Osteoporosis during pregnancy and breastfeeding

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Both generalised and regional osteoporosis are diseases which, exceptionally, are associated with pregnancy and breastfeeding, although, undoubtedly, diseases that are underdiagnosed.

Compensatory physiological mechanisms allow, in the majority of cases, those requirements necessary for the formation and mineralisation of the foetal skeleton and the nutrition of the newborn to be met, overcoming this period without major difficulties. However, some mothers experience bone demineralisation which may become complicated with fractures, and a small group suffers regional demineralisation which temporarily disables them.

The fundamental problem in these situations is the diagnosis since, on the one hand, some of the associated symptoms may be attributed to "normal" problems related to gestation, such as pelvic pain, which are frequently seen in the typical clinical picture during the third quarter of pregnancy, while on the other, there is the impossibility of carrying out diagnostic procedures such as DXA during pregnancy.

In addition, ultrasounds do not help to establish clearly changes in bone mineral density associated with gestation. And markers for bone remodelling do not help either, since during gestation they have significant biases in interpretation due to the haemodilution which occurs, the increase in glomerular filtrate and the placental production of alkaline phosphatase. It should also be added that the ranges of normality during gestation have not been established.

During pregnancy and breastfeeding, compensatory mechanisms occur which meet the needs of the foetus. Calcium is actively transferred through the placenta, especially in the third quarter, with the aim of ossifying the collagen matrix of the foetal skeleton. The blood levels of calcium in the mother are reduced by the haemodilution, and the greater requirements are compensated for essentially by an increase in intestinal absorption, related with an increase in the production of 1,25 (OH)₂D.

Initially it was considered that gestation resulted in a state of hyperthyroidism; however, with advances in technology, it has been possible to establish that levels of PTH are found to be slightly reduced in the first quarter and normal in the other two. Nevertheless, the level of the peptide related with PTH (PTHrP) is found to be raised during gestation. This is a prohormone which produces many peptides with various biological properties. This finding has opened up an exciting field in the study of these molecules, with various sources of production being identified, such as the placenta, myometrium, breast, decidua, amniotic membranes and foetal parathyroids.

In spite of the fact high levels of calcitonin are found in pregnancy, produced by hypertrophied thyroid C cells, and possibly in the breast and placenta, it does not appear to have a significant impact on calcium metabolism during pregnancy.

This is not the case with vitamin D, which increases its blood levels to twice that in non-gestation, possibly related to an increase in globulin transporters produced in gestation, although an increase in blood levels of its free parts has also been found, due more to an increase in production than a reduction in clearing. This increase is independent of the action of PTH and is, principally, due to an increase in the activity of 1α-hydroxylase in the maternal kidney. Estrogens, prolactins, placental lactogen and PTHrP also increase renal enzyme activity. The placenta and
the foetal kidneys may also be additional sources. Vitamin D crosses the placenta and in the foetus reaches levels of around 20% lower than the mother’s.

A question has been raised about what the impact of these adaptive changes would be on the maternal bone. Histomorphometric studies in gestating animals suggest that there are no substantial modifications in the mineral content of bone, nor in its structure.

Some studies whose evaluation of bone mineral density was carried out immediately after birth, or a miscarriage at different times in the gestation period, show results which are variable and difficult to interpret due to the presence of factors which create confusion, such as changes in the composition, weight and volume of the skeleton. Nevertheless, it appears that gestation does not significantly affect the density or resistance of the bone and, in fact, epidemiological studies in the postmenopause have established no relationship between giving birth and the period of breastfeeding and low bone mass and risk of fracture later in life.

During breastfeeding, 400 mg/day is lost due to giving milk. Differently from the way that this happens during gestation, where the main mechanism for the maintenance of calcium homeostasis is an increase in intestinal absorption, the increase in demand is mainly offset by an increase in bone resorption and, partly, by an increase in renal resorption. Both mechanisms are found to be mediated by high levels (some 1,000 times higher than those in gestation) of PTHrP, which is mainly secreted by the breast, and not by PTH, which remains low during breastfeeding. The intestinal absorption of calcium returns to pre-gestation levels after birth.

The increase in prolactin during breastfeeding is associated with a decrease in estrogens which are subject to the increase in PTHrP resulting in an observation of loss of bone mass of around 2-3% per month of breastfeeding. However, these losses are recovered rapidly after the end of breastfeeding, with gains of between 0.5 and 2.0% per month, which means that the recovery is total after a period of 2-6 months. It has been noted that supplementing calcium during breastfeeding does not prevent the bone loss which occurs during this period.

In view of the above, it can be stated that in a healthy woman pregnancy and breastfeeding induce changes in the bone, but that they do not, however, effect the health of the bone over the long term.

It is not known why in some women osteoporosis occurs during pregnancy. The rareness of this association means that the series of cases published is short, and therefore that no patterns of risk which may be associated with osteoporosis during gestation have been established, although primiparity, maternal history of fracture, the use of corticoids and low weight have all been indicated.

The diagnosis is usually carried out in the presence of severe back pain making it possible for vertebral fractures to be identified. It is accepted that there is no recurrence in later pregnancies, although cases followed up over the long term with a number of pregnancies in the same woman are very scarce.

Transitory osteoporosis during pregnancy happens suddenly in the third quarter, and progressively immobilises the mother. Radiological studies show a large loss of bone mass and oedema which may result in fracture. It has been suggested that the origin of this may be found in neurological problems (compression of the obturator nerve), vascular compromise, oedema in the bone medulla, or nutritional deficiencies, but none of these clearly explains the clinical picture.

In addition, no definitive course of treatment has been established, with recommendations varying from bed rest to waiting for the end of gestation to start antiresorptive treatment or to perform orthopaedic surgery.

In summary, it is important to consider generalised or regional osteoporosis during pregnancy, although it is a rare association, by carrying out differential diagnosis of pain which appears suddenly and leads to immobilisation, especially in the third quarter of pregnancy, in order to make an early diagnosis and avoid complications such as fracture.

Bibliography