Original Article

Role of Oxidative Stress Biomarkers and Antioxidant Enzyme Activity in the Pathophysiology of Maternal Anemia During Pregnancy

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ABSTRACT

Objective: To compare oxidative stress markers and antioxidant levels in pregnant women with and without anemia, and to look at how these markers relate to hemoglobin concentrations.

Study Design: Case-control study

Place and Duration of Study: This study was conducted at the Antenatal Clinics of Kerbala Obstetrics & Gynecology Teaching Hospital, Iraq from 1st June 2025 to 30th November 2025.

Methods: Forty-nine pregnant women with anemia (hemoglobin under 11 g/dL) were enrolled alongside 50 non-anemic pregnant women who served as controls. Serum levels of malondialdehyde, superoxide dismutase, catalase, and reduced glutathione were determined using standard spectrophotometric techniques.

Results: Antioxidant capacity was lower across the board in anemic women compared with controls: superoxide dismutase (0.051±0.055 vs 0.073±0.047; p=0.029), catalase (2.62±1.93 vs 8.48±4.43; p<0.001) and glutathione (49.05±17.5 7 vs 187.10±56.07; p<0.001). Malondialdehyde, on the other hand, was markedly elevated in the anemic group (7.16±5.01 vs 2.91±2.66 µmol/mL; p<0.001). Hemoglobin showed positive correlations with glutathione (r = 0.614), CAT (r = 0.521), and superoxide dismutase (r = 0.386), and a negative correlation with malondialdehyde (r = −0.547), all reaching p<0.001. In the multiple linear regression model, glutathione (β = 0.391) and malondialdehyde (β = −0.315) emerged as the strongest predictors of hemoglobin, and together the model accounted for 48% of the variance ($R^2 = 0.48$; $p < 0.001$).

Conclusion: Pregnant women with anemia showed both heightened lipid peroxidation and weakened antioxidant defenses. The strong links of glutathione and malondialdehyde with hemoglobin point to a role for oxidative stress in maternal anemia and suggest that improving antioxidant status may be worth exploring as a therapeutic avenue.

Key Words: Maternal anemia, Pregnancy, Oxidative stress, Malondialdehyde, Glutathione, Antioxidant enzymees

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INTRODUCTION

Among the complications that arise during pregnancy, anemia stands out as one of the most widespread. A woman's body undergoes substantial shifts in metabolism, hormonal output, and blood physiology as it works to sustain the developing fetus, and these adaptations place real strain on the hematological system.

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According to the World Health Organization, a hemoglobin concentration below 11 g/dL constitutes anemia in pregnancy.[1] Roughly four in every ten pregnant women worldwide fall below this threshold, with prevalence climbing much higher in low- and middle-income countries.[2] The clinical fallout is significant: anemia during pregnancy has been associated with preterm delivery, low birth weight, postpartum hemorrhage, and elevated maternal and perinatal mortality.[3]

While shortages of iron, folate and vitamin B12 continue to be recognized as the leading drivers of anemia in pregnancy[4], particularly the severity of certain cases and the inconsistent response to treatment and looked beyond nutritional factors. One mechanism drawing growing interest is oxidative stress, which arises when reactive oxygen species (ROS) outpace the antioxidant systems meant to keep them in check.[5] Pregnancy itself tilts the balance toward a pro-oxidant state. Both the placenta, with its high metabolic activity, and the elevated iron load that comes with gestation, encourage ROS production.[5] As pregnancy

advances normally, several oxidative stress markers have been observed to rise.[7] The trouble starts when antioxidant defenses can no longer match this increase leaving an excess of ROS free to inflict damage on maternal tissues.

Superoxide, hydrogen peroxide and the hydroxyl radical are generated continuously during normal metabolism and are ordinarily neutralized by enzymatic and non-enzymatic antioxidants.[5] Once production exceeds this capacity, ROS attack lipids, proteins, and DNA.[8] Erythrocytes are especially vulnerable. They carry a high oxygen load and abundant heme iron, their membranes are rich in polyunsaturated lipids, and, lacking a nucleus, they cannot replace oxidized proteins.[9] Sustained oxidative injury therefore shortens red-cell survival and can trigger hemolysis and eryptosis, the regulated death of erythrocytes, which provides a direct route from a pro-oxidant state to anemia.[10]

Stable biomarkers offer a practical way to gauge the extent of oxidative damage. The most commonly used of these is malondialdehyde e (MDA), an end product generated during lipid peroxidation.[8] Damage to circulating proteins can be tracked through advanced oxidation protein products (AOPP)[11], while 8- hydroxy-2′-deoxyguanosine (8-OHdG) serves as an indicator of oxidative injury to DNA.[12] Elevated readings of these markers have been documented in conditions such as preeclampsia, gestational diabetes, and fetal growth restriction.[13] How they behave in the context of maternal anemia, however, has not been examined as closely, and the small body of work that does exist tends to be constrained by limited sample sizes and methodological shortcomings.

Counterbalancing ROS is the job of the antioxidant network. Superoxide dismutase (SOD) takes superoxide converts it into hydrogen peroxide, which catalase then breaks down further into water and oxygen. Glutathione peroxidase, originally described in erythrocytes, clears hydrogen peroxide as well as lipid hydroperoxides, drawing on reduced glutathione (GSH) as its substrate.[14] Measuring the activity of these enzymes gives a sense of how effectively the maternal system is holding oxidative stress at bay. Reduced levels of SOD, catalase, and glutathione have been documented in iron-deficiency anemia in non-pregnant individuals[15], yet evidence connecting these defenses to the severity of anemia during pregnancy is still in short supply.

METHODS

The analytical case- control study was done at the antenatal care clinics of Obstetric & Gynecology Teaching Hospital in Kerbala, Iraq from 1st June 2025 to 30th November 2025 vide letter No. 293 dated May 8, 2025. These clinics serve as a referral and follow-up centre for pregnant women across Kerbala and its surrounding districts, which gave access to a broad patient population. The study was carried out in accordance with the principles set out in the Declaration of Helsinki[16] and its reporting follows the STROBE recommendations for observational research.[17]

Ninety-nine pregnant women were enrolled and classified into two groups. The case group comprised 49 women with anemia, defined as a hemoglobin concentration below 11 g/dL according to WHO criteria.[1] The control group comprised 50 non-anemic women with hemoglobin of 11 g/dL or above, matched to cases for age (within 2 years) and gestational age (within 2 weeks). Participants were enrolled consecutively, with eligible women approached in order of attendance until the target for each group was reached.

The women who confirmed singleton pregnancy with gestational age established by last period of menstration orultrasound in first-trimester, were aged 18 to 40 years, attended the clinic for routine follow-up, and gave written informed consent were included. Women were excluded for multiple gestation; pre-existing diabetes mellitus, chronic hypertension, renal, hepatic, or autoimmune disease; hemoglobinopathies such as sickle cell disease or thalassemia; use of antioxidant supplements (vitamin C, vitamin E, or N-acetylcysteine) within the preceding four weeks; active infection or inflammation; preeclampsia or other hypertensive disorders of pregnancy; blood transfusion within the preceding three months; or inability to give consent.

After enrolment, a structured interview recorded sociodemographic and obstetric data, including age, education, parity, gravidity, gestational age, inter-pregnancy interval, and dietary habits. Each participant's weight and height were recorded and body mass index was derived by dividing weight (in kilograms) by the square of height (in metres). Clinical examination note signs of anemia, including conjunctival, nail-bed and palmar pallor, and obstetric history were taken from the antenatal records. Blood pressure, heart rate and gestational ages were recorded at enrolment. A 10 mL venous blood sample was obtained from the antecubital vein under aseptic conditions between 08:00 a nd 10:00 following an overnight fast of no less than eight hours to minimise the effects of diurnal variation. Of the total volume drawn, 3 mL was transferred to EDTA tubes for the complete blood count while the remaining 7 mL went into plain gel- separator tubes. After allowing the latter to clot for 30 minutes, they were spun at 3,000 rpm for 10 minutes. The serum was then separated, divided into aliquots and kept at −80°C until analysis. Every sample was processed within two hours of collection and freeze-thaw cycles were kept to a minimum.

The complete blood count was run on an automated analyzer (Sysmex XN-550 or its equivalent) and covered hemoglobin, hematocrit, red blood cell count, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, platelet count and white blood cell count.

To classify the type of anemia present, serum ferritin, iron, total iron-binding capacity and folate were also measured.

Serum MDA, used here as an indicator of lipid peroxidation, was quantified using the thiobarbituricacid reactive substances (TBARS) method. Serum samples were mixed with thiobarbituric acid and trichloroacetic acid, heated at 95°C for 60 minutes, and the resulting chromogen measured at 532 nm . Concentrations were determined from a standard curve prepared with 1, 3, 3-tetramethoxypropane and reported as µmol/mL.

Antioxidant assays: Superoxide dismutase activity was estimated by measuring the degree to which the enzyme inhibited the reduction ofnitroblue tetrazolium by superoxide produced through the xanthine-xanthine oxidase system, with absorbance recorded at 505 nm. One unit was taken as the amount of enzyme needed to bring about 50% inhibition and the activity was normalised to sample protein, which was determined by the Lowry method. Catalase activity was assessed by tracking the decomposition of hydrogen peroxide at 240 nm in a phosphate buffer (pH=7.0) containing 30 mmol/L hydrogen peroxide and was likewise expressed in relation to sample protein. Reduced glutathione was measured following the procedure, which relies on the reaction between thiol groups and 5,5′-dithiobis (2-nitrobenzoic acid) at 412 nm, with results given in µmol/mL.

Quality control: All assays were carried out in duplicate and the mean of the two readings was used for analysis. Intra-assay and inter-assay coefficients of variation were maintained below 5% and 8%, respectively. Each batch included both positive and negative controls and laboratory staff remained blinded to the group assignment of every sample.

Data were analysed using SPSS-26. Distribution was checked with the Shapiro–Wilk test. Comparisons between groups were made using the independent-samples t- test. Pearson correlation was applied to look at the associations between biomarkers and hematological parameters and multiple linear regression was used to identify which biomarkers were independently linked to hemoglobin, with adjustments made for age, body mass index, gestational age and parity. A two-sided p value of less than 0.05 was treated as statistically significant.

RESULTS

Ninety nine had anemia and 50 were non-anemic controls. Antioxidant defenses were lower in the anemia group across all measures. SOD, catalase, and reduced glutathione were each significantly reduced relative to controls, while MDA was more than twice as high, indicating greater lipid peroxidation in anemic women (Table 1).

Hemoglobin was low in the anemia group compared to control (109.22 vs 115.92 g/L; p=0.005), in keeping with the case definition. The red blood cell count did not significantly differ between groups, whereas the count of white blood cell was higher among anemic women (Table 2).

Table No 1: Oxidative stress biomarker and antioxidant levels in the anemia and control groups

Variable	Maternal anemia (n = 49)	Control (n = 50)	t value	P value
SOD (µmol/mL/min/mg protein)	0.051±0.055	0.073±0.047	2.211	0.029
CAT (µmol/mL/min/mg protein)	2.623±1.933	8.483±4.427	8.503	< 0.001
MDA (µmol/mL)	7.158±5.005	2.913±2.662	−5.284	< 0.001
GSH (µmol/mL)	49.05±17.57	187.10±56.07	16.459	< 0.001

Table 2: Hematological parameters in the anemia and control groups

Variable	Maternal anemia (n = 49)	Control (n = 50)	t value	P value
RBC ($\times 10^{12}$/L)	3.87±0.44	3.73±0.45	−1.47	0.144
HGB (g/L)	109.22±11.85	115.92±11.33	2.87	0.005
WBC ($\times 10^{9}$/L)	7.35±5.54	5.09±2.07	−2.69	0.008

Table No 3: Correlations among oxidative stress biomarkers and antioxidants

Variable	Pearson correlation (r)	P value
GSH – MDA	−0.277	0.054
SOD – MDA	−0.527	< 0.001
CAT – SOD	0.270	0.061
CAT – MDA	−0.469	0.001

Table No. 4: Hemoglobin level Correlation and oxidative stress biomarkers

Variable	Pearson correlation(r)	P value
SOD (µmol/mL/min/mg protein)	0.386	< 0.001
CAT (µmol/mL/min/mg protein)	0.521	< 0.001
GSH (µmol/mL)	0.614	< 0.001
MDA (µmol/mL)	−0.547	< 0.001

Table No. 5: Multiple linear regression for factors associated with hemoglobin level

Predictors	β coefficients	SE	t test value	P value
SOD	0.182	0.073	2.49	0.015
CAT	0.267	0.088	3.03	0.003
GSH	0.391	0.095	4.12	< 0.001
MDA	−0.315	0.081	−3.89	< 0.001
Constant	8.742	1.654	5.29	< 0.001

Among the oxidative markers, MDA correlated inversely with SOD ($r = -0.527$, $p<0.001$) and with catalase ($r = -0.469$, $p = 0.001$). The SOD–catalase and GSH–MDA correlations were in the expected direction but did not reach significance (Table 3).

Hemoglobin positively correlated with glutathione ($r = 0.614$), catalase ($r = 0.521$), and SOD ($r = 0.386$) and negative with MDA ($r = -0.547$); all correlations were significant at $p<0.001$ (Table 4).

In the multiple linear regression model, glutathione and MDA were the strongest independent predictors of hemoglobin, with glutathione the leading positive predictor and MDA the leading negative predictor. The explained 48% by the model of the hemoglobin variance ($R^2 = 0.48$; adjusted $R^2 = 0.46$; $F = 22.11$; $p<0.001$) (Table 5).

DISCUSSION

In the present study, serum MDA, a measure of lipid peroxidation, was more than twice as high in anemic women as in controls. The antioxidants SOD, catalase and reduced glutathione were all lower in the anemia group and across the sample hemoglobin fell as MDA rose and increased with each antioxidantwith glutathione and MDA emerging as its strongest independent predictors. The pattern is consistent with real oxidative component to maternal anemia rather than incidental association.[18]

The higher MDA in anemic women fits a broad literature that links iron deficiency and anemia to lipid peroxidation. Sundaram et al[19] found raised plasma MDA in iron-deficiency anemia that fell after treatment and Tiwari et al[18] reported increased lipid peroxides alongside depleted antioxidants in pregnant anemic women. Yoo et al[20] found higher oxidant activity and lower total antioxidant capacity in women with iron-deficiency anemia, with values returning to normal after iron therapy and Sharif Usman et al[21] reported raised MDA in iron-deficient and anemic schoolchildren in northern Nigeria. The consistency of higher lipid peroxidation across pregnant women, non-pregnant women, and children suggests that it is a general feature of iron-deficient and anemic states rather than finding peculiar to one population. The susceptibility of erythrocytes helps to explain it: a high oxygen load, abundant heme iron, and polyunsaturated membranes favour radical formation, and the mature red cell cannot renew the proteins that oxidation destroys.[9] Peroxidation of membrane lipids lowers deformability and raises permeability, which ends in hemolysis and a shorter cell lifespan.[10] The elevated MDA in our anemic group is therefore likely to mark accelerated red-cell loss that adds to any nutritional deficit.

The parallel fall in SOD, catalase, and glutathione points to antioxidant defenses that have been outstrip ed. SOD produces hydrogen peroxide from superoxide, and catalase and the glutathione system then remove it[5], so depletion at several steps leaves erythrocytes poorly protected. Our SOD and catalase results agree with Tiwari et al[18], who found these enzymes reduced across grades of anemia, Yoo et al[20] reported lower catalase in iron-deficiency anemia, and Isler et al[22], who described reduced erythrocyte SOD and glutathione peroxidase that improved with treatment. The literature is not uniform, however. Khalid et al[15], working in pregnant women, found reduced SOD but a small, non-significant rise in glutathione peroxidase, which they read as a compensatory response under hypoxia. The earlier work of Macdougall[23] showed that glutathione peroxidase and catalase fall in iron-deficient red cells and that this loss raises their susceptibility to oxidative hemolysis. Such differences are not surprising. Studies vary in the tissue examined (serum or erythrocyte), the assay used, the cause and severity of anemia, and whether patients had already started treatment, and any of these can move enzyme activity in either direction. Set against that background, the lower SOD, catalase, and glutathione in our anemic group place this study with the larger body of work that reports weakened antioxidant defenses in anemia.

Reduced glutathione showed the largest difference between groups, close to a fourfold reduction. This fits its role as the main thiol antioxidant of the red cell and its heavy use under oxidative load.[24] Glutathione maintains protein thiols and supplies glutathione peroxidase, so its loss both reflects and worsens oxidative injury, and a fall in glutathione has long been tied to the oxidative hemolysis of iron-deficient cells.[23] Because iron is also a cofactor for catalase and supports glutathione-dependent reactions, iron deficiency may lower these defenses in its own right.[15]

The correlation and regression results add weight to the group comparisons. Hemoglobin rose with antioxidant capacity and fell with lipid peroxidation, and the regression model accounted for almost half of its variance with glutathione and MDA the leading predictors. Anemia brings tissue hypoxia and disturbed iron handling that promote ROS formation while the resulting oxidative damage impairs erythropoiesis and

shortens red-cell survival.[10] Redox balance also has a regulatory role in red-cell production: the transcription factor Foxo3 controls antioxidant gene expression in maturing erythroid cells, and its loss leads to ROS accumulation, oxidative damage, and a shorter erythrocyte lifespan.[25] Oxidative stress can in addition drive eryptosis, giving a further path from a pro-oxidant state to anemia.[10]

The findings have practical implications. If oxidative stress aggravates anemia, antioxidant status could serve as a marker of severity and potentially as a target for treatment. several studies report that iron therapy corrects hematological values and at the same time restores antioxidant defenses and lowers lipid peroxidation[15,20], which suggests that part of its benefit comes from easing oxidative stress. the picture is not one-sided. iron is itself redox-active, and Tiwari et al[26] found that oral iron supplementation raised markers of oxidative imbalance in anemic women, at least in the short term. This tension means that the dose and form of iron, and perhaps the addition of antioxidants, deserve careful study rather than assumption. Whether antioxidant supplementation improves outcomes in anemic pregnancy is not yet established and would need testing in controlled trials.[14]

This study assessed lipid peroxidation and thiol antioxidants but not protein or DNA oxidation. AOPP and 8-OHdG, which capture those forms of damage are raised in other complications of pregnancy[13], and adding them would give fuller view of the oxidative profile of maternal anemia. Their inclusion in future works help to define which forms of oxidative damage matter most in the disorder.

CONCLUSION

Maternal anemia was accompanied by greater lipid peroxidation and lower antioxidant defenses, with higher MDA and reduced SOD, catalase and glutathione in anemic women. Hemoglobin was closely related to these markers, and glutathione and MDA were its strongest independent predictors and the role for oxidative stress in the biology of maternal anemia and point to antioxidant status as a measure worth following and a possible target for treatment.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Manal Nasih Ahmed Hamdan
Drafting or Revising Critically:	Manal Nasih Ahmed Hamdan
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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