Ethnic disparities in pregnancy-related acute kidney injury in a United Kingdom population

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Abstract

The incidence of acute kidney injury in pregnancy (P-AKI) is rising and is associated with detrimental maternal and foetal outcomes. Ethnic disparities in pregnancy outcomes are well recognized with females who identify as Black or Asian are more likely to die during pregnancy compared to females who identify as White ethnicity. This study report rates of P-AKI and associated risk factors in pregnant females of different ethnicities.

All pregnancies were recorded between 2016-2020. AKI episodes were identified using electronic alerts. Ethnicity, AKI stage (1-3), obstetric outcomes and risk factors for P-AKI (chronic hypertension, pregnancy-induced hypertension and pre-eclampsia and haemorrhage) were assessed.

There were 649 P-AKI episodes from 16,943 deliveries (3.8%). Black females were more likely to have P-AKI (5.72%) compared to those who were White (3.12%), Asian (3.74%), mixed ethnicity (2.89%) and other/not stated (3.10%). Black females, compared to White females, were at greater risk of developing P-AKI if they had haemorrhage requiring blood transfusion (OR 2.44, 95% CI 1.31,4.54; $p<0.001$) or pregnancy-induced hypertension (OR 1.79, 95% CI 1.12, 2.86; $p<0.001$). After adjusting for risk factors, Black females had increased risk of developing P-AKI (OR 1.52, 95% CI 1.22, 1.80; P<0.001), compared to White females.

Black females were at increased risk of developing P-AKI compared to White females. Mode of delivery, pregnancy-induced hypertension and haemorrhage are likely to contribute. The increased risk persists despite accounting for these variables, suggesting other factors such as socioeconomic disparities need to be considered.

Introduction

Acute kidney injury in pregnancy (P-AKI) is associated with maternal and fetal morbidity and mortality.[1] The reported incidence of P-AKI varies widely from 0.02% to 7.3%.[2–4] This variability is multifactorial, due to differences in healthcare systems in low- and high-income countries and the lack of a uniform definition for AKI in pregnancy. This disparity in incidence between low and high-income countries, reflects the difference in burden on AKI in pregnancy, which is highlighted by the World Health Organisation's maternal mortality ratio of 415/100,000 women in *least developed countries* compared to 12/100,000 in Europe and North America.[5]

Typically, a bimodal distribution of P-AKI is seen in the first and third trimester. First trimester causes are usually related to hyperemesis gravidarum or septic abortion. The latter remains more prevalent in low- and middle-income countries, particularly where terminations are illegal, as unsafe procedures are more likely to be complicated by sepsis or significant haemorrhage, highlighting the potential impact of removing legalization of abortions and antenatal care.[6, 7] Furthermore, a termination in early pregnancy may be the only treatment option for women with severe AKI secondary to irreversible causes without delivery (e.g. pre-eclampsia or atypical haemolytic uraemic syndrome). Prevention of this opportunity to maintain maternal health and reduce the impact of AKI is likely to have long term effects on renal outcomes.

P-AKI in the third trimester is associated with obstetric complicatons, such as pre-eclampsia, haemorrhage and caesarean section,[8, 9] and results in a higher incidence of decreased mean gestational age, premature delivery, stillbirth and lower baby birthweight.[4] Although the overall prevalence of P-AKI remains low, the incidence is increasing, demonstrated by a study in USA showing a rise from 2.4 to 6.3 cases per 10,000 deliveries over a 10-year period.[2, 10] This may be attributed to increasing recognition, advanced maternal age, increasing medical comorbidities and obstetric risk factors including pregnancy-induced hypertension or pre-eclampsia.[10] Although most females fully recover, there is increased risk of future pregnancy complications, chronic kidney

disease (CKD) and cardiovascular disease in later life,[8] consistent with non-pregnant populations.[11]

Outside pregnancy, acute kidney injury (AKI) is defined using Kidney Disease: Improving Global Outcomes (KDIGO) criteria, which classifies AKI into 3 stages based on the rise in serum creatinine from baseline or change in urine output.[12] However, identification of P-AKI is challenging as these criteria have not been validated in pregnancy and gestational physiological changes, including increased glomerular filtration, renal blood flow and plasma volume expansion affect serum creatinine concentrations.[13]

Ethnic disparities in healthcare are well recognised. In non-pregnant populations, patients of Black ethnicity are more likely to develop AKI than white patients.[14] In pregnancy, females of Black ethnicity are at greater risk of developing hypertension or pre-eclampsia, delivering via caesarean section, delivering preterm and having smaller babies than white females.[15, 16] In the UK, compared to white females, Black and Asian females are four times as likely to die during pregnancy and twice as likely during childbirth.[17–19]

A recent observational study in the USA reported that hospitalized Black females were twice as likely to develop P-AKI compared to white females with increased risk of miscarriage and mortality.[20] However, ethnicity is a social construct, based on cultural, religious and linguistic factors rather than a biological construct. Consequently, heterogeneity exists within single categories such as “Black”, and therefore these results may not be applicable to populations outside of the USA. Therefore, the aim of this study was to determine any associations between the ethnicity of pregnant females delivering in the UK and the rates of P-AKI and associated risk factors.

Methods

We conducted a study in a UK tertiary hospital including all females receiving obstetric care aged between 18 – 55 years old, between November 2016 and October 2020. The study did not require research ethical approval and was reviewed and registered at King's College Hospital NHS Foundation Trust Nephrology Audit Register 2020. The cohort includes 288 pregnancies complicated by P-AKI and 576 control pregnancies which have also been described elsewhere.[9]

Data were collected from renal and obstetric electronic medical records: Sunrise Electronic Patient Records (EPR) and Badgernet respectively. Demographic data included maternal age at time of delivery (years), first recorded body mass index (BMI, kg/m^2) and self-reported ethnicity categorized as: Black, White, Asian, Mixed and Other/Not Stated. Obstetric data included date of delivery, mode of delivery (caesarean section, vaginal or instrumental delivery), estimated blood loss (mL) and receipt of packed red blood cells transfusion. Haemorrhage was defined as an estimated blood loss ≥ 1000mL. Birthweight (grams) and median gestation at delivery (days) were collected for singleton deliveries. Females with hypertensive disorders (pre-existing hypertension, pregnancy-induced hypertension and pre-eclampsia) were identified using clinical coding from a specialist obstetric hypertensive unit which reviews and records data for all pregnant females with hypertension.

Serum creatinine was measured using Jaffe method until replaced by the enzymatic method in September 2019, on the Siemens clinical chemistry analysers (Advia 2400, Siemens Diagnostics, Frimley, UK) in a UK Accreditation Service accredited laboratory. AKI was defined according to change in serum creatinine. AKI was identified via automated electronic alert (e-alert) for AKI stages 1 - 3, using the NHS England algorithm based on KDIGO guidelines. Therefore at least 2 serum creatinine levels were required to trigger an electronic alert. AKI stage 1 is defined as a 1.5-fold rise from baseline or increase in serum creatinine of greater than 26 μmol/L; AKI stage 2 is a 2-fold increase and AKI stage 3 is a 3-fold increase or a serum creatinine greater than 353 μmol/L. AKI e-alerts were triggered through the laboratory information management system (LIMS;

WinPath/Clinisys) and identified on Sunrise EPR. Females with underlying CKD were not excluded. Females with AKI e-alerts occurring greater than 3 months post-partum, with kidney failure and those who were not under the obstetric team were excluded.

Categorical data are presented as counts with percentages, parametric data as mean ± standard deviation and non-parametric data as median with interquartile ranges. Statistical significance (p<0.05) was assessed using chi-squared test for categorical data and Student's t-test or Mann Whitney U for parametric and non-parametric data, respectively. Analysis of variance (ANOVA) was performed for comparison between multiple groups.

Unadjusted odds ratios with 95% confidence intervals were calculated for each variable, using White ethnicity as the reference group. A multivariate logistic regression model was performed, using White ethnicity as the reference group, initially adjusting for age and chronic hypertension. Full adjustment also included BMI, mode of delivery, haemorrhage, pre-eclampsia and pregnancy-induced hypertension. The regression analysis was repeated for females with AKI stages 2 and 3 combined.

Results

Baseline Characteristics

In the four-year study period, there were 16,943 deliveries. Of these, 8,311 (49.1%) females were White, 4,493 (26.5%) were Black, 988 (5.8%) were Asian, 761 (4.5%) were Mixed ethnicity and 2,390 (14.1%) were 'other' ethnicity or not stated. Maternal age at delivery was 32.7 ± 5.5 years and BMI was 24.7 kg/m^2 (IQR 22.0, 28.6 kg/m^2). Females of Black ethnicity had a higher median BMI (28.1 kg/m^2) compared to other ethnicities (White 23.6 kg/m^2, Asian 24.5 kg/m^2, Mixed 24.3 kg/m^2 and Other/Not stated 24.5 kg/m^2). Baseline characteristics are summarized in Table 1.

Renal Outcomes

There were 649 (3.8%) AKI electronic alerts. Of these 500 (77.0%) were stage 1, 117 (18.0%) were stage 2 and 32 (4.9%) were stage 3. The incidence of Stage 2 and 3 AKI was 0.88%. Although not statistically significant, the incidence of P-AKI increased yearly from 3.6% in 2016-17 to 4.6% in 2019-20 (Figure 1). P-AKI was most common in females identifying as Black ethnicity (5.72%) compared to females identifying as Asian (3.74%), White (3.12%), Other or not stated (3.10%) and Mixed ethnicity (2.89%). Incidence of P-AKI for each stage is described in Table 2.

There were 13 (2.0%) females with underlying CKD; 7 females identified as White ethnicity and 6 as Black ethnicity. The majority had AKI stage 1 (6 identified as Black ethnicity and 4 as White ethnicity), 2 had AKI stage 2 (both identified as White ethnicity) and 1 had AKI stage 3 (White ethnicity).

Obstetric Outcomes

Obstetric outcomes are summaries in Table 3. P-AKI occurred more frequently among females who delivered via cesarean section (5.04%), compared to females who delivered vaginally (3.37%) or by instrumental delivery (2.11%). P-AKI was more common with females who had stillbirth or miscarriage (8.45%). Compared to females who identified as White, females who identified as Black were more likely to develop P-AKI with all modes of delivery. The risk of P-AKI was greatest with instrumental (OR 4.19; 95% CI 2.23,6.98), followed by caesarean section (OR 1.91; 95% CI 1.45, 2.52).

There were 1723 females with haemorrhage, of which 129 (7.4%) had P-AKI. Haemorrhage was more common in females with P-AKI compared to those without P-AKI (P<0.001). Females identifying as Black (OR 1.31; CI 0.08,1.97) or Mixed ethnicity (OR 1.21, CI 0.47, 3.13) tended to be more likely to have haemorrhage and P-AKI compared females identifying as White, although this was not statistically significant. P-AKI occurred in 66 (15.6%) females who required a blood transfusion (N=423); 27/112 (24.1%) of females identifying as Black ethnicity requiring blood transfusion developed P-AKI compared to 22/191 (11.5%) females identifying as White (p<0.001).

Hypertension in Pregnancy

Hypertensive disorder outcomes are summarised in Table 3. There were 1104 (6.52%) females with hypertensive disorders; 221 (1.3%) had chronic hypertension, 436 (2.6%) had pre-eclampsia and 447 (2.6%) had pregnancy-induced hypertension. Females with chronic hypertension were more likely to identify as Black (N=120, 54.3%) than other ethnicities (White = 61 (27.6%); Asian= 17 (7.7%); Mixed=3 (1.4%), Other/Not stated = 20 (9.0%)). However, P-AKI with underlying chronic hypertension occurred most frequently in those identifying as White ethnicity (10/61; 16.4%; P<0.001).

Females identifying as Black ethnicity were twice as likely to develop pre-eclampsia, compared to females identifying as White ethnicity (4.25% versus 2.19% respectively). P-AKI was present in approximately one-quarter of females with pre-eclampsia (160/436; 24.7%). However, the frequency of P-AKI in females with pre-eclampsia was similar between each group.

One in four females (114/447; 25.5%) with pregnancy-induced hypertension developed P-AKI. Females identifying as Black ethnicity were 1.79 times (CI 1.12-2.86) more likely to develop P-AKI than females identifying as White ethnicity.

Neonatal Outcomes

Neonatal outcomes were analysed for singleton pregnancies and are summarised in table 4. Females with P-AKI compared to those without P-AKI, delivered significantly earlier (275 vs 279 days; P<0.001) and had smaller babies (3258g vs. 3370g; P<0.001). For females with P-AKI, median birthweight was lowest in females identifying as Asian (2,915g; IQR 2,464, 3,510g).

Regression Analysis

The unadjusted odds of developing P-AKI were substantially higher in females identifying as Black at 1.89 (95% CI 1.58-2.25, p<0.001) compared to those identifying as White. There was no significant

difference in risk between females identifying as White ethnicity and Asian (OR 1.21; CI 0.85, 1.72), Mixed (OR 0.93; CI 0.60, 1.44) or Other/Not stated (OR 1.76; CI 1.29) groups, respectively.

After partially adjusting for age, BMI and chronic hypertension (N=12,898), females identifying as Black ethnicity were 1.61 times more likely to develop AKI compared to females identifying as White ethnicity (CI 1.31-1.99; $p<0.001$). After adjusting for chronic hypertension, pre-eclampsia, pregnancy-induced hypertension, haemorrhage and mode of delivery, females identifying as Black ethnicity remained an independent risk factor for developing AKI (OR 1.52, CI 1.22-1.80; $p<0.001$).

Repeating the analysis for females with AKI stages 2 and 3 only (N=149) versus those without AKI (N=16,294) demonstrated an increased risk of P-AKI in females identifying Black ethnicity (OR 1.47; CI 1.02-2.12; $p<0.05$). After fully adjusting, females identifying as Black had a 1.29-fold increased risk (CI 0.83-2.02; $p>0.05$). Regression analyses are summarized in Table 5.

Discussion

To our knowledge this is the first study exploring differences between ethnicities in P-AKI using electronic alerts. Major findings include the significantly increased risk of P-AKI in females identifying as Black ethnicity compared to White ethnicity and the high incidence of P-AKI (one in 26 females).

Ethnic disparities in P-AKI and associated risk factors

Females identifying as Black ethnicity had a significantly increased risk of developing P-AKI compared to females identifying as White ethnicity, even after adjustment for some confounding factors for P-AKI (age, BMI, hypertensive disorders, caesarean section and haemorrhage). Although this is the first study in the UK to demonstrate this disparity, similar findings have been reported in the USA. Two large scale American studies using the International Classification of Diseases (ICD)

coding for identification of P-AKI, reported an increased P-AKI risk in African Americans ranging from 15% - 52%.[20, 21]

When excluding those with AKI stage 1, there remained an increased risk, albeit smaller, for females who identify as Black ethnicity. This only remained significant with partial adjustment for confounders. However, this model may be limited by the small case numbers with a large number of confounding variables.

Factors that contribute to increased P-AKI risk in females identifying as Black are likely to be complex and multifaceted including comorbidity, socioeconomic status, environmental, engagement with health care services and for some individuals an increased risk of culpable genetic variants.

Pre-existing conditions and complications of pregnancy, such as chronic hypertension and pregnancy induced hypertension are known to be more prevalent in females who identify as Black compared to White ethnicity,[22] and are both associated with an increased risk of AKI.[3, 23] However, we found only pregnancy-induced hypertension, not chronic hypertension, carried a substantially greater risk of P-AKI in females identifying as Black compared White ethnicity.

Females identifying as Black ethnicity, compared to White ethnicity, had a higher risk of developing P-AKI with both instrumental delivery and cesarean section. Prior studies have highlighted higher rates of cesarean sections in females identifying as Black ethnicity in the USA adjusting for comorbidity and common indications for cesarean section.[24, 25] Therefore, the higher rates of caesarean section may contribute to the increased rates of P-AKI.

Females identifying as Black ethnicity were delivered earlier with smaller babies, which is consistent with other reports.[26, 27] Low birthweight, coupled with epigenetic changes in adulthood, is associated with increased risk of developing hypertension, diabetes mellitus and CKD.[28–30] Low birthweight babies also have a reduced nephron number with an inversely proportional relationship

between glomeruli number and glomeruli volume.[31, 32] This correlation, suggests that the reduced glomeruli undergo compensatory hyperfiltration and hypertrophy, which reduces the renal functional reserve.[33] Typically following an insult, such as a hypovolaemic state, minimal changes will be seen in GFR (and therefore serum creatinine) until after more than 50% nephron involvement.[31] Therefore, females with reduced nephron number (i.e. reduced reserve), would be at greater risk of developing AKI as they would reach this threshold sooner. Consequently, factors which contribute to delivering smaller babies for females identifying as Black ethnicity, such as obstetric complications or socioeconomic inequalities, may have implications on the risk of development of long-term health conditions as well as kidney injury development in future generations.

Socioeconomic deprivation has a negative impact on healthcare and disproportionately affects minority groups in European and North American nations. The detrimental healthcare outcomes continue, despite factoring in reduced access to healthcare (for example due to lack of insurance). Even in the UK and Netherlands, where healthcare services are free at the point of access, CKD outcomes remain worse for people identifying as Black ethnicity.[34, 35] In non-pregnant populations, increased AKI incidence in African Americans has also been reported,[36] although this difference was not significant after accounting for socioeconomic status.[36] In the UK, lower socioeconomic status is independently associated with increased risk of AKI,[37] as well as other comorbidities including CKD.[38] It also likely that decreased health literacy, decreased trust in public healthcare systems, language barriers and differing cultural and spiritual beliefs all affect engagement with healthcare services,[39, 40] which may further contribute to the differences in risk in P-AKI between ethnicities in the UK.

Genetic variants of Apolipoprotein 1 (APOL1) are now widely recognised to be associated with renal pathology in people of recent African ancestry including HIV-associated nephropathy, progressive CKD with hypertension and AKI.[41, 42] More recently, the increased incidence of pre-eclampsia was associated with risk variants of APOL1 in the infant and mother.[43, 44] However, the prevalence of APOL1 variants varies substantially across different parts of the world and within regions of higher

reported prevalence such as West Africa.[45, 46] Therefore, the potential role of APOL1 high risk variants and other genetic contributions with AKI to P-AKI warrants further exploration. However genetic factors assessed in isolation will not explain the complex multifaceted association between ethnicity and P-AKI, and have potential to propagate racist practice in medicine and science.

High reported incidence of AKI in pregnancy

The incidence of AKI (3.8%) is substantially higher than previous reports in developed countries, using different definitions of P-AKI, which vary from 0.04% to 0.12%.[3, 4] The annual incidence increased for all AKI stages during the four-year study period, which is likely which is likely predominantly related to increased recognition, through increased testing and the use of automation with electronic alerts. We report that 77% of cases were AKI stage 1, triggered by a 1.5-times rise in serum creatinine or a change of 26 μmol/L from baseline.[12]However, KDIGO definitions of AKI are not validated in pregnancy, therefore, the reported incidence is likely overestimated, as gestational changes in renal physiology may account for a substantial proportion of AKI stage 1 alerts. Equally, there will remain a group of females whose P-AKI were missed, particularly in the first trimester due to lack of routine testing pre-pregnancy and the early physiological changes (e.g. glomerular hyperfiltration and increased renal blood flow), which may mask an AKI. AKI stage 2 and 3 occurred less frequently (0.88%) but still remains higher than previous reports in developed countries and may reflect an increasingly comorbid population.[3]

One example of comorbidity is CKD, which is recognised as an independent risk factor for acute kidney injury. In our cohort, the number of females with AKI superimposed on CKD was small (13/649; 2.0%). We were unable to accurately determine the number of females in the control group with CKD and therefore the relative impact of this risk factor remains difficult to determine.

Limitations

Limitations of our study were its retrospective design and it being a single centre study, which limits it generalizability, particularly outside the United Kingdom. Confounding factors for P-AKI which

were not possible to be recorded, such as routine baseline serum creatinine pre-pregnancy, underlying diabetes mellitus, coronary artery disease and smoking history are a further limitation.

Furthermore, we acknowledge that the "Asian", "Mixed", "Other" and "Black" ethnicity groupings were broad categories in order to allow for larger cohorts for comparison; however, this limits the ability for participants to accurately self-identify. In particular, "Other" incorporated to be for people who identified as being within smaller minority groups in the United Kingdom, such as Latino ethnicity and therefore specific representation for females in such groups was not possible. The numbers in the Asian sub-population remained relatively small and therefore limited conclusions can be drawn.

Conclusion

The incidence of P-AKI is likely higher than previously stated in the literature. However, caution must be exercised, particularly with AKI stage 1, as the KDIGO system is not validated in pregnancy and gestational changes in renal physiology need to be considered. Pregnancy-specific AKI definitions are needed.

Females identifying as Black ethnicity have more than a 50% increase risk in developing P-AKI, after adjusting for co-morbidity compared with females identifying as White ethnicity. The causes of these health disparities are likely to be both complex and multifaceted. Therefore, the strategy to combat this will need to be multidimensional. The first step is recognition and acknowledgment of the problem, and some progress is now being made within the medical profession and beyond, but further awareness is needed. It is likely that socioeconomic deprivation plays a role and so political changes including targeted government spending to reduce inequalities are urgently needed.

Equally, lack of trust in healthcare services is substantially lower in people identifying as Black, which is likely to result in decrease attempted access to care and ongoing health inequalities.

Addressing these issues directly within these communities and co-designing novel strategies to deliver health care, alongside trusted members, may help combat this. In the United States, studies with medical professionals in barber shops, demonstrated significant reductions in blood pressure.[47] Similar schemes are now being trialled in the UK and there is likely to be considerable learning from implementing these novel and important approaches.

Disclosures

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Tables

Table 1. Baseline characteristics for females without AKI (control) and with AKI, stratified for ethnicity.

	Total	**Non-AKI, N (%)**	**AKI, N (%)**	***p-value***
All	16,943	16,294 (96.2)	649 (3.8)	-
White	8,311	8,052 (96.9)	259 (3.1)	<0.001
Black	4,493	4,236 (94.3)	257 (5.7)	
Asian	988	951 (96.3)	37 (3.7)	
Mixed	761	739 (97.1)	22 (2.9)	
Other / Not stated	2390	2316 (96.9)	74 (3.1)	
Mean Age ± SD, years				
All	32.7 ± 5.5	32.7 ± 5.5	32.6 ± 5.7	>0.05
White	33.7 ± 5.0	33.7 ± 5.0	33.3 ± 5.2	>0.05
Black	31.7 ± 6.0	31.7 ± 5.9	32.0 ± 6.3	
Asian	32.6 ± 5.3	32.6 ± 5.3	32.6 ± 5.1	
Mixed	31.1 ± 5.8	31.0 ± 5.8	32.7 ± 4.9	
Other / Not stated	32.1 ± 5.7	32.1 ± 5.7	32.0 ± 5.7	

Median BMI (IQR), kg/m2				
All	24.7 (22.0, 28.6)	24.6 (21.9, 28.5)	26.8 (23.4, 31.6)	<0.001
White	23.6 (21.4, 26.5)	23.5 (21.4, 26.4)	25.0 (22.4, 28.5)	<0.001
Black	28.1 (24.5, 32.4)	28.0 (24.4, 32.3)	29.3 (25.0, 33.7)	
Asian	24.5 (21.6, 27.8)	24.5 (21.5, 27.8)	27.1 (23.6, 29.8)	
Mixed	24.3 (21.8, 28.3)	24.2 (21.7, 28.2)	29.7 (25.4, 34.2)	
Other / Not stated	24.5 (22.0, 28.0)	24.4 (21.9, 27.9)	25.6 (23.0, 29.6)	

Percentages represent proportion of each ethnicity with AKI. AKI = acute kidney injury; BMI = body mass index; IQR = interquartile range.

Table 2. Incidence of P-AKI stratified by ethnicity and AKI stages 1-3.

Ethnicity	Incidence of AKI, N (%)	AKI S1, N (%)	AKI S2, N (%)	AKI S3, N (%)
All (N=16943)	649 (3.8)	500 (3.0)	117 (0.7)	32 (0.2)
White (N=8311)	259 (3.1)	193 (2.3)	51 (0.6)	15 (0.2)
Black (N=4493)	257 (5.7)	206 (4.6)	39 (0.9)	12 (0.3)
Asian (N=988)	37 (3.7)	31 (3.1)	6 (0.6)	0.0
Mixed (N=761)	22 (2.9)	16 (2.1)	5 (0.7)	1 (0.1)
Other / Not stated (N=2390)	74 (3.1)	54 (2.3)	16 (0.7)	4 (0.2)

AKI = acute kidney injury; S1 = stage 1; S2 = stage 2; S3 = stage 3.

Table 3. Summary of obstetric outcomes for females with and without AKI.

	All Patients	Control, N (%)	AKI, N (%)	*Unadjusted OR (95% CI)*	P-value
Vaginal Delivery	9,071	8765 (96.6)	306 (3.4)		<0.001
White	4,249	4129 (97.2)	120 (2.8)	-	
Black	2,663	2545 (95.6)	118 (4.4)	1.60 (1.23, 2.07)	
Asian	498	478 (96.0)	20 (4.0)	1.44 (0.89, 2.33)	
Mixed	421	410 (97.4)	11 (2.6)	0.92 (0.49, 1.73)	
Other / Not stated	1,240	1203 (97.0)	37 (3.0)	1.06 (0.73, 1.54)	
Caesarean section	5,134	4875 (95.0)	259 (5.0)		<0.001
White	2,512	2407 (95.8)	105 (4.2)	-	
Black	1,402	1294 (92.3)	108 (7.7)	1.91 (1.45, 2.52)	
Asian	284	271 (95.4)	13 (4.6)	1.10 (0.61, 1.98)	
Mixed	217	208 (95.9)	9 (4.1)	0.99 (0.49, 1.99)	
Other / Not stated	719	695 (96.7)	24 (3.3)	0.79 (0.50, 1.24)	
Unknown / Stillbirth/Miscarriage)	414	379 (91.5)	35 (8.5)		<0.001
White	141	130 (92.2)	11 (7.8)	-	
Black	151	138 (91.4)	13 (8.6)	1.11 (0.48, 2.57)	
Asian	29	27 (93.1)	2 (6.9)	0.88 (0.20, 3.72)	

Mixed	22	22 (100)	0	-	
Other / Not stated	71	62 (87.3)	7 (10.1)	1.72 (0.44, 2.06)	
Blood Transfusion	423	357 (84.4)	66 (15.6)		<0.001
White	191	169 (88.5)	22 (11.5)	-	
Black	112	85 (75.9)	27 (24.1)	2.44 (1.31, 4.54)	
Asian	37	31 (83.8)	6 (16.2)	1.49 (0.56, 3.96)	
Mixed	15	13 (86.7)	2 (13.3)	1.18 (0.25, 5.59)	
Other / Not stated	68	59 (86.8)	9 (13.2)	1.17 (0.51, 2.69)	
Haemorrhage	1723	1594 (92.5)	129 (7.5)		<0.001
White	884	820 (92.8)	64 (7.2)	-	
Black	452	410 (90.7)	42 (9.3)	1.31 (0.87, 1.97)	
Asian	89	84 (94.4)	5 (5.6)	0.76 (0.30, 1.95)	
Mixed	58	53 (91.4)	5 (8.6)	1.21 (0.47, 3.13)	
Other / Not stated	240	227 (94.6)	13 (5.4)	0.73 (0.40, 1.36)	
Chronic hypertension	221	195 (88.2)	26 (11.8)		<0.001
White	61	51 (83.6)	10 (16.4)	-	
Black	120	107 (89.2)	13 (10.8)	0.62 (0.25, 1.51)	

Asian	17	16 (94.1)	1 (5.9)	0.32 (0.04, 2.68)	
Mixed	3	3 (100)	0	-	
Other / Not stated	20	18 (90.0)	2 (10.0)	0.57 (0.11, 2.84)	
Pre-eclampsia	436	276 (63.3)	160 (36.7)		<0.001
White	182	117 (64.3)	65 (35.7)	-	
Black	191	119 (62.3)	72 (37.7)	1.09 (0.71, 1.66)	
Asian	20	11 (55.0)	9 (45.0)	1.18 (0.44, 3.11)	
Mixed	8	5 (62.5)	3 (37.5)	1.08 (0.25, 4.67)	
Other / Not stated	35	24 (68.6)	11 (31.4)	0.83 (0.38, 1.79)	
Pregnancy-induced hypertension	447	333 (74.5)	114 (25.5)		<0.001
White	222	175 (78.8)	47 (21.2)	-	
Black	151	102 (67.5)	49 (32.5)	1.79 (1.12, 2.86)	
Asian	25	19 (76.0)	6 (24.0)	1.18 (0.44, 3.11)	
Mixed	12	10 (83.3)	2 (16.7)	0.74 (0.16, 3.52)	
Other / Not stated	37	27 (73.0)	10 (27.0)	1.38 (0.62, 3.05)	

AKI = acute kidney injury; IQR = interquartile range; S1 = stage 1; S2&3 = stages 2 and 3 combined.

Table 4. Median gestation at delivery and birthweight for all females divided into those with and without acute kidney injury and stratified for ethnicity.

	All (N=16,559)	Control (N=15,923	AKI (n=636)	P-value
Median Gestation, Days (IQR)	279 (272, 285)	279 (272, 285)	275 (264, 283)	<0.001
White	280 (273, 286)	280 (273, 286)	277 (267, 283)	
Black	277 (270, 284)	277 (217, 284)	274 (262, 282)	
Asian	276 (269, 282)	276 (270, 282)	273 (256, 281)	
Mixed	279 (273, 285)	279 (273, 285)	279 (269, 288)	
Other / Not stated	278 (272, 285)	278 (272, 285)	278 (268, 287)	
Median Birthweight, grams (IQR)	3365 (3025, 3680)	3370 (3030, 3680)	3250 (2700, 3595)	<0.001
White	3440 (3115, 3755)	3440 (3120, 3760)	3390 (2940, 3675)	
Black	3265 (2930, 3590)	3270 (2940, 3595)	3155 (2523, 3493)	
Asian	3175 (2853, 3520)	3178 (2859, 3520)	2915 (2500, 3508)	
Mixed	3380 (3014, 3686)	3380 (3014, 3686)	3450 (3235, 3685)	
Other / Not stated	3360 (3000, 3645)	3360 (3000, 3650)	3255 (2760, 3615)	

AKI = acute kidney injury; IQR = interquartile range.

Table 5. Unadjusted and adjusted odds ratios for acute kidney injury and ethnicity.

Females with Acute Kidney Injury			
	Unadjusted (N=16,943)	**Partially adjusted (N=12,898)**	**Fully adjusted (N=12,898)**
White	Ref	Ref	Ref
Black	1.89 (1.58, 2.25) ***	1.61 (1.31, 1.99) ***	1.52 (1.22, 1.80) ***
Asian	1.21 (0.85, 1.72)	1.01 (0.67, 1.52)	1.12 (0.73, 1.72)
Mixed	0.93 (0.60, 1.44)	0.81 (0.50, 1.32)	0.97 (0.59, 1.61)
Other	0.99 (0.76, 1.29)	1.04 (0.79, 1.38)	1.19 (0.88, 1.60)
Females with Acute Kidney Injury Stage 2 and 3			
White	Ref	Ref	Ref
Black	1.47 (1.01, 2.12)	1.39 (0.90, 2.15)	1.29 (0.83, 2.02)
Asian	0.77 (0.33, 1.78)	0.64 (0.23, 1.77)	0.73 (0.26, 2.06)
Mixed	0.99 (0.42, 2.29)	1.08 (0.46, 2.56)	1.29 (0.54, 3.08)
Other	1.05 (0.64, 1.74)	1.17 (0.68, 2.03)	1.36 (0.78, 2.37)

*** *p<0.001*

Associations are presented as odds ratios (95% confidence intervals). Associations are presented as unadjusted, partially adjusted (for age, BMI and chronic hypertension) and fully adjusted (for age, BMI, chronic hypertension, pre-eclampsia, pregnancy-induced hypertension, haemorrhage and mode of delivery).

Figures

Figure 1. Incidence of P-AKI per 12-month interval over a 4-year period. Data is also stratified into those with stage 1 AKI and those with stage 2 and 3 AKI combined.