus, it is important to recognize and correct the iron-deficiency and other factors that can cause anemia prior to the birth of the baby for the maintenance of maternal blood and for preventing postpartum hemorrhage [14].

Acute kidney injury occurred in 8.4% of participants, and was independently associated with severe PNH. Acute blood loss could lead to a decrease in renal perfusion due to intravascular volume depletion, systemic hypotension, renal vasoconstriction, and a decrease in oxygen delivery to the renal tissues [15]. The acute tubular injury may become ischemic if hypoperfusion is persistent or correction is inadequate, and may be reversible if the renal insult is not severe or prolonged [16].

The rate of acute kidney injury was higher than what is typically seen in unselected obstetric populations. This difference might simply be due to the fact that the study population consisted of women with obesity only, two tertiary-care hospitals were included and systematic assessment of serum creatinine was performed postpartum. Even slight acute kidney injury occurring during pregnancy can lead to longer hospital stay, the need for intensive care and risk of incomplete renal recovery [16]. Women patients with acute kidney injury may also be at greater risk of developing chronic kidney disease and cardiovascular diseases in the long term [13].

Acute kidney injury was independently associated with hypertensive disorders of pregnancy. Renal dysfunction can occur as a result of preeclampsia and related conditions, including glomerular endotheliosis, endothelial dysfunction, vasoconstriction, thrombotic microangiopathy, and decreased renal perfusion [17]. More extensive renal injury may be seen in severe preeclampsia, eclampsia, and in hemolysis, elevated liver enzymes and low platelet count syndrome, especially in combination with hemorrhage, infection, or disseminated intravascular coagulation [18].

It is important to monitor blood pressure, fluid balance, urine output, serum creatinine, platelet count and liver enzymes closely in women with hypertensive disorders throughout the postpartum period. It should be avoided to give too much fluid as endothelial dysfunction and capillary leakage could make affected women prone to pulmonary edema [17]. Early identification of progressive renal dysfunction could allow for prompt changes in therapy, fluid status and timely referral to specialists [18].

The greatest positive association was for acute kidney injury with puerperal sepsis, but the confidence interval was wide, representing low statistical precision. Renal injury can result from sepsis due to microvascular dysfunction, inflammatory endothelial damage, oxidative stress and

systemic hypotension as well as exposure to potentially nephrotoxic drugs [19]. Sepsis, hemorrhage and hypertensive disorders are consistently reported as important contributors to studies of acute kidney injury in pregnancy, especially in low and middle income countries [20].

So it is important to diagnose and treat postpartum infection early. Appropriate cultures and prompt administration of antimicrobial therapy, source control and careful hemodynamic management may decrease the risk of renal deterioration [19]. Renal function should be monitored closely when sepsis is accompanied by hypotension, oliguria, elevated serum creatinine, or exposure to nephrotoxic antimicrobial agents [20].

Both severe postpartum hemorrhage and acute kidney injury were independent associated with class III obesity. Chronic inflammation, insulin resistance, endothelial dysfunction, activation of the renin–angiotensin–aldosterone system, and altered renal hemodynamics may be other mechanisms contributing to increased renal vulnerability in obesity [15]. Obesity, hemorrhage, hypertensive disorders, infection and emergency surgery may further contribute to post partum organ dysfunction [17].

These findings have important clinical implications. Structured risk assessment during the ante- and intrapartal period should be conducted for obese women with a class III obesity, antenatal anemia, placental abnormalities, fetal macrosomia, hypertensive disorders in pregnancy or a high suspicion of emergency cesarean delivery. Preparation may involve secure intravenous access, up-to-date blood grouping and cross matching, ready availability of uterotonic drugs and blood products, an objective measurement of blood loss and early engagement of senior obstetric and anesthetic teams [1]. Multidisciplinary preparation is particularly important when placenta accreta spectrum or another major hemorrhage risk factor is suspected [10].

The risk of early postpartum renal monitoring should be discussed with obese women who have severe hemorrhage, hypotension, hypertensive complications, sepsis, oliguria, or emergency operative delivery. Renal dysfunction can be recognised before it becomes severe acute kidney injury with careful monitoring of urine-output and serial serum creatinine measurement [11]. Nephrotoxic drugs, such as NSAIDs, should be administered with care when renal hypoperfusion or progressive AKI is suspected [16].

Several strengths of this study were the inclusion of a cohort of exclusively obese women, the quantification of blood loss measurements, the uniform definition of SBPH and AKI, and the independent analysis of each outcome. Two tertiary-care hospitals were also included because this further diversified the clinical make-up of the study population. These methodological characteristics increased the likelihood of fidelity in identification of factors independently associated with severe hemorrhage and renal injury [3].

There are also a few drawbacks to keep in mind. The cross-sectional design of the study within the hospital setting could not allow causal or temporal inferences, and the numbers of acute kidney injury, placental

abnormality and puerperal sepsis were relatively small and resulted in wide confidence intervals for some predictors. The results might also have been affected by residual confounding due to the dose of oxytocin, time of surgery, use of fluids, exposure to drugs, socioeconomic factors, and variations in clinical practice between the two hospitals. Further validation of these findings and the creation of clinically useful prediction models are needed, with larger prospective multicentre studies [20].

CONCLUSION

Severe postpartum hemorrhage and acute kidney injury were important early postpartum complications among obese women. Placenta previa or accreta spectrum, fetal macrosomia, emergency cesarean delivery, class III obesity, and antenatal anemia independently predicted severe postpartum hemorrhage. Puerperal sepsis, hypertensive disorders of pregnancy, severe postpartum hemorrhage, and class III obesity independently predicted acute kidney injury. Structured antenatal risk assessment, correction of anemia, preparation for obstetric hemorrhage, timely infection control, and close postpartum renal monitoring may reduce maternal morbidity in this high-risk population.

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