Adverse Effects of Diclofenac after Cesarean Section: A Case Report

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Abstract: A 27-year-old nullipara, 38 weeks pregnant, was admitted for elective cesarean section under spinal anesthesia. Past medical history was unremarkable. Spinal anesthesia was performed with 10 mg of 0.5% heavy bupivacaine. Ephedrine 10 mg was added to Ringer solution for prevention of hypotension. Hemodynamic parameters were normal and stable during surgery. Several hours later, the patient asked for analgesic drug and two 100mg diclofenac suppositories were administered. Three hours later, the patient suffered from an extensive and cramping abdominal pain. The pain was located in the right upper quadrant and radiated to the right infrascapular region. Electrocardiogram obtained was normal. Intravenous pantoprazole and oral anti-acid were administered, but no decrease in pain was seen after fifteen minutes. Subsequently, a sublingual nitroglycerine perle was administered and the intensity of pain was decreased significantly. The diagnosis of Oddi spasm was assumed.

Keywords: Diclofenac, Cesarean section, Oddi spasm, Coronary spasm.

INTRODUCTION

Pain control is the goal of obstetric analgesia in patients undergoing elective cesarean section. Surgical incision is associated with a severe pain especially in the first 24 h of the postoperative period. Opioid administration is the first step for the management of pain in these patients, but respiratory depression restricts the use of this medication [1]. Epidural analgesia, infiltration analgesia or intra-venous patient control analgesia with low dose opioid regimens are some good alternative techniques. Administration of nonsteroidal anti-inflammatory drugs suppositories is a very simple and non-expensive method, but it can also have some adverse effects.

CASE REPORT

A 27-year-old nullipara, thirty eight-weeks pregnant, was admitted for elective cesarean section under spinal anesthesia at nine o'clock in the morning. No smoking or alcohol or drug consumption was reported in past medical history. Routine tests, including coagulation tests, were normal. After arrival in the operating room and before the spinal block, 15 ml/Kg of Ringer solution was infused within 10 minutes. Maternal heart rate and arterial blood pressure were measured by an automatic non invasive monitor during surgery. Spinal anesthesia was performed in the sitting position with a 25 gauge Whitacare needle, using a midline approach at L4-5 interspace. Once free flow of CSF had been recognized the intrathecal anaesthetic solution (10 mg of 0.5% heavy bupivacaine) was injected. Following this, the patient was turned in supine position with left uterine displacement. Ephedrine 10 mg was added to ringer solution for prevention of hypotension. Surgery commenced after a sensory block up to T5 dermatome was achieved. Hemodynamic parameters were normal and stable during surgery. After clamping of the umbilical cord, 30 IU of oxytocin was added to 1000 ml of lactated Ringer's solution and allowed to infuse over a 45 min period. The newborn was delivered with normal Apgar score.

At 11PM, the patient asked for analgesic drug and two 100mg diclofenac suppositories were administered by anal route. At 2 AM, the patient suffered from an extensive and cramping abdominal pain. The pain was located in the right upper quadrant and radiated to the right infrascapular region. Heart rate was 80/min and BP was 120/80. Electrocardiogram obtained was normal. Intravenous pantoprazole and oral anti-acid were administered, but no decease in pain was seen in 15 minutes. Subsequently, a sublingual nitroglycerine perle was administered and the intensity of pain was decreased significantly. The diagnosis of Oddi spasm was assumed. The patient refused more diagnosis evaluations and was discharged the day after surgery.

DISCUSSION

Sphincter of Oddi dysfunction normally presents with pain in the right upper quadrant or epigastrium.
The symptoms of this mal function can be similar to some other disorders including: cholelithiasis, cholescystitis, gastroesophageal reflux disease, functional dyspepsia, irritable bowel syndrome, and pancreatitis [2].

The evaluation for differential diagnosis includes transabdominal ultrasound, abdominal computed tomography, magnetic resonance cholangiopancreatography, upper endoscopy, endoscopic ultrasound, and endoscopic retrograde cholangiopancreatography (ERCP). In addition, therapeutic trials of medications such as antispasmodics or proton pump inhibitors should be considered in patients with symptoms suggestive of possible irritable bowel syndrome or gastroesophageal reflux/functional dyspepsia [3].

Medical treatment of Oddi dysfunction includes calcium channel blockers, trimebutine and nitrates [4].

Nitroester drugs can relax vascular smooth muscles, including that of gastrointestinal tract. They also can effectively relax the muscle of Oddi’s sphincter. Among these drugs the effect of glyceryl trinitrate on the sphincter of Oddi has been well researched [4].

Staritz reported that sublingual administration of 1.2 mg of glyceryl trinitrate markedly lowered the sphincter of Oddi basal tone and phasic contraction amplitude [5]. Brandstatter found a remarkable decrease of Oddi contraction, which lasted for many minutes after intravenous administration of nitroglycerine [6].

Uchida declared that sublingual glyceryl trinitrate enables the endoscopic extraction of small (6-12 mm) common bile duct (CBD) stones [7]. Luman et al found that topical infusion 5 or 10 mg of glyceryl trinitrate significantly decreased the Oddi’s sphincter tone. They stated that local administration of glyceryl trinitrate was not accompanied by adverse effects [8].

We think that the pain of our patient was due to Oddi spasm, but non-steroidal anti-inflammatory drugs (NSAID) use can be complicated but some other adverse effects.

The most common adverse effects of nonsteroidal anti-inflammatory drugs are gastritis, peptic ulceration, and depression of renal function, all of which result primarily from prostaglandin inhibition. The types of side effects observed with diclofenac are similar to those of other nonsteroidal anti-inflammatory drugs and are unavoidable given that the drugs are prostaglandin inhibitors.

Use of NSAIDs is associated with cardiovascular and coronary spasm risk [9]. Rofecoxib and diclofenac were associated with increased cardiovascular morbidity and mortality [10-11].

Hayashi et al reported that symptomatic NSAID-induced small-bowel injuries exhibit a variety of patterns of ulcerative lesions as observed in the ileum in many cases [12]. The ileocecal region is a potential site for a variety of NSAID-induced injuries including erosions, ulcers, strictures, perforation, and the formation of diaphragms, which can lead to bowel obstruction [12]. NSAIDs can also lead to colitis resembling inflammatory bowel disease (IBD), exacerbate pre-existing IBD, or complicate diverticular disease (perforation or bleeding) [13].

CONCLUSION

The same dose of diclofenac were administrated to many of patients for post operative analgesia, but the obstetric patients may be at a greater risk of adverse effects due to their upper cardiac output, and so upper mucosal uptake [14]. The pain in this case is not relieved by antacids and proton pump inhibitors, so we suggested that the source of this pain can be Oddi’s sphincter spasm.

REFERENCES

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