THE PREVALENCE OF HYPOCALCEMIA IN PREGNANCY AND PEURPERIUM IN NORTHWESTERN NIGERIA.

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ABSTRACT

Background: The occurrence of hypocalcaemia in pregnancy and puerperium is not often reported in literature even though calcium supplementation is not routine in Nigeria. A finding of a relatively increased number of cases seen with hypocalcaemia in pregnancy and following childbirth necessitated a review of records to find risk factors and other associations.

Objectives: To evaluate the prevalence of hypocalcaemia and identify risk factors for its occurrence among women presenting with carpopedal spasm in pregnancy and puerperium in our area of practice.

Study Design Setting And Subjects: This is a five year retrospective study involving the retrieval of patient records presenting with carpopedal spasms from 1st June 2010 to 30th June 2015 at Rasheed Shekoni specialist hospital, Duste Jigawa State Nigeria

Method: The total number of deliveries within review period as well as the case records of women presenting with features of hypocalcaemia were retrieved manually and relevant data extracted including demographics, parity and duration of symptoms as well as laboratory indices. Analysis was done using relevant statistical software.

Result: There were a total of 3989 deliveries with 52 presenting with carpopedal spasm giving a prevalence of 1.3%. The average age was 26.6 years ± 6.6 years and parity of 4.1±2.9. The mean serum calcium level was 1.72mmol/l ± 0.32 mmol/l. There was a negative correlation between parity and serum calcium levels (ρ= -0.296, p=0.151)

Conclusion: The prevalence of hypocalcaemia at 1.3% was higher than earlier reports elsewhere. High parity was found to be a likely risk factor. We suggest that calcium supplementation should be considered for these groups of women who in addition have other risk factors in the antenatal period.

Keywords: Parathormone, 1, 25-dihydroxyvitamin D, Hypocalcemia, Parity

INTRODUCTION

Pregnancy and lactation is a physiological state of altered calcium metabolism among women. The growing fetus derives calcium from maternal stores and circulation for skeletal development. The total requirement of calcium for fetal development has

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been reported to range between 25-30g with an increase from 2-3mg/day in first trimester to 250mg/day in the final trimester.[1] This demand, during pregnancy, is met by increased intestinal reabsorption of calcium under the influence of 1,25-$(OH)_2$ D. The hormone, which is under the regulation of parathyroid hormone (PTH), increases in the course of pregnancy by 50-100% while PTH itself remains the same or decreases[2,3,4]. There is also increased urinary excretion of calcium probably due to increased GFR and preload of calcium[5,6]. The net effect is a fall in serum total calcium as pregnancy progresses due to above factors in addition to plasma volume expansion and hypoalbuminaemia[2]. However, the serum ionized calcium levels remain unchanged. Serum calcium levels during lactation are maintained mainly by PTH via bone resorbption with a fall in intestinal reabsorption and decreased urinary calcium excretion.In the setting above, a woman who goes into pregnancy with borderline or depleted serum calcium levels in addition to lactation in the postpartum period is prone to developing hypocalcaemia.

In our area of practice, several cases of carpopedal spasm due to hypocalcaemia were seen in the puerperium within the period under review. Studies on this clinical entity were few and so we report our findings from the setting of a specialist hospital in Duste, Jigawa state of north western Nigeria.

MATERIALS AND METHOD

This is a five year cross sectional retrospective study of medical records of patients who presented with carpopedal spasm from 2010 to 2015 at RasheedShekoni Specialist Hospital in Northern Nigeria.A total of 52 cases were recorded in both pregnancy and puerperium of which only 35 case notes were retrieved giving a retrieval rate of 67%. Their socio demographic data, interval between delivery and presentation, clinical presentation, co-morbidities as well as results of laboratory investigations were obtained. The treatment, duration of symptoms and reoccurrences were all documented. The reference serum calcium level was 2.25-2.75 mmol/L while that of serum albumin was 32.0-50.0g/dl. The PCV was used to identify anaemic patients, with those having <30% considered anaemic. The corrected serum calcium level was obtained by adding the measured serum calcium and the product the difference between patients' serum albumin levels and the average reference value of 41g/dl,by a constant factor of 0.02.Values below 2.25mmol/L were regarded as hypocalcaemia. The data was analysed using IBM® SPSS® Statistics version 20

RESULTS

The total deliveries within the study period were 3989, an average of 998 per year. There were 52 cases with symptoms suggestive of hypocalcaemia, giving a prevalence rate of 1.3%.

77.1% were illiterate while 57.1% had no antenatal care. Only 33.3% of the women who presented in pregnancy were attending antenatal clinic. (Table 1)

The mean age at presentation was 26.6 years ± 6.6years and average parity was 4.1±2.9. (Table 2)

Fourty percent of theparturients were multiparous while 37.1% had delivered 5 or more times. Most of the multiparous women were aged between 20-24years (50%) while majorities (46.2%) of the grandmutiparous women were between 30-34 years old. (Table 3)

Although 9(25.7%) presented while pregnant, most of them were in the puerperium (Table 4)

The most common presentation was carpopedal spasm only (37.1%), while 25.7% had hypertension as a cormidity, 17.1% hypokalaemia in addition to fever and gastrointestinal symptoms (Table 4)

The average onset of symptoms-to-presentation interval was 6 days while delivery-presentation

interval was averagely 19 days± 10 days with most presenting14 days or more after delivery(31.4%). (Table 4)

The averageserum calcium was 1.75±0.32 mmol/L, mean PCV 27.6±7.3% and mean serum albumin of 36.27±10.61mg/dl. (Table 2)

Therewas a negative correlation between parity and serum calcium levels (ρ=-0.296,p=0.151). This was stronger for multiparity(ρ=-0.405, p=0.246) thangrandmultiparity

(ρ=-0.290,p=0.499) although both were not statistically significant. (Figure 1)

The correlation between serum calcium and PCV of both booked and unbooked patients was weakly positive but had no statistical significance (booked ρ=0.292,p=0.384; unbooked ρ=0.192,p=0.620).

The duration of symptoms was found to negatively correlate with levels of serum calcium but was only statistically significant after 5 or more deliveries (ρ=-0.698, p=0.037).

There was a strong positive correlation between serum calcium and delivery-presentation interval in those whose symptoms lasted 1-7 days but was not found to be of any statistical significance(N=10, ρ=1.000, p=0.494).

DISCUSSION

Calcium homeostasis is maintained by several hormones in humans. The most studied been parathyroid hormone (PTH) and1, 25-dihydroxyvitamin D(1,25-$(OH)_2$ D). The mechanism of this control is better understood outside pregnancy due to the physiological changes that occur in pregnancy. The calcium requirement in pregnancy increases in the third trimester[1,2,3,4]. This is needed for skeletal development of the growing fetus. The increased demand is met by enhanced intestinal reabsorption of dietary calcium mediated by 1,25-$(OH)_2$ D. While this increases by two-to-three fold in the prepregnant state, the levels of PTH, a major regulator outside pregnancy, remains relatively low to normal. In the puerperium and lactating mothers, the main regulator of calcium homeostasis is PTH, with minimal contribution of 1, 25-$(OH)_2$ D.

The average age of our study population was26.6 years with majority (77.1%) of the multiparous women aged between 20-34 years.This was similar to 79.4% reported among women with postpartum hypocalcaemia in Maiduguri[7]. Though 40.0% of women were multiparous and 37.1% grand multiparous, there was only significant association between parity and hypocalcaemia for women with 2-4 deliveries. The significance was not evident for 5 or more deliveries. This however shows that the level of calcium depletion and duration of symptoms increases with parity. There was a similar finding by workers in Maiduguri[7].

Most of our patients presented in puerperium with only nine cases of hypocalcaemia in pregnancy.Several studies have shown that calcium levels are fairly maintained in pregnancy by 1,25-dihydroxyvitamin D via increased intestinal reabsorption of calcium as opposed to bone demineralisation in the postpartum period in both lactating and non-lactating women[8,9] A study by Sanchez et.al from Maiduguri among teenage pregnant mothers revealed that the level of both 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D are maintained within normal limits in both second and third trimesters of pregnancy[10]. However, the parity of the participants was not stated. In case control study of 160 pregnant Iranian women, 81% were found to bedeficient of vitamin D[11]. There are other studies that also show that there is increase prevalence of vitamin D deficiency in pregnant women in developing countries[12,13].

Poor nutritional status of pregnant women, measured by prealbumin levels, was found to be associated withhypovitaminosis D andhypocalcemia in pregnancy. This is more pronounced in the 3rd

trimester of pregnancy[14]. Proper Vitamin D supplementation in pregnancy, therefore improved the calcium levels in susceptible women and their neonates[11]

The level of postpartum calcium is influenced more by PTH, pre delivery calcium levels and decreased renal excretion of calcium than 1, 25-$(OH)_2$ D and intestinal reabsorption[14]. A deficiency of this hormone in puerperium and lactation can therefore result in persistent hypocalcaemia[15].In this study however, the level of PTH was not measured but the average serum albumin levels were within normal limits suggesting a probable influence of PTH in postpartum hypocalcaemia.

Majority of the patients(37.1%) presenting had no additional morbidity, only 25.7% had hypertension, 20% had hypokalaemia and5.7% had eclampsia.

The study revealed hypertension as the commonest morbidity associated with low calcium levels. The evidence of an association between low calcium and pre-eclampsia is well documented with benefits from supplementation in prevention of pregnancy induced hypertension in high risk patients[16,17]

Although a previous study found a linear relationship between PCV and calcium levels[7], it failed to test the strength of this relationship (R^2 linear=0.292, p=0.001). Our findings of a weakly positive correlation between PCV and serum calcium levels were not statistically significant in both patients who had antenatal care and haematinics and those who did not.

All of our patients had their calcium levels corrected parentally with calcium gluconate and maintained on oral calcium carbonate with improvement in symptoms and subsequent discharge. Hypokalaemia and hypertension were treated too.

In conclusion, the high prevalence of hypocalcaemia presenting in women following delivery in our environment might be explained by their poor nutritional status, lack of calcium supplementation in pregnancy and high parity. Though current National obstetric protocol does not include routine vitamin D or calcium supplementation in pregnancy, the RCOG guideline recommendssupplementation in women at risk of hypocalcaemia who are pregnant or breast feeding[18]. An identified additional risk factor from this study is high parity.

Further studies to determine the PTH and 1, 25-dihydroxyvitamin D levels following delivery in women with hypocalcaemia will help improve management modalities. These investigations were not available in our facility.

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