A 70 kg, previously healthy, 28-year-old primigravida was admitted to our delivery ward at 36 weeks of gestation. The patient was suffering from severe preeclampsia with H.E.L.L.P (hemolysis, elevated liver enzymes and low platelets count) syndrome. She was treated with magnesium sulfate 4 g bolus followed by 2 g/h continuous infusion (her magnesium plasma levels were 5-6 mg%) and hydralazine but, since her clinical status was worsening (severe hypertension and decreasing platelets count from 80,000 to 40,000/µL), an urgent Cesarean delivery was planned. In view of the low platelet count it was decided to perform general anesthesia using a rapid-sequence induction with propofol 3 mg/kg and succinylcholine 1.5 mg/kg. Standard anesthetic monitoring included a nerve stimulator (TOF-Watch SX, Organon-Technika Boxtel, the Netherlands) with the active electrode attached over the ulnar nerve. Anesthesia was maintained with 1-2% isoflurane in a mixture of 50% oxygen/nitrous oxide. Fentanyl 100 mcg was administered after the baby was delivered and the umbilical cord was clamped. A healthy baby was delivered. To this stage, no further muscle relaxant was administered. Following closure of the uterus (10 minutes after skin incision), patient movement was noticed despite an apparently adequate depth of anesthesia. Rocuronium 15 mg was administered with disappearance of the TOF response. At the time of skin closure (45 minutes from the skin incision and 35 min after rocuronium administration) the end tidal isoflurane was zero, end tidal CO₂ was 37 mmHg, core temperature was 35.5°C and the pupils were not pin-point. The train-of-four (TOF) and post-tetanic count (PTC) responses were zero. Therefore, reversal with sugammadex rather than with neostigmine/atropine was chosen. Sugammadex 400 mg (5.7 mg/kg) was administered and within 30 seconds, the TOF ratio was 80% and 95% at 60 seconds. The patient was awake and able to lift her arms and head and hold them up for more than 10 seconds. She was transferred to the PACU after an uneventful extubation. No adverse effects related to sugammadex and no evidence of recurarization was observed.

Sugammadex is a novel aminosteroid muscle relaxant-binding agent. Three previous reports of obstetrics anesthesia patients [1–3] have shown its efficacy to reverse a profound neuromuscular blockade caused by rocuronium administered at the induction of general anesthesia for cesarean delivery. In this report, we present a case where the muscle relaxant effect of a low dose rocuronium was potentiated by concomitant magnesium administration. Magnesium antagonizes calcium, thus decreasing the release of acetylcholine to the synapse of the motor-neuron end plate. This will potentiate the competitive block caused by a non-depolarizing muscle relaxant. Sugammadex binds to free aminosteroid relaxants (in our case rocuronium) in plasma, thus reducing the amount of muscle relaxant at the receptor site and causing reversal of the muscle relaxants’ activity [4]. To our knowledge this is the first reported administration of sugammadex in the presence of a deep neuromuscular block caused by a potentiated effect of rocuronium by magnesium. In our case, sugammadex reversed the deep block produced by a small dose of rocuronium, in the presence of magnesium treatment for severe preeclampsia.