Magnesium sulphate for preventing preterm birth in threatened preterm labour

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RHL summary

Key Findings

This Cochrane review found that treatment with magnesium sulphate (compared to placebo, no treatment, or alternative tocolytic agents) was not effective for reducing the risk of preterm birth within 48 hours and its use may be associated with a borderline increased risk of fetal, neonatal or infant mortality.

Clinical Implications

- There is no evidence of a meaningful tocolytic effect in the use of magnesium sulphate, and it may be associated with a borderline increase in paediatric deaths
- However, short-term use of magnesium sulphate as an anticonvulsant to prevent eclampsia or for neuroprotection of the very preterm fetus has been shown to be safe and beneficial

Evidence included in this review

Thirty seven randomized trials were included (3,571 women and 3,600 babies) comparing treatment with magnesium sulphate to no treatment, another tocolytic drug or placebo.

Quality assessment

Overall, the included trials were of moderate to high risk of bias; adequate allocation concealment was reported in only six trials and use of a placebo and/or adequate blinding was reported in only 4 trials.

Further Research

Research on the longer-term effects of magnesium sulphate on neurodevelopmental status is needed.

Cochrane review
Abstract

Magnesium sulphate has been used in some settings as a tocolytic agent to inhibit uterine activity in women in preterm labour with the aim of preventing preterm birth.

To assess the effects of magnesium sulphate therapy given to women in threatened preterm labour with the aim of preventing preterm birth and its sequelae.

We searched the Cochrane Pregnancy and Childbirth Group's Trials Register (last searched 31 January 2014).

Randomised controlled trials of magnesium sulphate as the only tocolytic, administered by any route, compared with either placebo, no treatment or alternative tocolytic therapy (not magnesium sulphate) to women considered to be in preterm labour.

At least two review authors assessed trial eligibility and risk of bias and undertook data extraction independently.

The 37 included trials (total of 3571 women and over 3600 babies) were generally of moderate to high risk of bias. Antenatal magnesium sulphate was compared with either placebo, no treatment, or a range of alternative tocolytic agents.

For the primary outcome of giving birth within 48 hours after trial entry, no significant differences were seen between women who received magnesium sulphate and women who did not (whether placebo/no alternative tocolytic drug, betamimetics, calcium channel blockers, cox inhibitors, prostaglandin inhibitors, or human chorionic gonadotropin) (19 trials, 1913 women). Similarly for the primary outcome of serious infant outcome, there were no significant differences between the infants exposed to magnesium sulphate and those not (whether placebo/no alternative tocolytic drug, betamimetics, calcium channel blockers, cox inhibitors, prostaglandin inhibitors, human chorionic gonadotropin or various tocolytic drugs) (18 trials; 2187 babies). No trials reported the outcome of extremely preterm birth. In the seven trials that reported serious maternal outcomes, no events were recorded.

In the group treated with magnesium sulphate compared with women receiving antenatal placebo or no alternative tocolytic drug, a borderline increased risk of total death (fetal, neonatal, infant) was seen (risk ratio (RR) 4.56, 95% confidence interval (CI) 1.00 to 20.86; two trials, 257 babies); none of the comparisons between magnesium sulphate and other classes of tocolytic drugs showed differences for this outcome (10 trials, 991 babies). The outcomes of neonatal and/or infant deaths and of fetal deaths did not show differences between magnesium sulphate and no magnesium sulphate, whether compared with placebo/no alternative tocolytic drug, or any specific class of tocolytic drug. For most of the other secondary outcomes, there were no significant differences between magnesium sulphate and the control groups for risk of preterm birth (except for a significantly lower risk with magnesium sulphate when compared with barbiturates in one trial of 65 women), gestational age at birth, interval between trial entry and birth, other neonatal morbidities, or neurodevelopmental outcomes. Duration of neonatal intensive care unit stay was significantly increased in the magnesium sulphate group compared with the calcium channel blocker group, but not when compared with cox inhibitors or prostaglandin inhibitors. No maternal deaths were reported in the four trials reporting this outcome. Significant differences between magnesium sulphate and controls were not seen for maternal adverse events severe enough to stop treatment, except for a significant benefit of magnesium sulphate compared with betamimetics in a single trial.
Magnesium sulphate is ineffective at delaying birth or preventing preterm birth, has no apparent advantages for a range of neonatal and maternal outcomes as a tocolytic agent and its use for this indication may be associated with an increased risk of total fetal, neonatal or infant mortality (in contrast to its use in appropriate groups of women for maternal, fetal, neonatal and infant neuroprotection where beneficial effects have been demonstrated).

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Home > Magnesium sulphate for preventing preterm birth in threatened preterm labour