"SERUM LEAD LEVELS DURING PREGNANCY- A CROSS SECTIONAL STUDY (A DESCRIPTIVE OBSERVATIONAL STUDY) IN BELGAUM, KARNATAKA, INDIA"

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LIST OF ABBREVIATIONS USED

µg/dL	-	Micro gram per deciliter
ALA	-	Aminolevulinic acid
ALAD	-	Aminolevulinic acid dehydratase
ANC	-	Antenatal care
ANOVA	-	Analysis of variance
APA	-	American Pediatric Association
BLL	-	Blood lead level
BMI	-	Body mass index
CDC	-	Center for Disease Control
CI	-	Confidence interval
CNS	-	Central nervous system
EDD	-	Estimated date of delivery
eg	-	For example
EP	-	Erythrocyte protoporphyrin
EPA	-	Environmental Protection Agency
g	-	Grams
G6PD	-	Glucose-6-phosphate dehydrogenase
GM BLLs	-	Geometric mean blood lead levels
gm	-	Gram
GM	-	Geometric mean
HB	-	Haemoglobin
HBsAg	-	Hepatitis B antigen
HIV	-	Human immunodeficiency virus
HTN	-	Hypertension

HW	-	Housewife
I.D.No.	-	Identification number
IARC	-	International Agency for Research on Cancer
IQ	-	Intelligence quotient
kg/m^2	-	Kilogram per square meter
LMP	-	Last menopause
LSCS	-	Lower segment caesarean section
MCLG	-	Maximum Contaminant Level Goal
mL	-	Milli liter
mm of Hg	-	Millimeter of mercury
mumol/l	-	Micromole per litre
n	-	Total number
NHANES	-	National Health and Nutrition Examination Surveys
OPD	-	Out patient department
p	-	Probability
PbB	-	Maternal blood lead
PR	-	Pulse rate
RR	-	Relative risk
SD	-	Standard deviation
SFH	-	Symphysio fundal height
UK	-	United Kingdom
USG	-	Ultrasound
WHO	-	World Health Organization
XRF	-	X-ray fluoroscopy
ZPP	-	Zinc protoporphyrin

ABSTRACT

SERUM LEAD LEVELS DURING PREGNANCY - A CROSS SECTIONAL STUDY (DESCRIPTIVE OBSERVATIONAL STUDY)

Background and objective

Serum lead levels increase during pregnancy and may affect health of pregnant women and fetus. This study was aimed to know the serum lead levels during early, mid and late pregnancies as well as its association with maternal outcomes like anemia and hypertension in pregnant women.

Methodology

The present cross sectional study (descriptive observational study) of two year duration was carried out in the Department of Obstetrics and Gynaecology, attached teaching hospital, KLE University's Jawaharlal Nehru Medical College, Belgaum. A total of 32 pregnant women were studied for serum lead levels and haemoglobin levels.

Results

The average age of the study population was 22.00 ± 3.10 years. The gestational age in 13 (56.25%) women was 13 weeks and 23 (71.88%) women reported gravida 1. During the early pregnancy, all the 32 women (100%) had serum lead levels less than 10. During mid and late pregnancy, 3 (9.37%) and 11 (34.37%) women had serum lead levels of > 10 The mean serum lead levels showed an increasing trend from early pregnancy (2.87 ± 1.61) to late pregnancy (8.59 ± 8.33). At early pregnancy, 19 (59.38%) women were anaemic while in mid and late pregnancy anaemia was present in 24 (75%) and 30 (93.75%)

women respectively. The mean haemoglobin levels reduced as the pregnancy progressed. The correlation of anaemia and mean serum lead levels in mid and late pregnancy showed significant rise in mean serum lead levels with severity of anaemia ($p<0.050$).

Conclusion and interpretation

The serum lead levels in pregnant women increase with duration of gestation and are associated with anaemia.

Keywords:

Anaemia; Gestational hypertension; Preeclampsia; Serum lead levels;

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INTRODUCTION

The concentration of lead is increasing in environment due to industrialization and mining, so it is found more in air, water, soil and also a contaminant in humans. It is known that lead has no known physiological function.[1] It is a well known environmental and industrial toxin with a wide range of toxic effects.[2] It is known to cause harmful effects on major human organs including the reproductive system.[3]

Pregnancy is most vulnerable period to toxic substances. During pregnancy the effect of toxic substances like lead is more. Blood lead levels increases during pregnancy either from endogenous source (bone saved) or from environmental pollution and it effects health of pregnant women as well growing fetus.[4]

Continuous or recurrent exposure to toxic metals even in low concentrations during pregnancy, which can accumulate in the body will generally lead to significant damage to fetal development and maternal morbidities like anemia, preeclampsia and gestational hypertension.[5] Once lead is in blood stream, it enters fetus through placenta and effects fetal developing bone and other organs. Lead causes vascular endothelium damage and endothelial dysfunction is mediator of gestational hypertension.[4]

Increased bone turnover seen during pregnancy and lactation causes mobilization of bone lead stores especially in women with history of prior lead exposure.[4]

Recurrent or chronic exposure to high lead levels causes increase in arterial blood pressure of humans. This may result in maternal morbidities such as

miscarriage, pregnancy induced hypertension or preeclampsia, defects in hematopoietic system, cognitive decline, renal impairment. It also effects anthropometric measures and associated with fetal low birth weight and preterm labor.[6]

Numerous sources of lead are available in environment such as dust pollution, air pollution and soil pollution from leaded gasoline, paint, paint chips from older houses, plumbing and water supplies from lead pipes, cooking in leaded pots, use of cosmetics like surma and many other sources. It is difficult to avoid exposure to lead due to its widespread distribution in environment. Environmental lead is directly absorbed in the body either through respiration to lungs or by ingestion of substances contaminated with lead to gastrointestinal tract.

Primary biomarker used to calculate individual's lead burden levels in body is blood lead concentration or serum lead level.[7]

Blood lead levels < 5 µg/dL is considered normal. Blood lead levels between 5 µg/dL and 10 µg/dL requires follow up. Blood lead level greater than 10 µg/dL are managed with environmental assessment and abatement of exposures. Chelation therapy is considered in individuals with blood lead levels greater than 40 µg/dL. Lead levels more 70 µg/dL is considered as medical emergency.[6]

Lead poisoning remains an important public health problem in both developed and developing countries. It is considered to be one of the most difficult health issues during pregnancy. Prenatal lead exposure has known adverse effects on maternal health and fetal outcome depending on maternal serum blood lead levels. Maternal blood lead levels ≥ 10 µg/dL causes number of adverse outcomes in women like

anemia, infertility, miscarriage, hypertension and preterm delivery. However there is lack of direct association between maternal lead levels and some of the adverse pregnancy outcomes.[8,9]

Various studies have shown that there is a significant decrease in lead blood lead levels from 12 weeks to 20 weeks of gestation and significant increase in blood lead levels from 20 weeks to parturition, because of increased bone turnover and increased intestinal absorption during this period. Increased bone resorption occurs during pregnancy to accommodate the mineral needs of the fetus, which may lead to transient increase in serum lead levels.[10]

In 2010, the Centers for Disease Control and Prevention issued the first guidelines regarding the screening and management of pregnant and lactating women who have been exposed to lead.[11]

However little information is available about the blood lead levels in pregnancy due to the scarcity of data in the literature especially in south Indian population. Hence the present study was undertaken to know serum lead levels during early (10-14weeks), mid (24-28weeks) and late (32-36weeks) pregnancies and its association with maternal outcomes like anemia in pregnant women.

OBJECTIVES

The objectives of the present study were;

Primary

To know the blood level of lead during pregnancy.

Secondary

To find out is there an association between blood lead level and adverse maternal outcomes (anemia and hypertension).

REVIEW OF LITERATURE

Lead exposure remains a major public health problem for in women of childbearing age, in developing fetus and in nursing infant. Prenatal lead exposure has severe adverse effects on maternal health, infant outcome and neuro-developmental of child. Bone lead stores are mobilized during increased bone turnover seen in pregnancy and in lactating women with history of prior lead exposure, which is a concern since lead released into maternal blood and breast milk can adversely effect the fetus and newborn.[12]

Certain subgroup populations of women who are at increased risk for lead exposure have been identified. Some of the subgroups are as following: workers in lead factories, paint factories, lead pipe factories, recent immigrants from areas where lead contamination is high; and those group of women practicing certain habits associated with lead exposure, such as pica (Eating non-food substances such as soil) or renovation of older homes, Women who consume water contaminated with lead, Coking in lead glazed ceramic pottery, people taking herbal products/medications.[12]

Sources of Lead Exposure

Lead paint is a one of the primary source of lead exposure and the major source of lead toxicity. Drinking water, lead-glazed ceramics, food eaten or stored in containers painted with lead-based paint will contain significant amounts of lead. Ayurvedic remedies and products from India, China, and other parts of Asia may be potential sources of lead exposure. Workers in certain occupations such as working in lead pipe factories, paint factories are also exposed to high levels of lead. Lead exposure occurs during the manufacture of batteries, lead sheets, some brass and

bronze plumbing, ceramic glazes, circuit boards, military equipment (military tracking systems, jet turbine engines), intravenous pumps, fetal monitors, and some surgical equipment. Construction workers and farmers working with pesticides are known to have a high risk for lead exposure.[12]

Toxicology of Lead Absorption and Storage

Lead is man made source of environmental pollution which contributes to exposure in post-industrial era. Pre-industrial Native Americans living in the United States 700-1,000 years ago are estimated to have blood lead levels of 0.016 µg/dL. These lead levels are 50-200 times lower than those individuals living in remote regions of the Himalayas with no known lead exposure (0.78- 3.2 µg/dL) was reported currently. On the other hand, children with lead toys or soil dust exposure can have blood lead levels as high as 90 µg/dL.[13]

Bone levels in pre-industrial skeletal remains indicate that current body lead burdens are 500-1,000 times greater than the individuals with pre-industrial exposure to lead.[14]

Lead exposure occurs mainly through the respiratory and gastrointestinal tracts. Approximately 30-40% of inhaled lead is absorbed into the bloodstream.[15]

Gastrointestinal absorption varies depending on nutritional status and age. Iron is believed to impair lead uptake in the gut, while iron deficiency is associated with increased blood lead concentrations in children and pregnant women.[16]

Increased intakes of magnesium, phosphate, alcohol, and dietary fat have also been shown to decrease gastrointestinal absorption of lead.[17,18]

Gastrointestinal absorption of lead is more in infants. Infants can absorb up to 50% of lead ingested from food, water, contaminated dust, or soil and adults absorb only 10-15%.[19]

Inorganic lead (food, paint, water, toys, vinyl products) is minimally absorbed through the skin, but tetraethyl- or alkyl-lead (leaded gasoline) which is still legally allowed in aircraft, watercraft, and farm machinery, is well absorbed through the skin.[20]

Once lead is absorbed in circulation, 99% of lead is bound to erythrocytes for approximately 30-35 days (only one percent of absorbed lead is found in plasma and serum) and is transferred to the soft tissues – like liver, renal cortex, aorta, brain, lungs, spleen, teeth, and bones – over 4-6 weeks. Due to the short half-life of erythrocytes (35 days) in the bloodstream, blood lead levels cannot be used to diagnose or rule out the amount of exposure that occurred more than six weeks prior to testing.[21]

In adults approximately 80-95 % of retained lead is stored in the bone, while in children approximately 70 % is stored in bone, resulting in more soft tissue lead in children compared to adults.[15]

Lead is stored in bone and its estimated half-life is for a period of 20-30 years, including the bone turnover that occurs in childhood and also in adolescence. There is evidence to suggest that by the seventh decade of life, more than one-third of bone mass contains lead which has been acquired in childhood and adolescence.[22]

Lead in bone appears to increase significantly with age, due to slow turnover and release of lead from bone. Mean bone lead concentrations for adolescents have

been measured at 3 µg/g, concentrations for adults at 30-50 years is 17 µg/g and at age more than 75 years is 30 µg/g.[23]

Storage of lead in different compartments of bone is also an age-dependent process. During infancy and childhood, lead is deposited in trabecular bone because it is the most active site of remodeling; whereas, in adults lead is deposited in both trabecular and cortical bone.[24]

By adulthood most of the lead burden which is stored in cortical bone, teeth and also in trabecular bone is partially released back into the bloodstream and soft tissues by diffusion and resorption.[25]

Bone lead can contribute to elevated blood lead levels long after the exposure no longer exists.[26]

Situations that increase bone turnover, such as pregnancy, lactation, postmenopausal women, hyperthyroid women and in women undergoing cisplatin chemotherapy have been shown to increase blood lead levels as a result of the mobilization of bone stores.[12]

Bone lead is also readily transferred to the fetal skeleton through placenta during pregnancy.[12]

Inorganic lead is not metabolized in liver but is excreted unchanged mainly in urine. The mechanisms for fecal excretion of absorbed lead are not clearly understood. However about one-third of absorbed lead by fetus is excreted in the form of, secretion into bile, gastric fluid and saliva.[21]

Organic or alkyl-lead, (leaded gasoline, also identified as tetraethyl- and tetramethyl-lead) undergoes oxidative dealkylation into highly neurotoxic metabolites, triethyl- and tri- methyl-lead.[27] In the liver, the oxidative reaction is catalyzed by a cytochrome p450-dependent monooxygenase system.[28]

Lead is also known to be excreted through nails and sweat in subjects undergoing sauna therapy, there is significant losses of lead in sweat as compared to their urine levels.[29,30]

Understanding Lead Toxicity

Diagnosis and monitoring blood lead concentrations is the most commonly accepted and easily available biomarker for lead exposure. There is an agreement among the CDC, the Agency for Toxic Substances and Disease Registry and the Environmental Protection Agency, that lead is an unique toxicant and that there is no toxic threshold for lead. This means that any amount of lead in the body is harmful.[12]

The EPA (United States Environmental Protection Agency) has listed a Maximum Contaminant Level Goal (MCLG) of zero ppb (parts per billion) for lead in water and states that low level exposure of lead causes adverse health effects like interfering with red blood cell chemistry, delay in normal physical and mental development of infants and young children, deficits in attention span, deficits in hearing, learning abilities in children and mildly increases the blood pressure in some adults. Even low blood lead levels can cause changes in blood enzymes and in-turn effecting neurobehavioral development in children.[12]

Current excessive exposure guidelines developed by the CDC (Center for Disease Control and Prevention) and the American Pediatric Association (APA)

consider blood lead levels ≥10 µg/dL to be excessive for infants, children and women of childbearing age. Occupational exposure is hazardous when worker's blood levels exceed 30 µg/dL. In occupational settings effort is made to reduce air lead levels to limit exposure to the blood lead levels so that lead levels do not exceed 60 µg/dL.[31]

Decrease in production of leaded gasoline and lead- contaminated paints contributed to decrease in mean blood lead levels in the United States. Between 1976 and 1980, children ages 1-5 years had a mean blood lead level of 15.0 µg/dL;[32]

By 1999 the mean blood lead level had dropped to 1.9 µg/dL. Current mean blood lead levels in United State adults aged between 20 to 59 years as estimated by the National Health and Nutrition Examination Surveys (NHANES) III 1999-2002 are 1.5 µg/dL (95% CI, 1.5-1.6). Blood lead concentrations are highest in the strata that includes adults of age 60 years and older (2.2 µg/ dL; 95% CI, 2.1-2.3).[33]

Biomarkers of Hematological Toxicity Lead is a divalent cation. It has a strong binding capacity for sulfhydryl proteins and creates interference with enzymes and structural proteins. This is the most known cause for interference with the heme synthetic pathway, specifically the enzyme delta-aminolevulinic acid dehydratase (delta-ALAD; ALAD). Interference with heme production and sub- sequent reduction of the heme body pool is one of the main causes of lead-related pathology in anemia. When whole serum lead levels exceed 20 µg/dL, the activity of ALAD is inhibited by 50 %. However, ALAD activity may also be impaired in porphyria, liver cirrhosis and alcoholism. In addition, ALAD levels cannot be used to diagnose degrees of lead toxicity because the correlation between serum lead levels and ALAD is not linear.[34]

Inhibition of delta-ALAD prevents aminolevulinic acid (ALA) from being converted to porphobilinogen, inhibiting incorporation of iron into the protoporphyrin ring. The result is reduced heme synthesis, both for hemoglobin and for cellular respiration, contributing to the fatigue and anemia seen in chronic lead toxicity. In addition, inhibiting this enzyme results in increased circulating levels of ALA, leading to decreased GABA release in the central nervous system (CNS). This may explain some of the behavioral disorders seen in both porphyria and lead toxicity.[35]

Human ALAD has been shown to be a polymorphic enzyme with two common alleles, ALAD1 and ALAD2. The genetic polymorphism resulting in the expression of the ALAD2 allele appears to increase susceptibility to lead toxicity. Studies in various populations with lead exposure have found consistent relationships between the ALAD2 allele and elevated levels of blood and bone lead.[36]

A marked increase in urinary excretion of aminolevulinic acid (ALA), the substrate that accumulates as a result of decreased ALAD, has been used in the past as a marker for lead toxicity, but can be detected only when serum lead levels exceed 35 µg/dL in adults and 25-75 µg/dL in children. It is, therefore, not a useful biomarker in low-level toxicity.[37]

Anemia due to glucose-6-phosphate dehydrogenase (G6PD) deficiency is also known to increase the susceptibility to lead toxicity.[12]

Ferrochelatase, the enzyme that catalyzes the insertion of iron into protoporphyrin IX, is also impaired by lead resulting in increase of the substrate erythrocyte protoporphyrin (EP) when bound to iron and increase in zinc protoporphyrin (ZPP) when bound to zinc. Although these are used to diagnose acute

lead toxicity, these elevations do not appear in the blood until lead levels reaches 35 µg/dL. The threshold for EP or ZPP is 30 µg/dL in adults and 15 µg/ dL in children.[12]

Another limitation for using EP levels to assess lead exposure is that other diseases or conditions such as porphyria, liver cirrhosis, iron deficiency, and alcoholism may also produce similar effects on heme synthesis.[12]

Lead can also impair the activity of pyrimidine 5'- nucleotidase, increasing the pyrimidine nucleotides in red blood cells and preventing the maturation of erythroid elements, which leads to decreased red blood cell counts and eventually anemia. Hemoglobin levels do not start to decrease as a result of lead exposure until blood lead levels are 50 µg/dL for adults and 40 µg/dL for children. A potential biomarker for the hematological effects of lead is the observation of basophilic stippling and premature erythrocyte hemolysis. This effect, however, can occur in other toxicant exposures, including benzene and arsenic, and in a genetically-induced, enzyme-deficiency syndrome.[12]

Basophilic stippling and microcytic or normocytic, hypochromic anemia only occur after significant levels of exposure at blood lead levels over 50 µg/dL in adults and 25-40 µg/dL in children. Neither basophilic stippling nor hypochromic anemia are indicators of low level exposure.[12]

Bone Lead

Lead is considered to be stored in one of two compartments in the bone – the exchangeable pool at the bone surface and the non-exchangeable pool deeper in cortical bone. Lead can readily enter the plasma from the exchangeable pool, but can only leave the non-exchangeable pool and move to the surface of the bone when bone

is actively being resorbed. A study of lead-stable isotope signatures revealed that approximately 40-70% of blood lead in adults comes from bone lead.[37]

During pregnancy about 10-88% of blood lead may come from bone due to increased mobilization of bone during pregnancy. In the same study, the author concluded that approximately 80% of cord blood may result from liberated bone lead in the mother.[12]

Mobilization of bone during pregnancy may contribute not only to increased blood lead levels in pregnancy, but also during lactation. The same phenomenon of fetal exposure from maternal bone has been documented in primates where 7-39% of the maternal lead burden transferred to the fetus appeared to have been derived from lead in the maternal skeleton.[12]

Lead appears to have an osteoporotic effect in bone according to NHANES data from 1988-1994, bone mineral density alone was inversely correlated with blood lead levels in women age. [12]

Menopause appeared to compound the problem; lead levels were 25-35% higher in both natural and surgical menopause.[39] Cortical bone lead, currently measured by x-ray fluoroscopy (XRF) is considered a sensitive biomarker for cumulative lead exposure and correlates well with historical rather than current blood lead measurements which do not reflect total body burden as measured by bone stores.[40]

Both EDTA mobilization testing and bone XRF are considered the most consistent and sensitive tools for assessing body lead burden.[12]

Lead is stored in soft tissues; autopsy studies show the liver as the largest repository of soft tissue lead (33%), followed by renal cortex, renal medulla, pancreas, ovary, spleen, prostate, adrenal gland, brain, fat, testis, heart, and skeletal muscle. Though there is a high turnover rate of lead during the life time, the lead levels in soft tissues appear to be relatively constant.[12]

Toxic Effects of Lead

Lead toxicity affects the central and peripheral nervous systems, renal function and the vascular system. The toxic effects of lead vary greatly, manifesting as subtle changes in neurocognitive function in low-level exposure or as the potentially fatal encephalopathy due to acute lead poisoning. As exposure progresses, symptoms of toxicity may manifest differently. Laboratory abnormalities will also differ based on the chronicity and level of exposure.[12]

The final hydroxylation of vitamin D into its active hormonal form 1,25-dihydroxyvitamin D takes place in renal tubule and lead is believed to interfere with the conversion of vitamin D to its active hormonal form.[12]

Workers exposed to lead in manufacturing facilities demonstrate an increased frequency of still-births, miscarriages, reduced sperm counts, reduced sperm motility, hypospermia, increased rates of teratospermia, decreased fertility and decreased libido. Women who have lead-exposed male partners have higher rates of miscarriage. Children of lead-exposed workers have increased rates of morbidities such as congenital epilepsy and cardiovascular disease.[12]

Lead was recently upgraded from the status of a possible to a probable human carcinogen by the International Agency for Research on Cancer (IARC), based on

sufficient evidence for carcinogenic effects in humans.[41] Lead exposure has been related to increased incidence of cancers such as stomach, lung and bladder cancer.[42]

A study done on subjects with lead exposure and age-related cataract incidence, found that men having highest tibial bone lead levels are at 2.5 times greater risk of developing cataract as compared to those men with the lowest level of tibial lead.[43]

Individuals with cataracts have significant levels of lead in the ocular lens along with decreased levels of zinc in lens tissue. An increased ratio of lead to zinc in the lens was related to decreased lens transparency.[44]

Reproductive and Prenatal Effects

Lead exposure remains a concern for pregnant and lactating women, particularly those who have an occupational exposure to lead, who are recent immigrants, engaged in home renovations. Prenatal lead exposure resulting in maternal serum lead levels <10 μg/dL has morbities such as anemia, infertility, hypertension and effects infant neurodevelopment. In addition, because lead persists in bone for decades, as bone stores are mobilized to meet the increased calcium needs of pregnancy and lactation, the women and their infants might be exposed to lead long even after external sources have been removed.[45] Adverse reproductive effects are not only limited to women but also males with occupational lead exposure like abnormal sperm morphology and decreased sperm count have been observed at blood lead levels <40 μg/dL.[46]

Historical Trends in Blood Lead Levels

Since the 1970s, NHANES data have been used to track blood lead levels for the non-institutionalized United States population. In the 1991–1994 NHANES, the overall prevalence of blood lead levels ≥10 μg/dL was 2.2% but decreased to 0.7% by the 1999–2002 survey. Overall, the geometric mean (GM) decreased significantly ($p<0.05$; two-tailed t-test) from 2.3 μg/dL to 1.6 μg/dL during the same time period.[47]

Although the decrease in GM BLLs (Geometric mean blood lead levels) in women aged 20–59 years from 1.8 μg/dL in 1988–1994 to 1.2 μg/dL in 1999–2002, in-utero exposure is also a substantial public health issue for certain populations, particularly in new immigrants coming from lead contaminated regions.[48]

In 2003 a study conducted in New York City, blood lead levels ≥ 5 μg/dL were more prevalent among pregnant women who were born outside the United States than pregnant women born in the United States (odds ratio=8.2, 95% confidence interval = 3.8–17.3).[47]

Risk Factors for Lead Exposure in Pregnant Women[49]

- Immigrants from the areas where ambient lead contamination is high, women from countries where leaded gasoline is still being used (or was recently phased out) or where industrial emissions are not well controlled.

- Living near a point source of lead which includes lead mines, battery recycling plants (even if the establishment is closed).

- Women who works or her family members work in an industry that uses lead (eg, lead production, battery manufacturing, paint manufacturing, ship building, ammunition production or plastic manufacturing).

- Women who cook, store, or serve food in lead-glazed ceramic pottery made in a traditional process.

- Eating nonfood substances (pica) — women who eat or mouth nonfood items that may be contaminated with lead, such as soil or lead-glazed ceramic pottery.

- Using alternative or complementary substances, herbs, or therapies women who use imported home remedies or certain therapeutic herbs traditionally used by East Indian, Indian, Middle Eastern, West Asian, and Hispanic cultures that may be contaminated with lead.

- Using imported cosmetics or certain food products — women who use imported cosmetics, such as kohl or surma or certain imported foods or spices that may be contaminated with lead.

- Women who engage in high-risk activities (eg, stained glass production or pottery making with certain leaded glazes and paints) or have family members who do it.

- Renovating or remodeling older homes without lead hazard controls in place—women who have been disturbing lead paint, creating lead dust, or both or have been spending time in such a home environment.

- Consumption of lead-contaminated drinking water—women whose homes have leaded pipes or source lines with lead.

- Having a history of previous lead exposure or evidence of elevated body burden of lead — women who may have high body burdens of lead from past exposure, particularly those who have deficiencies in certain key nutrients (calcium or iron).

- Living with someone identified with an elevated lead level — women who may have exposure in common with a child, close friend, or other relative living in the same environment.

Lead Screening during Pregnancy

Environmental policies and public health education programs have led to significant reductions in cases of lead exposure in the United States.[50] Despite these improvements, approximately 1% of women of childbearing age (15–49 years) have blood lead levels greater than or equal to 5 micrograms/dL.[11]

Although no threshold has been found to trigger the adverse health effects of lead,[11] in nonpregnant adults blood lead levels less than 5 μg/dL are considered normal, blood lead levels between 5 μg/dL and 10 μg/dL require follow-up and blood lead levels greater than 10 μg/dL are managed with environmental assessment and abatement of exposures. Chelation therapy is considered at blood lead levels greater than 40 μg/dL for symptomatic individuals and levels greater than 70 μg/dL are considered as medical emergency. In children treatment is recommended at blood lead levels of 45 μg/dL or greater.[49]

Adverse Health Effects of Prenatal Exposure

Lead readily crosses the placenta by passive diffusion and has been detected in the fetal brain as early as the end of the first trimester. Elevated lead levels in pregnancy have been associated with several adverse outcomes, including anemia, gestational hypertension, miscarriage, low birth weight and impaired neurodevelopment.[49]

Lead exposure has been associated with an increased risk of gestational hypertension, but the magnitude of the effect, the exposure level at which risk begins to increase and whether risk is most associated with acute or cumulative exposure remain uncertain. Also, it is unclear whether lead induced increase in blood pressure during pregnancy causes severe hypertension or preeclampsia.[49]

Evidence shows that maternal exposure to high levels of lead increases the risk of spontaneous abortion.[51] However, data for an association between low or moderate lead levels and spontaneous abortion are inconsistent. The strongest available evidence comes from a prospective study of 668 pregnant women in Mexico City that demonstrated a statistically significant dose–response relationship between low-to-moderate maternal blood lead levels and the risk of spontaneous abortion.[52]

There is a longitudinal study done in Japan among 351 pregnant women which showed no difference in serum lead levels between spontaneous abortion cases (n=15) and ongoing pregnancies.[53]

More recent and well-designed studies suggest that maternal lead exposure during pregnancy is inversely related to fetal growth, as reflected by duration of pregnancy and infant size. One study that used a registry-based approach found that

offspring of mothers occupationally exposed to lead had an increased risk of low birth weight (relative risk [RR], 1.34; confidence interval [CI], 1.12–1.6) compared with infants of women not exposed to lead.[54]

A case–control study in Mexico City found umbilical cord blood lead levels to be higher in preterm infants (mean value, 9.8 micrograms/dL) compared with term infants (mean value, 8.4 micrograms/dL).[55] A birth cohort study, also conducted in Mexico City, found maternal bone lead burden to be inversely related to offspring weight[22], length and head circumference at birth.[56]

A large number of studies provide evidence that prenatal lead exposure impairs children's neurodevelopment. Some prospective studies have included that children with low levels of prenatal lead exposure had impaired neurodevelopment, although these data are less consistent than those related to the high levels of lead exposure. In one study, each 1 μg/dL increase in umbilical cord blood lead was found to be associated with a reduction of 0.6 points in the mental development index scores of the Bayley Scales of Infant Development at age 3 months, with similar results at age 6 months.[57,58]

However, another prospective cohort study found that the relationship between prenatal blood lead levels and early childhood IQ is not linear, with the strongest postnatal effects noted at low levels of prenatal exposure.[59]

The available data are inadequate to establish the presence or absence of an association between maternal lead exposure and major congenital anomalies in the fetus.[49]

The Centers for Disease Control and Prevention (CDC)[11] and the American College of Obstetricians and Gynecologists[49] do not recommend blood lead testing of all pregnant women in the United States. Obstetric health care providers should consider the possibility of lead exposure in individual pregnant women by evaluating risk factors for exposure as part of a comprehensive health risk assessment and perform blood lead testing if a single risk factor is identified. Assessment of lead exposure should take place at the earliest contact with the pregnant patient.

Lead-based paint is less likely to be an important exposure source for pregnant women than it is for children, except during renovation or remodeling in older homes. Women should take precautions when repainting surfaces with deteriorated paint or performing any remodeling or renovation work that disturbs painted surfaces, such as scraping off paint or tearing out walls.[11]

For pregnant women with blood lead levels of 5 μg/dL or higher, sources of lead exposure should be identified and women should receive counseling regarding avoidance of further exposure. Confirmatory and follow-up blood lead testing should be performed in accordance with the CDC's recommended schedules and maternal or umbilical cord blood lead levels should be measured at delivery.[11] Women with confirmed blood lead levels of 45 μg/dL or more should be treated in consultation with experienced clinicians in the management of lead toxicity and high-risk pregnancy. Once the source of lead exposure is identified and eliminated, the initial decrease in blood lead level occurs relatively rapidly because of lead's short (35-day) initial half-life in blood.[60] This initial rapid decrease is followed by a slow, continuous decrease over several months to several years because of mobilization of lead from stores in the bone.[11]

Adequate dietary intake of calcium, iron, zinc, vitamin C, vitamin D, and vitamin E is known to decrease lead absorption. Iron-deficiency anemia is associated with elevated serum lead levels and may increase lead absorption. During pregnancy and lactation, lead from prior exposures can be mobilized from bones because of the increased bone turnover. Pregnant and lactating women with a current or past serum lead level of 5 μg/dL or higher should receive specific nutritional recommendations regarding calcium and iron supplementation.[49] A balanced diet that contains 2,000 mg of calcium and 60–120 mg of iron daily is recommended.[11] This can be achieved through either food intake or supplementation. Supplements should be divided into doses of 500 mg of calcium and 60 mg of iron to improve absorption.

Frequency of Maternal Blood Lead Follow-up Testing During Pregnancy[11,49]

Venous Blood Lead Level* (micrograms/dL)	Perform Follow-up Test(s)†
Less than 5	• None (no follow-up testing is indicated)
5–14	• Within 1 month • Obtain a maternal blood lead level‡ or cord blood lead level at delivery
15–24	• Within 1 month and then every 2–3 months • Obtain a maternal blood lead level‡ or cord blood lead level at delivery • More frequent testing may be indicated based on risk factors

25–44	• Within 1–4 weeks and then every month • Obtain a maternal blood lead level‡ or cord blood lead level at delivery
45 or more	• Within 24 hours and then at frequent intervals depending on clinical interventions and trend in blood lead levels • Consultation with a clinician experienced in the management of pregnant women with blood lead levels in this range is strongly advised • Obtain a maternal blood lead level or cord blood lead level at delivery

Screening guidelines[49]

- Routine blood lead testing of all pregnant women is not recommended.
- Risk assessment of lead exposure should take place at the earliest contact with pregnant or lactating women and blood lead testing should be performed if a single risk factor is identified.
- Elevated lead levels in pregnancy have been associated with gestational hypertension, spontaneous abortion, low birth weight and impaired neurodevelopment.

- Prenatal lead exposure has known adverse effects on maternal health and infant outcomes across a wide range of maternal blood lead levels.

- Pregnant women with blood lead levels of 5 μg/dL or higher should be treated:

- Sources of lead exposure should be identified. Women should receive counseling regarding avoidance of further exposure and receive specific nutritional recommendations regarding calcium and iron supplementation because these strategies can decrease their lead levels.

- Confirmatory and follow-up blood lead testing should be performed in accordance with the CDC's recommended schedules.

- Women with confirmed blood lead levels of 45 μg/dL or more should be treated in consultation with clinicians experienced in the management of lead toxicity and high-risk pregnancy.

- Initiation of breastfeeding should be encouraged postpartum in a woman with a blood lead level less than 40 μg/dL.

- A breastfeeding woman with a confirmed blood lead level of 40 μg/dL or higher should be advised to pump and discard her breast milk until her blood lead level has decreased to less than 40 μg/dL.

- If no external source is identified, and the maternal blood lead level is greater than 20 μg/dL and the infant blood lead level is 5 μg/dL or more, breast milk should be suspected as the source and temporary interruption of breastfeeding until the maternal blood lead level decreases should be considered.

Prevalence of elevated lead in asymptomatic pregnant women

Blood lead levels and blood umbilical cord lead levels are frequently used to assess both the mother's and fetus' levels of lead exposure and risk.

In 1992, two large surveys of low-income pregnant women found 0%[7] and 6%[8] with blood lead levels >5 μg/dL.[61,62]

A study of all women who enrolled in prenatal clinics in Mahoning County, Ohio, from 1990 to 1992 found that 13% of prenatal patients had blood lead levels ≥10 μg/dL, with 1% having blood lead levels greater than 15 μg/dL.[63]

Population mean blood lead levels in women of childbearing age and pregnant women have fallen over the past two decades. Although it was estimated in 1990 that 4.4 million women of childbearing age, and over 400,000 pregnant women, had blood lead levels of >10 μg/dL,[64] a study of 1109 infants in Quebec, Canada, found a mean cord blood lead of 1.5 μg/dL (0.076 umol/l; 95% CI = 0.074, 0.079).[65]

In a review of NHANES data of 4,394 women of child-bearing age, the GM blood lead levels 1.78 μg/dL[67] and a longitudinal study of pregnant women in Boston demonstrated that umbilical cord blood lead levels declined 82% between 1980 and 1990.[67]

Adverse effects of lead exposure on pregnancy outcomes

The effects of very high blood lead levels during pregnancy on reproductive outcomes such as abortion and stillbirth have been recognized for many years.[21]

Observational studies in pregnant women with blood lead levels less than 30 μg/dL have reported associations between elevated levels and birth weight, length of gestation (including preterm delivery) and neonatal head circumference.[68]

The associations have been small, variable in direction of effect and not statistically significant in most studies. These data failed to detect important effects on other reproductive outcomes. Inconsistent results may be due in part to imprecise measures of fetal lead exposure.[68]

A meta-analysis[69] reported no association between antenatal or perinatal maternal blood lead levels and full-scale IQ measured at preschool or school age. Although very high lead levels in pregnancy are clearly hazardous, the adverse effects on the fetus of antepartum lead levels in the range typically found in the U.S. are not established.

In august 2012, the American College of Obstetricians and Gynecologists[49] documented that prenatal lead exposure has known adverse effects on maternal health and infant outcomes across a wide range of maternal blood lead levels. The recommendations are risk assessment of lead exposure should take place at the earliest contact with pregnant women. Blood lead testing should be performed if a single risk factor is identified. Elevated lead levels in pregnancy have been associated with gestational hypertension, spontaneous abortion, low birth weight and impaired neurodevelopment. Women with confirmed lead blood levels of 45µg/dl or more should be treated in consultation with clinician's expert in the management of lead toxicity and high risk pregnancy.

A study was conducted in 1998 to compare the blood lead levels of 97 pregnant women warded at the Kuala Lumpur hospital, according to their ethnicity, residence and place of work. The lead content of venous blood samples was determined with a graphite furnace atomic absorption spectrometer. Blood levels of Klang Valley women seem to have decreased from 17.3 µg/dl in 1982 to 7.71 µg/dl in this present study most probably attributed to the phasing out of leaded gasoline. This level is below the 10 µg/dl recommended by the United States Environmental protection agency for the public, even though 27.8% of them still have blood lead levels that are equal to or in excess of 10 µg/dl.[70]

In a cross sectional study conducted in the urban slums of Lucknow, north India in 1996, 500 pregnant women were enrolled. The mean lead was 14.3 µg/dl and 19.2% of women had lead ≥ 20 µg/dl. Lead was not associated with age, height, weight, gestation or history of abortions, although higher lead was associated with higher parity. Women living in inner city neighborhood near heavy vehicular traffic had lead 2.2 µg/dl higher than those living in other neighborhoods. The lead was not associated with reported use of piped water or the presence of paint in homes and increase in lead was unexpectedly associated with decreasing use of the eye cosmetic 'surma' and the duration of gestation. The high lead found in this population raises concern about fetal development and points to the urgent need to reduce the exposure to lead.[71]

In a cross sectional study done in 105 pregnant women residing in Valley of Mexico between march 1987 and june 1992, serum blood was taken from week 12 to week 36 of pregnancy, again at parturition and was analyzed. Although geometric mean lead level averaged around 7.0µg/dl with a range of 1.0µg/dl – 35.5µg/dl

throughout pregnancy, analysis of variance revealed a significant decrease in mean lead from week 12 to week 20 and various significant increases in mean lead from week 20 to parturition. Regression analysis confirmed the positive linear lead trend from 20 weeks to parturition and additional contributions of dietary calcium, reproductive history, life time residence in Mexico city, coffee drinking and use of indigenous lead-glazed pottery.[72]

METHODOLOGY

The present study was conducted in the Department of Obstetrics and Gynaecology, attached teaching hospital, KLE University's Jawaharlal Nehru Medical College, Belgaum.

Study design

The study design was a cross sectional study (descriptive observational study).

Time line for the study

PHASE	TIME PERIOD	OUTLINE
I	June 2012 to August 2012	1.Identification of problem 2.Review of Literature 3.Development of data collection instrument 4.Submission of Synopsis
II	December 2012 to July 2014	1.Enrolment 2.Data Collection
III	August 2014 to September 2014	1.Analysis of collection data 2.Discussion
IV	September & October 2014	1.Submission of dissertation

Source of data

Pregnant women in early, mid and late pregnancy attending out patient the Department of Obstetrics and Gynaecology, attached teaching hospital, KLE University's Jawaharlal Nehru Medical College, Belgaum were studied.

Sample size

Minimum sample of 30 pregnant women were studied.

Screened were: 43

Consented were: 35

Outcome data available for: 32

Sampling procedure

A minimum sample of 30 pregnant women was planned.

Selection Criteria

Inclusion

1. Eligible consenting pregnant women registering for ANC and following at the Department of Obstetrics and Gynaecology, attached teaching hospital, KLE University's Jawaharlal Nehru Medical College, Belgaum.

2. Gestational age of pregnant women included are:
    a) Early pregnancy : 10 - 14 weeks.
    b) Mid pregnancy : 24 - 28 weeks.
    c) Late pregnancy : 32 - 36 weeks.

Exclusion Criteria:

- Pregnant women with known medical disorders or on medication.
- HIV and HBsAg positive women.
- Known alcoholic or drug abuse.
- Use of prescribed ayurvedic medications.
- Active psychosis.

Method of Collection of Data

Ethical clearance

The ethical clearance was obtained from Review Board of Jawaharlal Nehru Medical College, Belgaum. (MDC/DOME/895) dated on 31/10/2012 (Annexure I). This study is also registered in CTRI (CTRI/2013/12/004229).

Screening and Enrolment

Consented women potentially eligible women visiting for early antenatal visit were screened for eligibility based on selection criteria. Informed consent was obtained at the time of enrolment into the study from the eligible women. The informed consent form was provided by the investigator to the patient to be enrolled. The investigator obtained a signature or left hand thumb impression from the consented subject. Adequate time was provided for describing the study and fielding questions from the patient and/or immediate family members. Fair balance was

maintained while describing the risks and benefits of participation in the study. No undue pressure was placed on the patient to enroll in the study.

It was further explained that lack of participation will not affect the usual and anticipated standard of care. The women were enrolled in the study only after taking their signature or left hand thumb impression on informed consent form (Annexure II).

A unique subject ID was assigned to those women who consented for the study staring from 001. The following scheme was used for this purpose:

Data collection form

Data collection instrument containing information about present and past pregnancies was completed. The investigator, previously trained in the administration of the questionnaire, completed the Antenatal Record of the data collection instrument at the time of enrolment into the study and the Delivery information.

Investigations

Pregnant women underwent investigations for :

- HB % during early, mid and late pregnancy.
- Serum lead level during early, mid, late pregnancy.

Assessment of serum lead levels

Under aseptic precautions, 5 mL of venous blood was taken in prescribed time of pregnancy as specified for every pregnant women and analyzed for serum lead levels using Perkin Elmer graphite furnace atomic absorption spectrometer. The

pregnant women were assessed for serum lead levels in early, mid and late gestational ages.

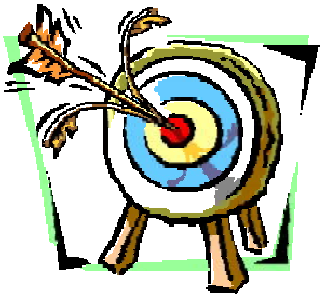
Assessment of haemoglobin

Hemoglobin estimation was done in all participants using HemoCue machine at early, mid and late gestational ages.

Statistical Analysis

The data entered into data monitoring system and master chart (Annexure III/IV) was prepared. The categorical data was expressed as percentages, rates and ratios and the comparison was done using the chi-square test. Continuous data was expressed as mean ± standard deviation (SD) and the comparison was done using independent sample t test. A probability value ('p' value) of less than or equal to 0.05 was considered as statistically significant.

Introduction



Objectives

Review of Literature

Methodology

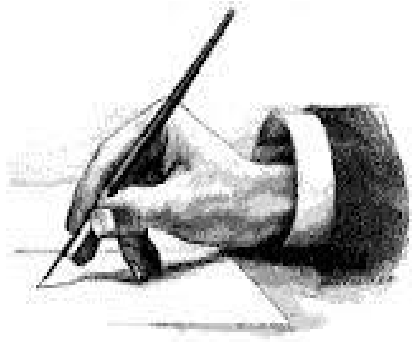
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-III

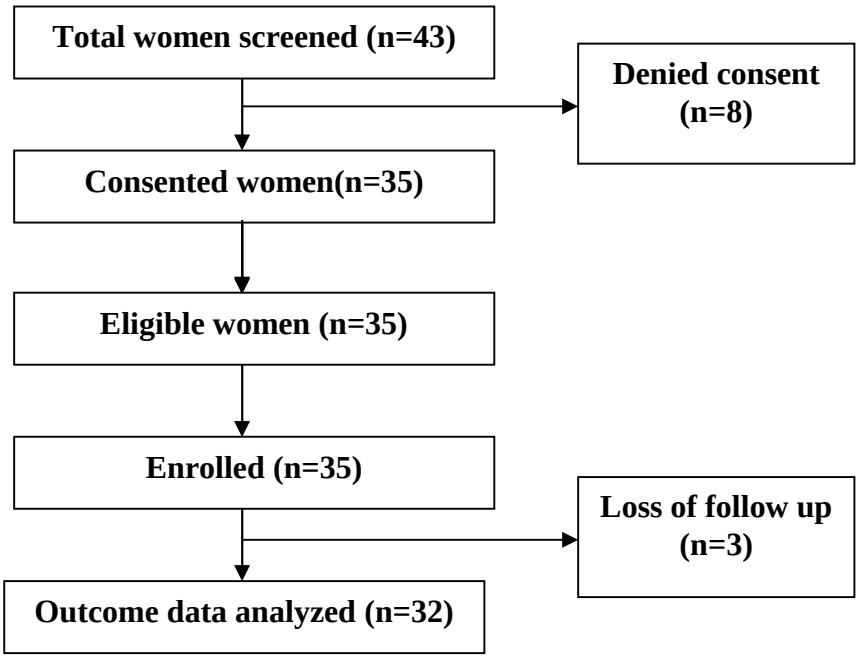
Annexure-IV

Annexure-V

RESULTS

This cross sectional study (descriptive observational study) was conducted in the Department of Obstetrics and Gynaecology attached to teaching hospital KLE University's Jawaharlal Nehru Medical College, Belgaum. In this study, a total of 43 cases were screened, out of which 35 had given consent for participating in the study. However three women were lost to follow up and outcome data was available in 32 women.

Flow chart of study population



The data obtained was analysed and the final results and observations were tabulated as below.

Table 1. Demographic characteristics of the study population

Variables	Sub groups	Distribution (n=32)	
		Number	**Percentage**
Mean age		22.00 ± 3.10	
Occupation	Agriculture	12	37.50
	Housewife	9	28.13
	Professional	6	18.75
	Labour	5	15.63
	Total	**32**	**100.00**
Education	Read / Write	9	28.13
	Illiterate	8	25.00
	Primary	8	25.00
	Secondary	5	15.63
	Graduate	2	6.25
	Total	**32**	**100.00**
Socio economic status	PAN card	13	40.63
	White Card	18	56.25
	Yellow card	1	3.13
	Total	**32**	**100.00**
Body mass index	19.8 to 25.99	30	93.75
	26.00 to 28.99	2	6.25
	Total	**32**	**100.00**

The demographic characteristics of the study population are as shown in table 1. Majority of the women (71.88%) were aged between 19 to 22 years. Most of the women (28.13%) were able to read and write and engaged in agriculture related activities (37.5%) having white card (56.25%).

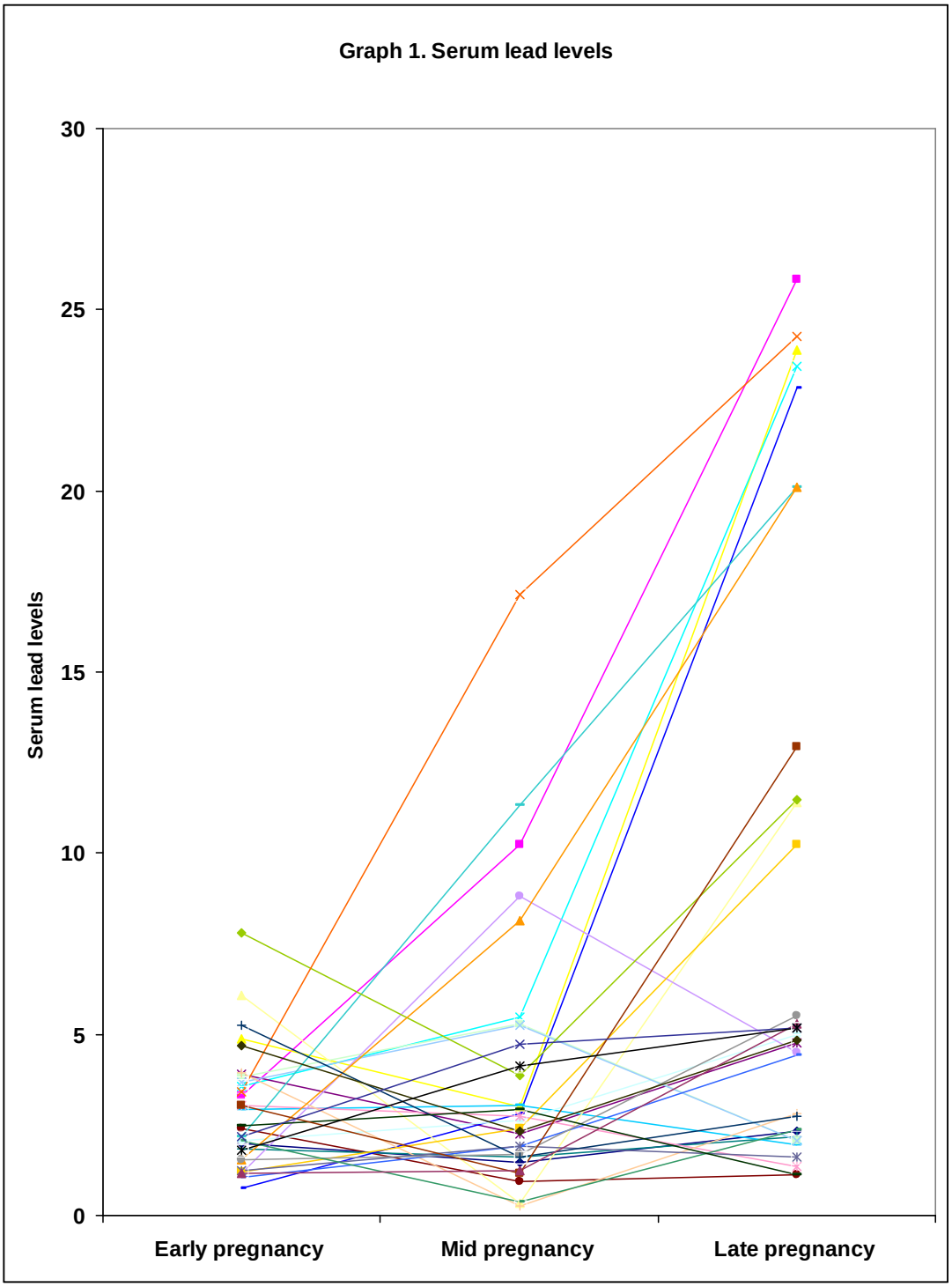

Graph 1. Serum lead levels
Serum lead levels
30
25
20
15
10
5
0
Early pregnancy
Mid pregnancy
Late pregnancy

Table 2. Obstetric history

Variables	Sub-groups	Distribution (n=32)	
		Number	**Percentage**
Gestational age at the time of enrolment in weeks.	10	1	3.13
	11	1	3.13
	12	11	34.38
	13	18	56.25
	14	1	3.13
	Total	**32**	**100.00**
Gravida	1	23	71.88
	2	6	18.75
	3	3	9.38
	Total	**32**	**100.00**

In the present study 56.25% of the women presented with gestational age of 13 weeks and 71.88% reported gravida 1.

Table 3. Serum Lead levels and haemoglobin in early pregnancy

Serum Lead levels	No. of women	Haemoglobin levels (gm%)							
		≥ 11		10 to 10.9		7 to 9.9		< 7	
		No	%	No	%	No	%	No	%
≤ 5.00	29	12	37.50	16	50.00	1	3.13	0	0.00
5.01 to 9.99	3	1	3.13	2	6.25	0	0.00	0	0.00
Total	**32**	**13**	**40.63**	**18**	**56.25**	**1**	**3.13**	**0**	**0.00**

In the present study during early pregnancy, 56.25% of the women had haemoglobin levels between 10 to 10.9 gm%. In this subset 50% had serum lead levels ≤ 5 and 6.25% had serum lead levels between 5.01 to 9.99.

Table 4. Serum Lead levels and haemoglobin in mid pregnancy

Serum Lead levels	No of women	Haemoglobin levels (gm%)								Gestational HTN		Pre-eclampsia	
		≥ 11		10 to 10.9		7 to 9.9		< 7					
		No	%	No	%	No	%	No	%	No	%	No	%
≤ 5.00	24	8	25.00	11	34.38	5	15.63	0	0.00	4	12.50	3	9.38
5.01 to 9.99	3	0	0.00	0	0.00	3	9.38	0	0.00	1	3.13	2	6.25
≥ 10	5	0	0.00	1	3.13	4	12.50	0	0.00	1	3.13	1	3.13
Total	**32**	**8**	**25.00**	**12**	**37.50**	**12**	**37.50**	**0**	**0.00**	**6**	**18.75**	**6**	**18.75**

In this study during mid pregnancy, 24 women had serum lead levels of ≤ 5. In this subset, 34.38% had haemologbin levels between 10 to 10.9 gm% followed by 25% with ≥ 11 gm% and 15.63% of the women with haemoglobin levels between 7.0 to 9.9 gm%. Most of the women with gestational hypertension and preeclampsia had serum lead levels of ≤ 5 and between 5.01 to 9.99 respectively.

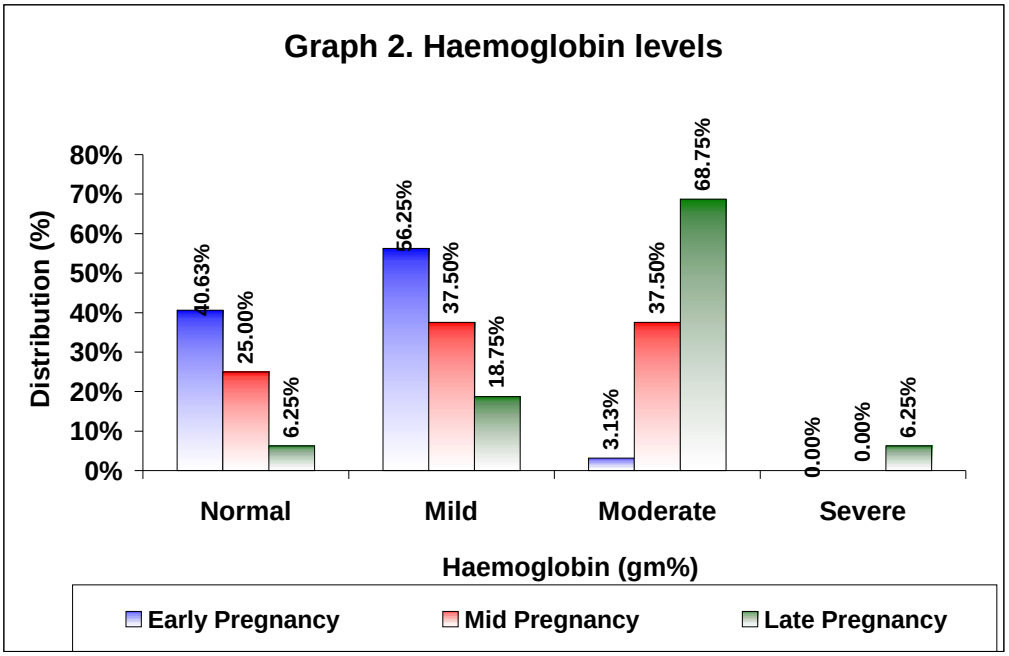
Table 5. Serum Lead levels and haemoglobin in late pregnancy

Serum Lead levels	No of women	Haemoglobin levels (gm%)								Gestational HTN		Pre-eclampsia	
		≥ 11		10 to 10.9		7 to 9.9		< 7					
		No	%	No	%	No	%	No	%	No	%	No	%
≤ 5.00	17	2	6.25	6	18.75	9	28.13	0	0.00	1	3.13	3	9.37
5.01 to 9.99	4	0	0.00	0	0.00	4	12.50	0	0.00	1	3.13	3	9.37
≥ 10	11	0	0.00	0	0.00	9	15.63	2	3.13	3	9.38	1	3.13
Total	**32**	**2**	**6.25**	**6**	**18.75**	**22**	**68.75**	**2**	**6.25**	**5**	**15.62**	**7**	**21.87**

In the present study during late pregnancy 11 women had serum lead levels of ≥ 10 and the haemoglobin levels were found to be between 7 to 9.9 in 15.63% and 3.13% had < 7 gm%. Gestational hypertension was noted in 9.38% of the women with serum lead levels of ≥ 10 and pre-eclampsia was present in 3.31%.

Table 6. Haemoglobin levels

Haemoglobin (gm%)	Early Pregnancy (n=32)		Mid Pregnancy (n=32)		Late Pregnancy (n=32)	
	No	%	No	%	No	%
Normal (≥ 11)	13	40.63	8	25.00	2	6.25
Mild (10 to 10.9)	18	56.25	12	37.50	6	18.75
Moderate (7 to 9.9)	1	3.13	12	37.50	22	68.75
Severe (< 7)	0	0.00	0	0.00	2	6.25
Total	**32**	**100.00**	**32**	**100.00**	**32**	**100.00**



The haemoglobin levels and severity of anaemia is as depicted in table 7 and graph 5. It was observed that, during early pregnancy most of the women had mild anaemia (56.25%). During mid pregnancy 37.5% of the women each had mild and moderate anaemia while in late pregnancy 68.75% had moderate anaemia.

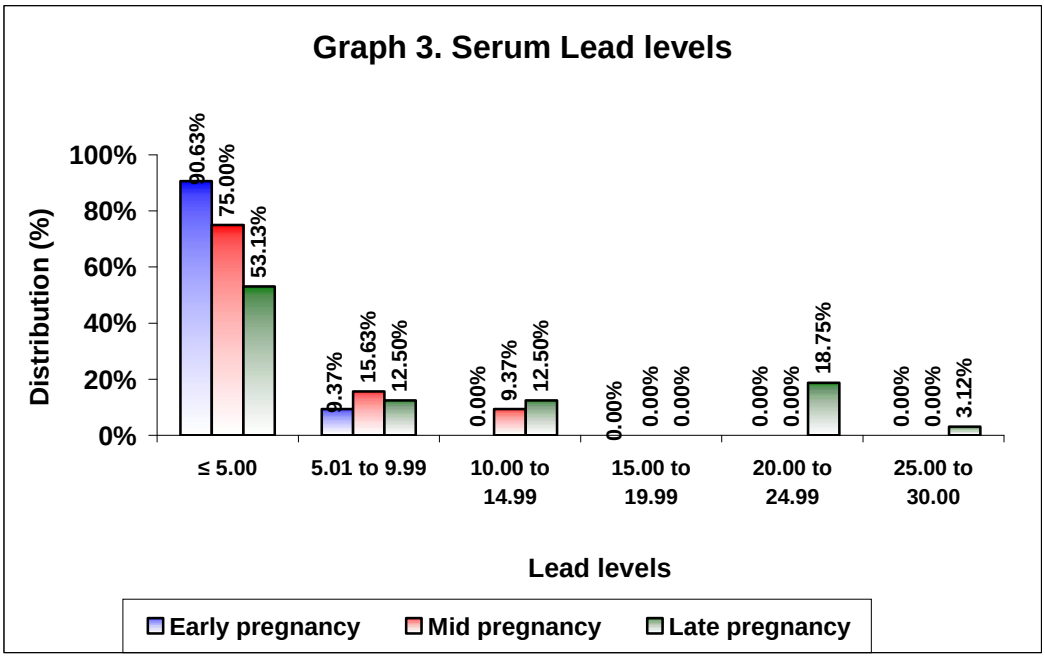
Table 7. Mean and median haemoglobin levels

	Mean (n=32)		Median (n=32)		
	Mean	**SD**	**Median**	**Minimum**	**Maximum**
Early pregnancy	10.68	0.75	10.4	9.40	12.40
Mid pregnancy	9.97	1.28	10	7.20	12.20
Late pregnancy	8.85	1.37	9.1	6.20	11.20

Table 8 shows mean and median haemoglobin levels. It was observed that the mean haemoglobin levels reduced from early pregnancy (10.68 ± 0.75 gm%) to late pregnancy (8.85 ± 1.37 gm%).

Table 8. Serum Lead levels

Lead levels	Early pregnancy (n=32)		Mid pregnancy (n=32)		Late pregnancy (n=32)	
	No	%	No	%	No	%
≤ 5.00	29	90.63	24	75.00	17	53.13
5.01 to 9.99	3	9.37	5	15.63	4	12.50
10.00 to 14.99	0	0.00	3	9.37	4	12.50
15.00 to 19.99	0	0.00	0	0.00	0	0.00
20.00 to 24.99	0	0.00	0	0.00	6	18.75
25.00 to 30.00	0	0.00	0	0.00	1	3.12
Total	**32**	**100.00**	**32**	**100.00**	**32**	**100.00**



In the present study 90.63% of the women had normal serum lead levels during early pregnancy. During Mid pregnancy, 15.63% of the women had serum lead levels between 5.01 to 9.99. In late pregnancy, 12.5% of the women each had serum lead levels between 5.01 to 9.99 and 10 to 14.99.

Table 9. Mean and median lead levels

	Mean (n=32)		Median (n=32)		
	Mean	**SD**	**Median**	**Minimum**	**Maximum**
Early pregnancy	2.87	1.61	2.43	0.76	7.80
Mid pregnancy	3.84	3.70	2.69	0.03	17.15
Late pregnancy	8.59	8.33	4.91	1.11	25.84

In this study, the mean serum lead levels increased from early pregnancy (2.87 ± 1.61) to late pregnancy (8.59 ± 8.33).

Table 10. Correlation of haemoglobin levels and mean lead levels

Haemoglobin (gm%)	Early pregnancy (n=32)		Mid pregnancy (n=32)		Late pregnancy (n=32)	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Normal (≥ 11)	2.2	1.80	1.5	1.00	1.5	0.20
Mild (10 to 10.9)	3.3	1.40	2.5	1.40	2.6	1.00
Moderate (7 to 9.9)	3.9	-	6.8	4.50	9.4	7.90
Severe (< 7)	-	-	-	-	24.3	2.10
F value	2.137		9.649		5.831	
p value	**0.136**		**0.0006**		**0.003**	

The correlation of haemoglobin and mean serum lead levels is as shown in table 13. During mid and late pregnancies it was observed that, the mean serum lead levels significantly increased with severity of anaemia ($p<0.050$).

DISCUSSION

The present study was aimed to know the serum lead level during pregnancy and its association with anaemia and hypertension in pregnant women.

This two year cross sectional study (descriptive observational study) was carried out in the Department of Obstetrics and Gynaecology, attached teaching hospital, KLE University's Jawaharlal Nehru Medical College, Belgaum. A total 32 women were studied from December 2012 to July 2014.

In the present study during the early pregnancy, all the 32 women (100%) had serum lead levels less than 10 while in mid pregnancy, 3 women (9.37%) were detected to have serum lead levels of > 10 and in late pregnancy the same were noted in 11 women that is 34.37% of the women. Similarly the mean serum lead levels showed an increasing trend with low being at first trimester (2.87 ± 1.61) and high during third trimester (8.59 ± 8.33) suggesting rise in serum lead levels with duration of gestation.

A study[54] in Mexico analyzed maternal blood lead (PbB) of 105 women living in the Valley of Mexico from week 12 to week 36 of pregnancy and again at parturition. Although geometric mean PbB level averaged around 7.0 micrograms/dl (0.34 mumol/l), throughout pregnancy, analysis of variance revealed a significant decrease in mean PbB from week 12 to week 20 (1.1 micrograms/dl) and various significant increases in mean PbB from week 20 to parturition (1.6 micrograms/dl). The finding of the present study were consistent with a study by Rothenberg SJ et al.

The ALSPAC study[75] to determine blood lead levels BLL in a large cohort of pregnant women in the UK found that, mean maternal BLL was 3.67±1.47 µg/dl (n=4285; geometric mean 3.43, median 3.42, range 0.41–19.14 µg/dl). The distribution was slightly skewed. The BLL was ≥5 µg/dl in 619 women (14.4%) and ≥10 µg/dl in 15 women (0.4%). Women of Indian, Pakistani and Bangladeshi ethnicity had significantly higher BLL than White women (4.37±1.92 (n = 23) vs 3.65±1.47 (n = 3585), respectively, ANOVA p = 0.019).

In this study during early pregnancy 19 women that is, 59.38% of the women were found to be anaemic. In mid and late pregnancy the anaemia was noted in 24 (75%) and 30 (93.75%) women respectively. It was observed that, during early pregnancy 18 women had mild anaemia (56.25%) while in mid pregnancy 12 (37.5%) women each had mild and moderate anaemia but in those with late pregnancy 22 women had moderate anaemia (68.75%). The mean haemoglobin levels showed a trend towards reduction as the pregnancy progressed that is, in early pregnancy the mean haemoglobin levels were 10.68 ± 0.75 gm% which reduced to 8.85 ± 1.37 gm% in late pregnancy. The correlation of anaemia and mean serum lead levels in mid and late pregnancy showed significant rise in mean serum lead levels with severity of anaemia ($p<0.050$). Further the linear correlation of haemoglobin with serum lead levels showed weak negative correlation during early pregnancy (R=-0.201) and in mid pregnancy there was moderate negative correlation (R=-0.5915) but in late pregnancy there was strong negative correlation (R=-0.7972). The findings strengthen hypothesis that, in mid and late pregnancy the increase in serum lead level are associated with anaemia.

Recently, a study[76] from Lucknow conducted a study to correlate blood lead levels (BLLs) and oxidative stress parameters in pregnant anemic women. A total of 175 pregnant women were found suitable and included for this study. Following WHO criteria, 50 each were identified as non-anemic, mild anemic and moderate anemic and 25 were severe anemic. Results showed significantly (p\0.01) high BLLs, in all groups of anemic pregnant women as compared with non anemic pregnant women. Study concluded that low BLLs perturb oxidant-antioxidant balance and negatively affected hematological parameters which may eventually Pb to Fe deficiency anemia during pregnancy.

In this study it was also found that there was positive co-relation between increase in serum lead levels in pregnancy and hypertension (gestational hypertension/ pre eclampsia)

It was found that about 6 women (18.75%) each had gestational hypertension and pre eclampsia in mid pregnancy and during late pregnancy the same were noted among 5 (15.62%) and 7 (21.87%) women.

Lead is transported in blood bound to haemoglobin, so it was unsurprising that haemoglobin level was found to be a predictor of BLL. As pregnancy progresses, the effect of haemodilution is counterbalanced by the increase in lead released into the blood through increased bone turnover, resulting in the U-shaped curve observed by Hertz-Piccioto et al.[77] The rate of bone turnover is thought to be greatest in the third trimester and so this may also account for the independent effect of gestational age.

In the present study the mean age was 22.1 ± 3.1 years. In contrast, a similar study from Mexico in 1994 reported higher mean age that is, 27.5 years. The disparity

in the mean age between the present study and study from Mexico could be attributed to the socio-cultural practices of early marriages in India.

It is evident from the present study that the serum lead levels increase to abnormal levels as pregnancy advances and it is associated with anaemia and hypertension. The findings of the study needs to continued with larger sample multicentric study.

Limitations of the study

1. Design of the study: Cross-sectional study (Descriptive observational study)
2. Smaller sample size
3. Cost of investigations:

Per investigation, per patient is	Rs. 1350/-
3 times each patient	Rs. 4050/-
Total cost for 32 patients	Rs. 1,29,600/-

CONCLUSION

Based on the findings of this study it is evident that, the serum lead levels in pregnant women increase with duration of gestation. Further there is moderate negative correlation between serum lead levels and haemoglobin during mid pregnancy and strong negative correlation during late pregnancy suggesting increase in lead levels associated with anaemia.

It is also observed that, there is positive co-relation between increase in serum lead levels and hypertension (gestational hypertension/ pre eclampsia). The findings of the same study need to be confirmed by larger multicentre trial.

SUMMARY

Pregnancy is most vulnerable period to toxic substances. Serum lead levels increase during pregnancy and affect health of pregnant women and growing fetus. This study was aimed to know the serum lead levels during early (10-14weeks), mid (24-28weeks) and late (32-36weeks) pregnancies as well as its association with maternal outcomes like anemia in pregnant women.

This two year cross sectional study (descriptive observational study) was carried out in the Department of Obstetrics and Gynaecology attached teaching hospital KLE University's, Jawaharlal Nehru Medical College, Belgaum. A total of 32 pregnant women who satisfied the selection criteria were enrolled from December 2012 to July 2014.

The mean age of the study population was 22.00 ± 3.10 years. Most of the women that is 9 (28.13%) were able to read and write, 12 (37.5%) were engaged in agriculture related activities (37.5%) and 18 (56.25%) had white card. The gestational age in 13 (56.25%) women was 13 weeks and 23 (71.88%) women reported gravida 1. During the early pregnancy, all the 32 women (100%) had serum lead levels less than 10 while in mid pregnancy, 3 (9.37%) women were detected to have serum lead levels of > 10 and in late pregnancy the same was noted in 11 (34.37%) women. The mean serum blood levels showed an increasing trend from early pregnancy (2.87 ± 1.61) to late pregnancy (8.59 ± 8.33). With regard to haemoglobin levels, at early pregnancy 19 (59.38%) women were anaemic while during mid and late pregnancy anaemia was present in 24 (75%) and 30 (93.75%) women respectively. The mean haemoglobin levels reduced as the pregnancy progressed. The correlation of anaemia

and mean serum lead levels in mid and late pregnancy showed significant rise in mean serum lead levels with severity of anaemia ($p<0.050$).

Based on the findings of this study it was concluded that, the serum lead levels in pregnant women increase with duration of gestation.

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ANNEXURE III

SCREENING FORM

Title: A cross sectional study (descriptive observational study) – Serum lead levels during pregnancy.

I. SCREENING:

Id.No:

OPD No:

Date:

Unit:

Patient's Name: F _____M_____S_____

Age:

Address: H. No._____ Street _____ Place _____

Taluka _____ District_____

Tel No: _____

Mobile: _____

1) Is she eligible? [ ]

a) Yes b) No

If no why (specify) _____

2) Is she enrolled ? [ ]

a) Yes b) No

3) Whether patients is willing to participate and give consent [ ]

a) Yes b) No

ANNEXURE IV

DATA COLLECTION INSTRUMENT

Title: A cross sectional study (descriptive observational study) – Serum lead levels during pregnancy.

Screening ID. No :

Screening questions

1) What is the gestational age of the patient? [ ]

2) Is there any maternal medical complications present? [ ]

a) No b) Yes

3) If ans is yes to Q.6 then specif ?

4) Did the patient used any prescribed ayurvedic medications:

5) Is patient habitual use of alcohol or drugs? [ ]

a) Yes b) No

6) Is patient recent emigration from or residency in areas where ambient lead contamination is high.

a) Yes b) No [ ]

7) Is patient Living near a point source of lead- examples include lead mines, smelters or battery recycling plants.

a) Yes b) No [ ]

8) Is patient Working with lead or living with someone who does?

a) Yes b) No [ ]

9) Is patient Using lead-glazed ceramic pottery?

a) Yes b) No [ ]

10) Using imported cosmetics or certain food products- imported cosmetics like kohl or surma or certain imported foods or spices that may be contaminated with lead.

a) Yes b) No [ ]

Demographic Data

1) Occupation: [ ]

a) HW

b) Working with pesticides

c) Labourer

d) Professional

2) Education: [ ]

a) Illiterate

b) Read

c) Write

d) Primary

e) Secondary

f) Graduate

g) Post graduate

3) What type of socio-economic card she has? [ ]

a) White

b) Green

c) Yellow

d) Red

e) Pan(income tax)

Obstetric history

4) Is it singleton pregnancy? [ ]

a) Yes b) No

5) Was her previous cycles regular? [ ]

a) Yes b) No

6) Gravida [ ]

7) Para [ ]

8) Living [ ]

9) Abortions:

10) LMP :

11) EDD :

12) Is there any history of previous LSCS? [ ]

a) Yes b) No

13) According to USG EDD is?

III. Examination:

14) Height (incms):

15) Weight (in kgs):

16) BMI (kg/m2):

17) PR:

18) Blood pressure:

Systolic (mm of Hg) :

Diastolic (mm of Hg):

Investigations

19) Antepartum Hb% (in gms):

Early pregnancy

Mid pregnancy

Late pregnancy

20) Serum lead level (at how many weeks):

Early pregnancy

Mid pregnancy

Late pregnancy

21) Did patients develop hypertension

Gestational hypertension Preeclampsia

ANNEXURE V – KEY TO MASTER CHART

AG	-	Agriculture
bpm	-	Beats per minute
Cms	-	Centimeters
G	-	Graduate
G	-	Gravida
gms	-	Grams
HW	-	Housewife
ID	-	Identification
IL	-	Illiterate
Kg/m^2	-	Kilograms per square meter
Kgs	-	Kilograms
L	-	Labour
LSCS	-	Lower segment caesarean section
mm Hg	-	Millimeters of mercury
N	-	No
P	-	Primary
PC	-	Pan card
PM	-	Primi
PR	-	Professional
R	-	Regular
S	-	Secondary
WC	-	White card
Y	-	Yes
YC	-	Yellow card

ANNEXURE II

INFORMED CONSENT FORM

I.D.NO:

"A cross sectional study (Descriptive observational study) - Serum Lead Levels During Pregnancy"

The study is conducted by Dr. _____Post graduate student in M.S Obstetrics and Gynaecology under guidance of _____ (Principal investigator), Professor, Department of OBG, J. N. Medical College, Belgaum.

Respected Sir/Madam, We invite you to participate in our study as you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study:

The purpose of this study is to study the variations in serum lead levels during pregnancy. All patients who fulfil the inclusion criteria will be requested to participate in this study during the period of one year.

Procedure and treatment:

Should you choose to participate, serum lead levels are measured by collecting 2ml of venous blood in each patient for each trimester i.e,three times in each patient in a EDTA bulb and analysis for blood lead will be done at the biochemistry division of the Institute of J N Medical college, Dr Prabhakar Kore Trust Hospital's and Medical Resaerch Center.

Risks and benefits:

You may undergo some amount of discomfort during the process of,which may include slight pain while pricking to collect venous blood. However all necessary steps

and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding of the different management practices and the outcome, and thus we will be able to provide improved patient care.

Alternatives:

If you decide not to participate in this study, you will still be receiving the usual standard care.

Privacy and confidentiality:

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional policy:

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project. In the event if you suffer any physical injury as the result of your participation in this study, you may contact Dr. _____ Telephone No_____ or Dr_____, Telephone No_____. In the event of an emergency, you should contact KLE'S Dr. Prabhakar Kore Hospital and MRC on Telephone No. 08312473777.

Financial incentives:

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results:

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation:

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. In case you need further information regarding your rights as a study participant, you may please contact Dr. Ganga Pillai, Chairman of the ethical committee, J N Medical College, Belgaum on telephone No. 08312473777

Statement of Consent:

I.D.NO:

I Mr/Ms/Mrs _____Volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

STATEMENT OF CONSENT:

I.D.NO:

I Mr/Ms/Mrs

Volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

ಅನುಬಂಧ - 5
ಹೇಳಿಕೆ ಒಪ್ಪಿಗೆ ಫಾರ್ಮ್

ಐ. ಡಿ. ನಂ.

ಗರ್ಭಾವಸ್ಥೆಯಲ್ಲಿ ರಕ್ತಸಾರ ಸೀಸದ ಸಮತಟ್ಟಿನ ಕುರಿತು ತಂಡಗಳ ಎದುರಿನಲ್ಲಿ ಅಭ್ಯಾಸ

ಸದರೀ ಅಭ್ಯಾಸವನ್ನು ಡಾ : ವಿದ್ಯಾರ್ಥಿನಿ,
ಎಮ್.ಎಸ್. ಆಬ್ಸೆಟ್ರಿಕ್ಸ ಹಾಗೂ ಗ್ಯಾನಾಕಾಲಾಜಿಯಲ್ಲಿ ಡಾ :
ಪ್ರಿನ್ಸಿಪಾಲ ಇನ್‌ವೆಸ್ಟಿಗೇಟರ) ಓಬಿಜಿ ವಿಭಾಗದ ಪ್ರೊಫೆಸರ್ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಬೆಳಗಾವ ಇವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಮಾಡುತ್ತಿದ್ದಾರೆ.

ಮಾನ್ಯ ಮಹನೀಯರೇ /ಮಹಿಳೇಯರೆ ತಮ್ಮನ್ನು ಆ ಈ ಅಭ್ಯಾಸದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಅರ್ಹರಿದ್ದುದರಿಂದ ಆಹ್ವಾನಿಸುತ್ತಿದ್ದು ಆ ಸಮಯದಲ್ಲಿ ಸಧ್ಯದ ನಿಮ್ಮ ತಕರಾರುಗಳನ್ನು ವಿವರವಾಗಿ ಕೇಳುತ್ತೇವೆ

ಅಭ್ಯಾಸದ ಉದ್ದೇಶ : ಈ ಅಭ್ಯಾಸದ ಉದ್ದೇಶ ಗರ್ಭವತಿ ಸಮಯದಲ್ಲಿ ರಕ್ತಸಾರ ಸೀಸಗಳ ಹಂತದಲ್ಲಿ ಆಗುವ ಏರಿಳಿತಗಳು, ಎಲ್ಲ ಪೇಶಂಟಗಳು ಎಲ್ಲ ಪೇಶಂಟುಗಳಲ್ಲಿ ಸದರೀ ಗುಣಲಕ್ಷಣಗಳನ್ನು ಹೊಂದಿದವರು ಮಾತ್ರ ಇದರಲ್ಲಿ 1 ವರ್ಷ ಅವಧಿಯವರೆಗೆ ಭಾಗವಹಿಸಲು ವಿನಂತಿಸಿದೆ.

ಪದ್ಧತಿ ಹಾಗೂ ಚಿಕಿತ್ಸೆ :

ನೀವು ಇಚ್ಛಿಸಿದಲ್ಲಿ ರಕ್ತಸಾರ ಸೀಸದ ಪ್ರಮಾಣವನ್ನು ತಮ್ಮ ರಕ್ತ ನಾಳದ ಮೂಲಕ 2 ಎಮ್.ಎಲ್. ರಕ್ತ ಪ್ರತಿಯೊಂದು ಪೇಶಂಟಿನಿಂದ 3 ಸಾರೆ ಇ.ಡಿ.ಟಿ.ಎ. ಬಲ್ಬನಲ್ಲಿ ತೆಗೆದುಕೊಂಡು ಅದನ್ನು ಅನಾಲಿಸಿಸ್ ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಡಾ : ಪ್ರಭಾಕರ ಕೋರೆ ಟ್ರಸ್ಟ ಹಾಸ್ಪಿಟಲ್ ಹಾಗೂ ಎಮ್.ಆರ್.ಸಿ. ಯ ಬಾಯೋಕೆಮೆಸ್ಟ್ರಿ ವಿಭಾಗದಲ್ಲಿ ಮಾಡಲಾಗುವದು.

ಹೊಣೆಗಾರಿಕೆ ಹಾಗೂ ಲಾಭಗಳು :

ಈ ಪದ್ಧತಿಯಲ್ಲಿ ತಮಗೆ ಆ ಸೌಲಭ್ಯ ಎನಿಸಿದಲ್ಲಿ ಸ್ವಲ್ಪ ಹಣ ದೊರೆಯುವದು ಅಂದರೆ - ಸಣ್ಣ ನೋವು ರಕ್ತನಾಳದಿಂದ ರಕ್ತವನ್ನು ಪಡೆಯುವಾಗ ಅನಿಸಿದರೆ ಮಾತ್ರ. ಆದರೆ ತಮ್ಮ ಸುರಕ್ಷತೆಗಾಗಿ ಆವಶ್ಯವಿದ್ದ ಎಲ್ಲ ಮುಂಜಾಗ್ರತಾ ಕ್ರಮಗಳನ್ನು ಕೈಕೊಳ್ಳಲಾಗುವದು. ತಾವು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೋಳ್ಳುವದರ ಪರಿಣಾಮ ಶರೀರ ಆರೋಗ್ಯದ ಬಗ್ಗೆ ಕಾಳಜೀ ತೆಗೆದುಕೊಳ್ಳಲು ಸಹಾಯವಾಗುವದು ಬೇರೆ ಬೇರೆ ಲಕ್ಷಣಗಳಿಗೆ ಅದರಂತೆ ಚಿಕಿತ್ಸೆ ನೀಡಲು ಸಹಾಯವಾಗುವದು ತಿಳಿದುಕೊಳ್ಳಲೂ ಸಹ ಅನುಕೂಲವಾಗುವದು ಜೊತೆಗೆ ಆರೋಗ್ಯ ಸುಧಾರಿಸುವದು.

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ಐಚ್ಛಿಕ :

ತಮಗೆ ಇದರಲ್ಲಿ ಭಾಗವಹಿಸುವದು ಬೇಡವಾದಲ್ಲಿ ಸಹ ಸಾಮಾನ್ಯವಾಗಿ ದೊರೆಯುವ ಚಿಕಿತ್ಸಾ ದೊರೆಯುವದು.

ಖಾಸಗೀತನ ಹಾಗೂ ಗೌಪ್ಯತೆ :

ತಮ್ಮ ಖಾಸಗಿತನ ಗೌರವಿಸುತ್ತೇವೆ. ಸದರೀ ಅಭ್ಯಾಸದಲ್ಲಿ ತಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ವಿಷಯಗಳ ಗೌಪ್ಯತೆ ಕಾಯ್ದುಕೊಳ್ಳುತೆಗೆವೆ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗ ಪಡಿಸುವದಿಲ್ಲ.

ಸಂಸ್ಥೆ ಜೊತೆಗಿನ ಸಂಬಂಧ ಕುರಿತು.

ಈ ಯೋಜನೆಯಲ್ಲ ಪಾಲ್ಗೊಂಡಾಗ ಪೇಶಂಟಗಳು ಗಾಯಗಳಿಂದ ಬಳಲಿದಲ್ಲಿ ಕರ್ನಾಟಕ ಸರಕಾರದ ಕಾನೂನಿನ,ತೆ ಅಂತಹ ಪೇಶಂಟುಗಳಿಗೆ ಜೆ. ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಹಲವು ಸೌಲಭ್ಯಗಳನ್ನು ನೀಡುವದು ಮತ್ತು ವೈದ್ಯಕೀಯವಾಗಿಯೂ ಗಮನಿಸಲಾಗುವದು. ಒಂದು ವೇಳೆ ತಮಗೆ ಈ ಯೋಜನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಂಡಿದ್ದರಿಂದ ಗಾಯಗಳೇನಾದರೂ ಆದರೆ ಡಾ : ಅಥವಾ

ಇವರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು ಅತ್ಯವಸರದ ಪ್ರಸಂಗದಲ್ಲಿ ಕೆ.ಎಲ್.ಇ. ಡಾ : ಪ್ರಭಾಕರ ಕೋರೆ ಹಾಸ್ಪಿಟಲ್ ಹಾಗೂ ಎಮ್.ಆರ್.ಸಿ. ಫೋನ ನಂ. 0831-2473777.

ಹಣಕಾಸಿನ ಸೌಲಭ್ಯ :

ನೀವು ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಿದ್ದಕ್ಕೆ ಯಾವುದೇ ರೀತಿಯಿಂದ ಹಣಕಾಸಿನ ಸೌಲಭ್ಯ ದೊರೆಯುವದಿಲ್ಲ.

ಪರಿಣಾಮ ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ :

ಈ ಸಂಶೋಧನಾ ಅಭ್ಯಾಸದ ಪರಿಣಾಮವನ್ನು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕೆ ಅಥವಾ ವೈಜ್ಞಾನಿಕ ಸಮೂಹಗಳಲ್ಲಿ ಹಾಜರ ಪಡಿಸಲಾಗುವದು. ಯಾವುದೇ ಪರಿಸ್ಥಿತಿಯಲ್ಲಿ ತಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವದಿಲ್ಲ.

ಸ್ವ-ಇಚ್ಛೆಯಿಂದ ಪಾಲ್ಗೋಳ್ಳುವಿಕೆ -

ನೀವು ಈ ಸಂಶೋಧನಾ ಅಭ್ಯಾಸದಲ್ಲಿ ಪಾಲ್ಗೋಳ್ಳುವದು ಐಚ್ಛಿಕವಾದದ್ದು ನಿಮ್ಮ ನಿರ್ಣಯ ಪಾಲ್ಗೋಳ್ಳಬಾರದೆಂದಿಲ್ಲ ನಿಮ್ಮ ಈಗಿನ ಚಿಕಿತ್ಸೆಗಳಿಗೆ ಹಾಗೂ ಡಾಕ್ಟರುಗಳಿಂದ ಆಸ್ಪತ್ರೆಯಿಂದ ಯಾರಿಂದಲೂ ಸಧ್ಯದ ಹಾಗೂ ಭವಿಷ್ಯ ದೊರೆಯುವ ಚಿಕಿತ್ಸೆಗಳ ಮೇಲೆ ಯಾವ ಅಡ್ಡ ಪರಿಣಾಮಗಳಾಗುವದಿಲ್ಲ ಒಂದು ವೇಳೆ ತಮಗೆ ಸದರೀ ಅಭ್ಯಾಸದ ಕ್ರಮದ ಕುರಿತು ಹೆಚ್ಚಿನ ವಿವರ ಬೇಕಾದಲ್ಲಿ ಡಾ : ಗಂಗಾ ಪಿಲ್ಲೈ ಚೇರಪರಸನ್ ಎಥಿಕಲ್ ಕಮೀಟಿ ಜೆ. ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಬೆಳಗಾವ ಫೋನ ನಂ. 0831- 2473777 ಮೂಲಕ ಸಂಪರ್ಕಿಸಬಹುದು.

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ಸಮ್ಮತಿ ಹೇಳಿಕೆ

ಐ. ಡಿ. ನಂ.

ನಾನು ಶ್ರೀ / ಕು. / ಶ್ರೀಮತಿ _____

ಈ ಅಭ್ಯಾಸದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಸ್ವ-ಇಚ್ಛೆಯಿಂದ ಸಮ್ಮತಿ ಕೊಟ್ಟಿದ್ದೇನೆ. ನಾನು ಸಮ್ಮತಿ ಪತ್ರ ಓದಿದ್ದೇನೆ / ಓಡಿ ನನ್ನ ಮಾತೃ ಭಾಷೆಯಲ್ಲಿ ಹೇಳಿದ್ದು ನಾನು ಈ ಅಭ್ಯಾಸದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪಿಕೊಂಡಿದ್ದೇನೆ. ನನಗೆ ಅಭ್ಯಾಸದ ಬಗ್ಗೆ ಎಲ್ಲ ಮಾಹಿತಿಗಳನ್ನು ಕೊಟ್ಟಿದ್ದು ಅವುಗಳನ್ನು ನಾನು ತಿಳಿದುಕೊಂಡಿದ್ದೇನೆ. ನನಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ಕೊಟ್ಟಿದ್ದು. ನಾನು ಸಮರ್ಪಕ ಉತ್ತರಗಳನ್ನು ಪಡೆದಿದ್ದೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು

ಭಾಗವಹಿಸುವವರ ಸಹಿ ಅಥವಾ ಎಡಗೈ ಹೆಬ್ಬಟ್ಟಿನ ಗುರುತು.

ಸಾಕ್ಷಿ ಹೆಸರು

ಸಾಕ್ಷಿ ಸಹಿ

ಇನ್‌ವೆಸ್ಟಿಗೇಟರ ಸಹಿ,

ದಿನಾಂಕ :

(ಒಂದು ವೇಳೆ ಭಾಗವಹಿಸುವವರು 18 ವರ್ಷಗಳ ಒಳಗೆ ಇದ್ದಲ್ಲಿ ಅವರ ಪೋಷಕರು ಅಥವಾ ಭಾಗವಹಿಸುವವರು ಫಾರ್ಮ ಮೇಲೆ ಸಹಿ ಮಾಡಬೇಕು)

अनुबंध – ५
जवाब सम्मती फॉर्म

आय्. डी. नं.

गर्भावस्थेत रक्तसार सिस् (लेड) समास्थरा बाबत गटां समोर अभ्यास

सदर अभ्यास क्रम डॉ. जी. निकीला, पोस्ट ग्रॅज्युयेट विद्यार्थीनी, एम्. एस्. अब्स्टेट्रीक्स व गॅनाकालॉजी मध्ये डॉ. एम्. बी. बेल्लद प्रिन्सिपाल ईनवेस्टीगेटर) ओबीजी विभागाचे प्रोफेसर जे. एन्. मेडीकल् कॉलेज बेळगाव यांच्या मार्गदर्शना खाली करीत आहेत.

मा. महोदया / महीलां आपणास या अभ्यास क्रमात भाग घेणेस योग्य असल्यामुळे आपणास आमंत्रीत करीत आहे. या वेळेत सध्याचे आपले तकरार सविवर विचारण्यात येईल.

अभ्यासाचे उद्देश : या अभ्यास क्रमाचे उद्देश गर्भावस्थाच्या वेळेत रक्तसार सिस (लेड) टप्यात होणारे बदलाव, सर्व पेशंटानी सर्व पेशंटामध्ये सदर गुणलक्षणे असलेले मात्र या १ वर्षाच्या काळावधी पर्यंत भाग घेणेस विनंती आहे.

पद्धत व चिकीत्सा :

आपण ईच्छुक असल्यास रक्तसार सिस (लेड) प्रमाण आपल्या रक्तनाळ मार्फत २ एम्.एल्. रक्त प्रत्येकी प्रशंट कडून घेऊन ३ वेळा इ.डी.टी.ए. बल्ब मध्ये घेऊन ते ॲनालेसीस् जे.एन्. मेडीकल कॉलेज, डॉ. प्रभाकर कोरे ट्रस्ट हॉस्पिटल व एम्.आर्.सी. चे बायोकेमेस्ट्रि विभागात करण्यात येईल.

जबाबदारी व लाभ :

या पद्धतीत आपणास ही सवलतीत थोडक्यात रक्कम मिळेल म्हणजे – लहान प्रकारचे जखम रक्तनाळातुन रक्त घेते वेळी वाटल्यास मात्र. परंतु आपल्या सुरक्षतेसाठी आवश्यक सर्व योग्य क्रम घेण्यात येईल. आपण संशोधनेत भाग घेतल्या मुळे शरीरातील आरोग्याची काळजी घेण्यास सहाय्यक होईल. निर निराळे लक्षण तसेच चिकीत्सा देणेस सहाय्य होईल समझण्यास सुद्धा सहाय्य होईल व सोबत तब्यत सुधारण होईल.

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एच्छिक :

आपणास यात भाग घेणेचे नसल्यास सुद्धा सामान्यतह मिळणारी चिकीत्सा आपणास मिळेल.

खासगी व गोपनीयता :

आपले खासगीपणाचा आम्ही सम्मान करतो. सदर अभ्यास क्रमात आपल्या कडून घेतलेली माहीतीची गोपनीयता राखण्यात येईल. आपली ओळख जाहीर करणार नाहीं.

संस्थे सोबत संबंधी बाबत :

या योजनेत भाग घेतल्यास पेशंट जखम वगैरे पासून त्रास झाल्यास कनॉटक सरकारचे कानून रित्या त्या पेशंटानां जे. एन्. मेडीकल् कॉलेज कांही सवलती देतील व वैद्यकीय तपास सुद्धा करण्यात येईल. जर कदाचीत आपणास या योजनेत भाग घेतल्या मुळे जखम वगैरे झाल्यास डॉ. जी. निकीला फोन. नं. ८७२२५३८४४३ किंवा डॉ. एम्. बी. बेल्लद फोन नं. ९४४८१२४८९३ यानां संपर्क साधावा. एमर्जेन्सि प्रसंगात के.एल्.ई. डॉ. प्रभाकर कोरे हस्पताल व एम्.आर्.सी. फोन नं. ०८३१–२४७३७७७

आर्थीक सहाय्य :

आपण यात भाग घेतल्या बद्दल कोणतेही रित्या आर्थीक सहाय्य मिळणार नाहीं.

परीणाम जाहीर करणेचा अधीकार :

हा संशोधन अभ्यास क्रमाचा परीणाम वैज्ञानीक उद्देशासाठी किंवा वैज्ञानीक समूहा मध्ये सादर करण्यात येईल. कोणतेही परीस्थीतीत आपली ओळख जाहीर करणार नाहीं.

स्व–ईच्छेने भाग घेणे :

आपण या संशोधन अभ्यास क्रमात भाग घेणेस स्वतंत्र आहात आपला निर्णय भाग न घेणे असे नाहीं. आपल्या सध्याच्या चिकीत्सा बाबत व डॉक्टरानां हस्पताल वतीने कोणत्याही सध्याच्या व भविष्यकाळात मिळणारे चिकीत्सा वर कोणतेही दुष्परीणाम होणार नाहीं. जर कदाचीत आपणास सदर अभ्यास क्रमा बाबत अतीरीक्त माहीतीची आवश्यकता असल्यास डॉ. गंगा चेअरपरस् एथीकल् कमीटी जे. एन्. मेडीकल कॉलेज बेळगांव फोन नं. ०८३१–२४७३७७७ मार्फत संपर्क साधावा.

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सम्मती जवाब

आय्. डी. नं.

मी, श्री / कु / श्रीमती _____

या अभ्यास क्रमात भाग घेणेस स्व-ईच्छेने सम्मती दिली आहे. मी, सम्मती पत्र वाचून घेतला आहे. / वाचून माझ्या मातृभाषेत कळविले असून मी या अभ्यास क्रमात भाग घेणेस सम्मती दिला आहे. माझ्या अभ्यास बाबत सर्व माहीती दिले असून ते मी समझून घेतले आहे. मला प्रश्न विचारण्यासाठी संधी दिले असून मी समर्पक उत्तर घेतली आहे.

भाग घेणाऱ्यांचे नाव

भाग घणाऱ्यांची सही किंवा अंगठ्याचा ठस्सा

साक्षीदारांचे नाव

साक्षीदारांचे सही

ईनवेस्टीगेटर सही

दिनांक :

(जर कदाचीत भाग घेणाऱ्यानी १८ वर्षा पेक्षा आत असल्यास त्यांचे पालक किंवा भाग घेणाऱ्यानीं फॉर्म वर सही करणे.)

ANNEXURE III - MASTER CHART

						Screening																						Examination												Assessment of haemoglobin			Assessment of Lead			Mid Pregnacy		Late pregnancy	
Serial Number	Screening ID	Identification number	Out Patient Number	Unit	Age (Years)	Gestational age (weeks)	Singleton pregnancy	Parity	Previous LSCS	Maternal medical complications	Complications	Emigration from ambient lead contamination	Point source of lead	Working with lead or living	Lead contaminated cosmetics/food	Emigration from ambient lead contamination	Occupation	Education	Socio economic status	Cycle	Gravida	Para	Living	Abortions	Ayurvedic medications	Habits of alcohol	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Pulse rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Cardiovascular system	Respiratory system	Estimated fetal weight (gms)	Eligibility	Early pregnancy	Mid pregnancy	Late pregnancy	Early pregnancy	Mid pregnancy	Late pregnancy	Gestational hypertension	Preeclampsia	Gestational hypertension	Preeclampsia
1	1	1	2E+06	A	30	12	Y	0	N	N	N	N	N	N	Y	N	PR	G	PC	R	PM	0	0	0	N	N	142	50	25	72	120	72	N	N	2700	Y	11	12	10	1.98	1.45	2.34	N	N	Y	N
2	2	2	3E+06	A	23	12	Y	0	N	N	N	N	N	Y	N	N	AG	IL	WC	R	PM	0	0	0	N	N	152	50	22	80	108	82	N	N	2300	Y	10.2	7.8	6.2	3.31	10.2	25.84	N	Y	N	N
3	3	3	3E+06	A	21	12	Y	0	N	N	N	N	Y	N	N	N	HW	READ	PC	R	PM	0	0	0	N	N	143	52	25	80	126	80	N	N	2400	Y	10	10	7	4.86	3.01	23.9	N	N	N	Y
4	4	4	2E+06	B	20	13	Y	0	N	N	N	N	N	N	Y	N	PR	S	PC	R	PM	0	0	0	N	N	142	46	23	84	106	72	N	N	2400	Y	10.2	9.8	7	3.56	5.47	23.45	N	N	Y	N
5	5	5	3E+06	C	19	13	Y	0	N	N	N	N	N	N	Y	N	PR	S	PC	R	PM	0	0	0	N	N	144	48	23	76	128	74	N	N	2600	Y	9.4	7.4	9.8	3.89	2.25	4.77	N	N	N	N
6	6	6	3E+06	A	22	12	Y	0	N	N	N	N	N	N	Y	N	HW	P	PC	R	PM	0	0	0	N	N	144	50	24	80	124	70	N	N	2800	Y	10	12.2	9.6	2.4	0.92	1.11	N	N	N	Y
7	7	7	3E+06	C	20	12	Y	1	N	N	N	N	Y	Y	N	N	L	IL	YC	R	G2	1	1	0	N	N	144	52	25	78	112	70	N	N	2700	Y	10.4	10	9.8	1.84	1.6	2.18	N	N	N	N
8	8	8	3E+06	C	22	13	Y	2	Y	N	N	N	N	Y	N	N	AG	WRITE	WC	R	G3	2	2	0	N	N	144	54	26	82	124	78	N	N	2600	Y	11.2	10.2	6.2	0.76	2.83	22.85	N	N	Y	N
9	9	9	3E+06	A	19	12	Y	0	Y	N	N	N	N	Y	N	N	AG	WRITE	WC	R	PM	0	0	0	N	N	152	48	21	84	112	70	N	N	2800	Y	11.8	11	10.6	2.93	3.03	1.96	N	N	N	Y
10	10	10	3E+06	A	20	13	Y	0	N	N	N	N	N	Y	N	N	AG	P	WC	R	PM	0	0	0	N	N	144	46	22	78	112	72	N	N	2700	Y	10.4	9.8	9	2.08	2.64	4.97	N	N	N	Y
11	11	11	3E+06	A	19	13	Y	0	N	N	N	N	N	Y	N	N	AG	WRITE	WC	R	PM	0	0	0	N	N	152	54	23	80	114	70	N	N	2800	Y	10.2	10	10	3.83	5.3	2.07	Y	N	N	N
12	12	12	3E+06	A	22	12	Y	1	N	N	N	N	Y	Y	N	N	AG	IL	WC	R	G2	1	1	0	N	N	146	54	25	84	112	70	N	N	2400	Y	10	10.7	8.2	6.09	0.32	11.41	N	N	N	N
13	13	13	3E+06	A	22	13	Y	2	Y	N	N	N	N	N	Y	N	PR	G	PC	R	G3	2	2	0	N	N	144	46	22	82	106	74	N	N	2600	Y	10.2	9.6	10.2	3.66	5.24	2.06	N	Y	N	N
14	14	14	3E+06	D	21	12	Y	1	Y	N	N	N	N	Y	N	N	AG	WRITE	WC	R	G2	1	1	0	N	N	142	52	26	84	112	68	N	N	2800	Y	10.2	10	11	3.04	2.74	1.36	N	N	N	N
15	15	15	276300	B	28	12	Y	0	N	N	N	N	N	N	Y	N	PR	S	PC	R	PM	0	0	0	N	N	146	46	22	82	114	72	N	N	2700	Y	10	9.2	10	1.16	8.82	4.5	N	Y	N	N
16	16	16	3E+06	A	19	13	Y	0	N	N	N	N	N	Y	N	N	HW	READ	PC	R	PM	0	0	0	N	N	150	48	21	84	112	86	N	N	2800	Y	10.2	11	8.3	3.92	0.26	2.81	N	N	N	N
17	17	17	3E+06	A	21	13	Y	0	N	N	N	N	N	Y	Y	N	HW	S	PC	R	PM	0	0	0	N	N	146	52	24	82	128	74	N	N	2600	Y	11.1	10.8	7.2	1.06	1.92	4.43	N	N	N	N
18	18	18	3E+06	B	19	13	Y	1	N	N	N	N	N	Y	Y	N	HW	P	PC	R	G2	1	1	0	N	N	152	48	21	76	118	74	N	N	2200	Y	10.6	8.4	7	2.16	11.3	20.1	Y	N	N	N
19	19	19	3E+06	A	24	13	Y	0	N	N	N	N	N	N	N	N	PR	IL	PC	R	PM	0	0	0	N	N	144	52	25	82	124	72	N	N	2400	Y	12.4	10	8.2	7.8	3.88	11.47	Y	N	N	N
20	20	20	3E+06	A	20	13	Y	0	N	N	N	N	Y	N	N	N	AG	P	WC	R	PM	0	0	0	N	N	142	54	27	80	118	74	N	N	2600	Y	11.8	9.2	7.4	1.19	2.4	10.23	N	N	Y	N
21	21	21	3E+06	C	21	13	Y	0	N	N	N	N	Y	Y	Y	N	L	IL	WC	R	PM	0	0	0	N	N	146	54	25	86	114	70	N	N	2400	Y	11.2	8.8	7.4	1.54	8.13	20.1	N	N	N	N
22	22	22	3E+06	A	30	12	Y	1	Y	N	N	N	N	N	N	N	L	IL	WC	R	G2	1	1	0	N	N	142	46	23	84	114	70	N	N	2600	Y	10.6	8.2	8.6	3.43	17.2	24.25	N	N	N	N

ANNEXURE III - MASTER CHART

						Screening																				Examination										Assessment of haemoglobin			Assessment of Lead			Mid Pregnacy		Late pregnancy	
Serial Number	Screening ID	Identification number	Out Patient Number	Unit	Age (Years)	Gestational age (weeks)	Singleton pregnancy	Parity	Previous LSCS	Maternal medical complications	Complications	Emigration from ambient lead contamination	Point source of lead	Working with lead or living	Lead contaminated cosmetics/food	Emigration from ambient lead contamination	Occupation	Education	Socio economic status	Cycle	Gravida	Para	Living	Abortions	Ayurvedic medications	Habits of alcohol	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Pulse rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Cardiovascular system	Respiratory system	Estimated fetal weight (gms)	Eligibility	Early pregnancy	Mid pregnancy	Late pregnancy	Early pregnancy	Mid pregnancy	Late pregnancy	Gestational hypertension	Preeclampsia	Gestational hypertension	Preeclampsia
23	23	23	3E+06	A	20	13	Y	0	N	N	N	N	N	Y	Y	N	HW	READ	PC	R	PM	0	0	0	N	N	152	46	20	82	112	70	N	N	2700	Y	12.2	11	11.2	1.24	1.91	1.6	N	Y	N	N
24	24	24	3E+06	A	20	13	Y	0	N	N	N	N	N	Y	Y	N	AG	P	WC	R	PM	0	0	0	N	N	144	52	25	76	112	82	N	N	2600	Y	11	10	9.6	1.54	1.7	5.53	N	N	N	Y
25	25	25	3E+06	B	25	12	Y	0	N	N	N	N	N	Y	Y	N	L	IL	WC	R	PM	0	0	0	N	N	152	56	24	84	106	82	N	N	2700	Y	10	11.7	10.2	5.25	1.63	2.74	N	Y	N	N
26	26	26	3E+06	B	25	11	Y	1	N	N	N	N	N	Y	Y	N	L	IL	WC	R	G2	1	1	0	N	N	152	46	20	72	108	72	N	N	2700	Y	11.4	12	8.2	2.08	0.38	2.36	Y	N	N	N
27	27	27	2E+06	C	24	10	Y	0	N	N	N	N	N	N	N	N	AG	WRITE	WC	R	PM	0	0	0	N	N	144	52	25	80	108	72	N	N	2800	Y	11	7.2	9.6	2.46	2.94	1.11	Y	N	N	N
28	28	28	3E+06	C	22	14	Y	2	Y	N	N	N	N	N	N	N	AG	IL	WC	R	G3	2	2		N	N	144	42	20	84	112	70	N	N	2800	Y	10	11.7	9.8	4.69	2.34	4.85	N	Y	N	N
29	29	29	3E+06	A	19	13	Y	0	N	N	N	N	N	Y	Y	N	HW	P	WC	R	PM	0	0	0	N	N	142	50	25	84	110	72	N	N	2700	Y	10.2	10	8.9	3.05	1.17	12.93	N	N	N	Y
30	30	30	1E+06	A	26	13	Y	0	N	N	N	N	N	Y	Y	N	HW	S	WC	R	PM	0	0	0	N	N	142	50	25	84	112	80	N	N	2600	Y	10	10	9.2	1.17	1.24	5.27	N	N	Y	N
31	31	31	3E+06	A	20	13	Y	0	N	N	N	N	N	Y	Y	N	HW	P	PC	R	PM	0	0	0	N	N	144	52	25	78	122	70	N	N	2700	Y	12	9.3	8.9	2.19	4.73	5.17	N	N	N	Y
32	32	32	3E+06	B	22	13	Y	0	N	N	N	N	N	Y	Y	N	AG	WRITE	WC	R	PM	0	0	0	N	N	144	52	25	76	114	72	N	N	2800	Y	11	10.2	9.2	1.79	4.12	5.17	N	N	N	N

T1	T2	T3	T1	T2	T3
11	12	10	1.98	1.45	2.34
10.2	7.8	6.2	3.31	10.24	25.84
10	10	7	4.86	3.01	23.9
10.2	9.8	7	3.56	5.47	23.45
9.4	7.4	9.8	3.89	2.25	4.77
10	12.2	9.6	2.4	0.921	1.11
10.4	10	9.8	1.84	1.6	2.18
11.2	10.2	6.2	0.76	2.83	22.85
11.8	11	10.6	2.93	3.03	1.96
10.4	9.8	9	2.08	2.64	4.97
10.2	10	10	3.83	5.3	2.07
10	10.7	8.2	6.09	0.32	11.41
10.2	9.6	10.2	3.66	5.24	2.06
10.2	10	11	3.04	2.74	1.36
10	9.2	10	1.16	8.82	4.5
10.2	11	8.3	3.92	0.26	2.81
11.1	10.8	7.2	1.06	1.92	4.43
10.6	8.4	7	2.16	11.31	20.1
12.4	10	8.2	7.8	3.88	11.47
11.8	9.2	7.4	1.19	2.4	10.23
11.2	8.8	7.4	1.54	8.13	20.1
10.6	8.2	8.6	3.43	17.15	24.25
12.2	11	11.2	1.24	1.91	1.6
11	10	9.6	1.54	1.7	5.53
10	11.7	10.2	5.25	1.63	2.74
11.4	12	8.2	2.08	0.38	2.36
11	7.2	9.6	2.46	2.94	1.11
10	11.7	9.8	4.69	2.34	4.85
10.2	10	8.9	3.05	1.17	12.93
10	10	9.2	1.17	1.24	5.27
12	9.3	8.9	2.19	4.73	5.17
11	10.2	9.2	1.79	4.12	5.17
10.7	10.0	8.9	2.9	3.8	8.6
0.8	1.3	1.4	1.6	3.7	8.3
10.4	10.0	9.1	2.4	2.7	4.9
9.4	7.2	6.2	0.8	0.3	1.1
12.4	12.2	11.2	7.8	17.2	25.8

T1

N	ML
1.98	3.31
0.76	4.86
2.93	3.56
1.06	2.4
7.8	1.84
1.19	2.08
1.54	3.83
1.24	6.09
1.54	3.66
2.08	3.04
2.46	1.16
2.19	3.92
1.79	2.16
	3.43
	5.25
	4.69
	3.05
	1.17
2.2	3.3
1.8	1.4

Anova: Single Factor

SUMMARY

Groups	*Count*
Column 1	13
Column 2	18
Column 3	1

ANOVA

Source of Varia	*SS*
Between G	10.3442
Within Grou	70.18492

Total	80.52912

	T2			T1		
MD	N	ML	MD	N	ML	MD
3.89	1.45	3.01	10.24	1.36	2.34	23.9
	0.921	1.6	5.47	1.6	1.96	23.45
	3.03	2.83	2.25		2.07	4.77
	0.26	5.3	2.64		2.06	1.11
	1.91	0.32	5.24		4.5	2.18
	1.63	2.74	8.82		2.74	4.97
	0.38	1.92	11.31			11.41
	2.34	3.88	2.4			2.81
		1.7	8.13			4.43
		1.17	17.15			20.1
		1.24	2.94			11.47
		4.12	4.73			10.23
						20.1
						24.25
						5.53
						2.36
						1.11
						4.85
						12.93
						5.27
						5.17
						5.17

3.9	1.5	2.5	6.8	1.5	2.6	9.4
#DIV/0!	1.0	1.4	4.5	0.2	1.0	7.9

Anova: Single Factor

Sum	*Average*	*Variance*
28.56	2.196923	3.198123
59.5	3.305556	1.871026
3.89	3.89	#DIV/0!

SUMMARY

Groups	*Count*	*Sum*
Column 1	2	2.96
Column 2	6	15.67
Column 3	22	207.57
Column 4	2	48.69

df	*MS*	*F*	*P-value*	*F crit*
2	5.1721	2.137082	0.136213	3.327654
29	2.42017			

ANOVA

Source of Varia	*SS*	*df*
Between G	827.6956	3
Within Grou	1324.631	28

		31
Total	2152.327	31

Anova: Single Factor

SUMMARY

Groups	*Count*	*Sum*	*Average*	*Variance*
Column 1	8	11.921	1.490125	0.908637
Column 2	12	29.83	2.485833	2.062572
Column 3	12	81.32	6.776667	20.53582

ANOVA

Source of Varia	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Between G	169.6675	2	84.83375	9.649924	0.000613	3.327654
Within Grou	254.9428	29	8.791132			
Total	424.6103	31				

SV

25.84

22.85

24.3

2.1

Average	*Variance*
1.48	0.0288
2.611667	0.935697
9.435	62.64066
24.345	4.47005

MS	*F*	*P-value*	*F crit*
275.8985	5.831932	0.003159	2.946685
47.30826			

ANNEXURE I – ETHICAL CLEARANCE

K.L.E.SOCIETY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELGAUM-590010 (KARNATAKA-INDIA)
(Affiliated to KLE University, Belgaum)

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Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 895 **Date: 31/10/2012**

To,

Dr. G. Nikila Kantha,
PG student in OBG,
J.N.Medical College,
BELGAUM.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled "**SERUM LEAD LEVELS DURING PREGNANCY – A CROSS SECTIONAL STUDY**" is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr.Hema Dhumale)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belgaum.

(Dr.Ganga Pilli)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belgaum.