

Preliminary report on the use of magnesium sulfate in cases of severe pre-eclampsia and eclampsia.

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:: Introduction



In India maternal mortality in cases of severe pre-eclampsia and eclampsia continues to be high.[2]Management of these cases has always been a controversial subject. It has many aspects like control of convulsions, hypertension and obstetric management. Magnesium sulfate (MgSO₄) is one of the agents used as anticonvulsant all over the world. No maternal mortality has been reported by Pritchard et al[10] with the use of MgSO₄ in eclampsia cases.

The present study was undertaken to judge the efficacy of MgSO₄ therapy in the management of severe pre-eclampsia and eclampsia.

:: Material and methods



During the period from 1st October 1983 to 30th September 1984, at the K.E.M. Hospital,

Bombay, 18 cases of severe pre-eclampsia and eclampsia were managed with IV MgSO₄ therapy (study group) and 42 similar cases were managed with other regimens (control group) like IM pethidine and promethazine with either diazepam or chlorpromazine. Criteria for including the patients of severe pre-eclampsia in the study were presence of 2 or more of the following factors. (1) Blood pressure = 160/110 mm Hg, (2) Edema = 2 +, (3) Urine albumin = 2 +

25% MgSO₄ was administered as prescribed by Zuspan.[14],[15]A loading dose of 4 gm was given intravenously over a period of 10 minutes with a maintenance dose of 1 gm in 5% dextrose every hourly. In cases of severe pre-eclampsia, MgSO₄ was discontinued 8 hours after delivery whereas in eclampsia it was continued for 24 hours after delivery. Women whose diastolic BP exceeded 110 mm Hg were, in addition, given 12.5 mg chlorpromazine intramuscularly. The parameters monitored before administration of MgSO₄ were presence of knee jerk, respiratory rate and urine output.

Serum magnesium levels were estimated[11]in 8 cases of the study group just before and 5 minutes after the loading dose and then every 2 hourly till 24 hours of therapy. Progress of labour and outcome of pregnancy in terms of perinatal loss were monitored in both the groups. The postmortem study was done in cases of deaths.

:: Results

In the study group, 12 were suffering from severe pre-eclampsia and 6 from eclampsia whereas in the control group, 30 cases had severe pre-eclampsia and 12, eclampsia. [Table 1]shows the period when the patients developed eclampsia and mortality. No patient under MgSO₄ therapy developed convulsions after discontinuation of MgSO₄ therapy. In the control group, out of the 12 cases of eclampsia, 10 continued to have convulsions despite the treatment. Out of total 4 deaths, one had convulsions less than 5 in number where as rest 3 had 6 or more convulsions.

Postmortem study revealed that one death in antipartum and one in intrapartum eclampsia were due to cardiorespiratory failure, the other death of intrapartum eclampsia was due to disseminated intravascular coagulation, and the death in postpartum eclampsia was due to hepato-renal failure.

Serum magnesium levels varied from 2.3 to 8.1 mg% during therapy.

In the study group 6 cases required induction or acceleration of labour with slightly higher doses of oxytocin and 4 (22.2%) needed caesarean section. In the control group, 7 (16.61) required caesarean section. One patient in the study group who underwent caesarean section was not given MgSO₄ during operation just like others but she developed some amount of respiratory distress after the operation was over and Hence was administered calcium gluconate to counteract residual effect of MgSO₄ if any. It was then that the patient had a convulsion which was controlled by parenteral administration of diphenylhydantoin.

In the study group perinatal loss was 8 (44.4%) out of which 3 were premature still births and 4 were premature neonatal losses and 1 was full term baby dying of congenital

malformation. In the control group, perinatal loss was 13 (30.9%) out of which 5 were premature still births, 3 were premature neonatal losses and 5 were full term neonatal deaths.

:: Discussion



Lazard^[6] promoted use of magnesium in toxemia 60 years ago. Magnesium acts like curare at the motor end plates.^[5] It may cause peripheral vasodilation and decrease the response of vascular smooth muscles to sympathomimetic amines.^[7] But still its hypotensive effect is minimal.^{[4],[8]} Magnesium has little effect on the central nervous system.

An unrecognized side effect of magnesium is potentiation of both the depolarising (succinylcholine) and non-depolarising (curare) relaxants. So the patient receiving magnesium is given standard initial doses of curare and succinylcholine for rapid sequence intravenous induction of anaesthesia but the subsequent doses of relaxant if required are given in smaller amounts.^[3]

As magnesium can induce relaxation of the uterus resulting in slowing of contractions, slightly higher doses of oxytocin may be needed as happened in the study group.

According to Zuspan,^[15] the delivery should be expected within 12 hours using this regimen of MgSO₄. If the progress is not satisfactory then the patient may be delivered by caesarean section. The caesarean section rate in this group of patients would be 50% higher than in the general population.

No patient on magnesium therapy showed any signs of toxicity necessitating stoppage of therapy. Maximum level of 8.1 mg% obtained after 20 hours of therapy was well within the therapeutic range of 4.8-8.4 mg%.^[9] Sibai et al^[12] found that the rise of serum magnesium level was gradual until a maximum was reached after which the level remained constant. So adjusting the maintenance dose according to the clinical response of the patient remains the best approach.^[12] No case in the present study group required more than 1 gm/hour maintenance dose.

No untoward effect of MgSO₄ was noticed on the babies after birth. It has been stated that magnesium is rarely deleterious to the neonate.^[13] Cruikshank et al^[1] found levels of ionised calcium in foetal blood of the mothers under MgSO₄ treatment in the normal range but near the lower limits. Their results indicated that the principal maternal response to magnesium induced hypocalcemia involved increased parathyroid hormone secretion which tends to preserve maternal calcium homeostasis while the foetus is partially protected from hypermagnesemia and hypocalcemia by the placenta.

It is the stress of labour, premature delivery, anoxia due to convulsions and heavy sedation of eclamptic women in labour which increase the danger to foetus. It can be minimised by regular antenatal check-up, proper management during labour and prompt resuscitation of the newborn after birth.

Because of a small number of patients in the study group, no firm conclusion can be











drawn. But from this data showing no convulsion in the patients under MgSO₄ therapy compared to continuing seizures in 10 out of 12 patients of eclampsia in the control group and also no death in study group compared to 4 deaths in the control group, it appears that MgSO₄ is an effective drug in controlling convulsions and preventing material deaths in eclampsia and sever pre-eclampsia.





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