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# Comparison between dexmedetomidine and midazolam for sedation of eclampsia patients in the intensive care unit

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**Abstract**

**Purpose:** This study compares the effectiveness of midazolam and dexmedetomidine for the sedation of eclampsia patients admitted to our intensive care unit (ICU).

**Patients and Methods:** Forty women with eclampsia requiring termination of pregnancy by caesarean delivery were randomized in to 2 groups of 20 to receive either midazolam or dexmedetomidine. The midazolam group received a loading dose of 0.05 mg/kg followed by an infusion of 0.1 mg $kg^{-1}$ $h^{-1}$. The dexmedetomidine group loading dose was 1 $\mu$g/kg per 20 minutes, followed by continuous infusion at 0.7 $\mu$g $kg^{-1}$ $h^{-1}$. Heart rate, blood pressure, Ramsey sedation score, antihypertensive need, convulsion fits, and duration in ICU were monitored and recorded all through the ICU stay.

**Results:** Dexmedetomidine markedly reduced heart rates for the first 24 hours ($P < .05$) compared with midazolam, but there were no differences at 48 and 72 hours. Mean arterial blood pressures were similar in the 2 groups ($P > .05$), although in the dexmedetomidine group, it was lower at 5, 6, 12, and 24 hours compared with the first 4 hours ($P < .05$). Moreover, fewer patients given dexmedetomidine required nitroglycerine and nitroprusside ($P < .05$). The duration of ICU stay was less in the dexmedetomidine group, 45.5 hours (range, 15-118 hours), than in the midazolam group, 83 hours (minimum-maximum, 15-312 hours).

**Conclusion:** Dexmedetomidine sedation in eclampsia patients is effective in reducing the demand for antihypertensive medicine and duration of ICU stay.

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## 1. Introduction

Preeclampsia is characterized by hypertension diagnosed after 20 weeks gestation plus proteinuria. In severe cases, the diastolic blood pressure is 110 mm Hg or more, there is a persistent proteinuria of 2 or more, and any of the following can be present: headaches, visual disturbances, upper abdominal pain, oliguria, increased serum creatinine, thrombocytopenia, increased liver enzymes, fetal growth retardation, and pulmonary edema. Eclampsia is the new onset of seizures before, during, or after labor, which is not attributable to other causes, in a woman with preeclampsia. Eclampsia, a complication of pregnancy characterized by seizures and accompanied by severe hypertension, brain and lung edema, aspiration pneumonia, and acute renal failure, remains a major cause of maternal morbidity and mortality in both developed and developing countries. Proteinuria and acute respiratory distress syndrome can also follow [1].

The main goals of treatment are to stabilize the patient; control derangements of the cardiovascular, hematological, renal, pulmonary, and central nervous systems; and prevent

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potential future problems [2]. It is critical for both mother and baby that new seizures be effectively controlled; they are usually treated with magnesium sulfate, intramuscularly or intravenously, but may still occur, exacerbating maternal morbidity and mortality [1].

Several anticonvulsant drugs have been tried. Midazolam is a fast-acting benzodiazepine with a short elimination half-life; has powerful anxiolytic, amnesic, hypnotic, anticonvulsant, skeletal muscle relaxant, and sedative properties; and has been used for sedation in the intensive care unit (ICU) for many years. Its use has been proposed for the treatment of eclampsia. Midazolam undergoes extensive oxidation in the liver via the cytochrome P450 enzyme system to form water-soluble hydroxylated metabolites, which are excreted in urine. However, the primary metabolite, namely, 1-hydroxymethylmidazolam, has mild central nervous system depressant activity and may accumulate in the critically ill patient, especially in the case of kidney failure [3-5].

Dexmedetomidine is a centrally acting $\alpha_2$-agonist with sedative and analgesic properties; it is similar to clonidine but has much greater $\alpha_2$ to $\alpha_1$ binding affinity. The sedative properties are facilitated through the locus coeruleus site in the central nervous system, and the analgesic effects may occur via activation of the $\alpha_2$-receptors by accentuating the action of opioids. After extensive metabolism in the liver, dexmedetomidine is eliminated as methyl and glucuronide conjugates, mainly (95%) via renal excretion [6].

Dexmedetomidine used in intensive care as a sedative without respiratory depressive effects has analgesic properties and controls stress, anxiety, and pain [7].

In this clinical study, we compare dexmedetomidine and midazolam for sedation in eclampsia patients with regard to their effectiveness, hemodynamic characteristics, and ICU discharge time.

## 2. Patients and methods

This is a prospective, randomized, and controlled study. After obtaining ethic committee approval from Erciyes University Hospital, patients' first-degree relatives were informed about the study, and their written consent was taken. Forty patients whose pregnancies were terminated via caesarean delivery because of eclampsia and who needed ventilatory support were included in the present study. All patients who had chronic hypertension; cardiac, neurological, hepatic, renal, or endocrinal disease; or allergic reactions to the medicine used during the treatment or developed the Hemolysis, Elevated Liver Enzymes and Platelets (HELLP) syndrome were excluded from the study.

All patients received $MgSO_4$ 2 g/h for 24 hours. Invasive blood pressure, heart rate, oxygen saturation, central venous pressure (Hewlett Packard system no. M1205A), and sedation score were recorded hourly. The patients were randomly divided into 2 groups using coin toss. The group GrM (n = 20) received midazolam immediately after admission. After delivering a loading dose of 100 mg in 100 mL 0.9% NaCl at 0.05 mg/kg, it was continued at 0.1 mg $kg^{-1}$ $h^{-1}$. The other group, GrD (n = 20), received dexmedetomidine (Precedex Abott Labs, North Chicago, Ill) immediately after admission. A dexmedetomidine loading dose was administered at 1 $\mu$g/kg per 20 minutes, followed by a continuous infusion at 0.7 $\mu$g $kg^{-1}$ $h^{-1}$ (400 $\mu$g dexmedetomidine is put in 100 mL physiological saline). The sedation and analgesic scores were assessed at 1-hour intervals. Sedation was maintained to meet the Ramsey Sedation Scale 2-3 criteria (Table 1). When sedation became inadequate (Ramsey sedation score <2), propofol was given as a bolus (0.5 mg $kg^{-1}$) in both groups. Pain of the patients was assessed by Visual Analog Scale, when Visual Analog Scale greater than 4 fentanyl was administered in the dose of 1 $\mu$g $kg^{-1}$.

**Table 1** Ramsey sedation scale

| Score | |
|---|---|
| 1 | Nervous, agitated, and/or restless |
| 2 | Cooperative, orientated, quite patient |
| 3 | Only obeying the orders |
| 4 | Sleeping, suddenly responding to hitting the glabella, and high voice |
| 5 | Sleeping, slowly responding to hitting the glabella, and high voice |
| 6 | No response to any of these stimulations |

After admission to the ICU, mean arterial pressure (MAP) was maintained between 100 and 126 mm Hg. If it exceeded this, nitroglycerin (mean dose, 0.5-5 $\mu$g $kg^{-1}$ $min^{-1}$) was infused. If this was insufficient, it was replaced with sodium nitroprusside. Patients were continuously monitored for convulsions. Thiopental was used as an anticonvulsant in each group.

The weaning process was started if there were no signs of respiratory ($Pa_{O_2}$ >60 mm Hg, fraction of inspired oxygen <0.4, and positive end expiratory pressure (PEEP) ≤5 mm Hg) or hemodynamic impairment and if the patient was able to cough. Extubation was performed if there were no signs of respiratory (tidal volume >4 mL/kg, respiratory rate 10 to 25/min, $Pa_{O_2}$ >69 mm Hg, $Pa_{CO_2}$ <50 mm Hg, and fraction of inspired oxygen ≤0.4) or hemodynamic (MAP at 100-126 mm Hg without vasodilators) impairment and if the patient was able to follow commands.

Discharge from the ICU was performed if there were no signs of neurological (cooperative, oriented, and tranquil), respiratory ($Pa_{O_2}$ >69 mm Hg, $Pa_{CO_2}$ 35-45 mm Hg, and inspired $O_2$ <3 L/min), hemodynamic (MAP at 100-126 mm Hg without vasodilators), or surgical impairment.

### 2.1. Statistical analysis

The power analysis was calculated by the demand of antihypertensive ($df$ = 1, $\alpha$ = .05, power = 0.88). Statistical analysis was performed using SPSS (version 10.0, SPSS, Inc).

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