

Optimal Pain Management After Cesarean Delivery

Mohamed SH. Ramadan^{1*}, Khaled Mohamed², Omnya Khalifa Aldawy¹, & Tarek Mohamed Elbeheidy¹

¹Department of Obstetrics and Gynecology Faculty of Medicine, Zagazig University, Alsharqua, Egypt.

² Department of Anesthesiology and surgical intensive care Faculty of Medicine, Zagazig University, Alsharqua, Egypt.

Correspondence: Drmramadan83@gmail.com

ABSTRACT

Effective pain management is critical for women after caesarean delivery, and significant postoperative pain is related with persistent pain, higher opioid use, delayed functional recovery, and postpartum depression. Intrathecal morphine is the standard method for post-c-section pain, offering superior and extended analgesia. Scheduled non-steroidal anti-inflammatory medications and acetaminophen should be included in multimodal analgesia, with opioids reserved for severe breakthrough pain. Wound infiltration and transversus abdominis plane blocks are critical components of multimodal analgesia for patients who cannot receive neuraxial opioids or who do not have appropriate pain management. While analgesics may transfer to breastfeeding infants, transfer could be reduced by careful drug selection and administration timing.

1. INTRODUCTION

The rate of cesarean delivery in the United States has been increasing over the past decades and now exceeds 32% of births.¹ Effective postoperative analgesia is critical, because women who undergo cesarean delivery rank avoidance of pain during and after surgery as their highest priority.² Management of postcesarean pain may have lasting effects, and severe acute postoperative pain is associated with persistent pain, greater opioid use, delayed functional recovery, and increased postpartum depression.³ Effective pain relief after cesarean delivery improves a woman's ability to function and interact with her newborn infant.⁴ An individual patient's specific plan should be determined in the context of any medical and psychiatric comorbidities, chronic pain, and prior postoperative or postpartum experiences.⁵ The American Pain Society recommends that planning for postoperative pain management should begin in the preoperative period. Physicians should focus on individualizing perioperative pain management, often through a multimodal approach.⁵ Compared with other surgeries, formulating a plan for optimal anesthesia and analgesia for cesarean delivery involves several distinct considerations: Surgical anesthesia is almost exclusively neuraxial and is performed in awake, unsedated patients

Preemptive analgesic use is limited because of concerns for in-utero fetal drug transfer. The potential transfer of analgesic drugs to breastfeeding neonates should be considered. Maximal postoperative mobility of mothers in order to facilitate optimal neonatal care is extremely important. Multimodal analgesia options for providing optimal postoperative pain relief for women undergoing uncomplicated cesarean delivery with neuraxial anesthesia are summarized in this article. Analgesic options are appropriate for most parturients, but there are many women whose medical comorbidities require special consideration. Conditions that require alterations to pain management include chronic pain, obstructive sleep apnea, and a contraindication to neuraxial anesthesia. Although several key points are highlighted, detailed management of these conditions is beyond the scope of this article. NEURAXIAL

2. MEDICATIONS

Intrathecal morphine Epidural morphine Intrathecal hydromorphone Continuous and patient-controlled epidural infusions Nonopioid neuraxial adjuncts The American Society of Anesthesiology's Obstetric Anesthesia Practice Guidelines and the American Pain Society's Clinical Practice Guidelines both recommend the routine use of neuraxial anesthesia for cesarean delivery.^{5,6} The use of neuraxial anesthesia for cesarean delivery is promoted because of decreased maternal risk and improved fetal outcomes, but the additional benefit of superior postoperative analgesia with the use of neuraxial opioids deserves emphasis.⁷ Standard regimens for intraoperative cesarean anesthesia consist of a combination of local anesthetic and a lipophilic opioid (eg, fentanyl). Although neither drug provides prolonged postoperative analgesia,⁸ they provide analgesia in the early postoperative recovery period until the onset of longer acting neuraxial opioids; neuraxial morphine has an analgesic onset of approximately 60 to 90 minutes.

3. CESAREAN SECTION

Cesarean delivery is the birth of a fetus through incision in abdominal wall (laparotomy) and the uterine wall (hysterotomy) [1].

3.1. Historical background

The origin of the term "cesarean section", several explanations have been suggested. In the first, according to legend, Julius Caesar was born in this manner, with the result that the procedure became known as the Cesarean operation. Several circumstances weaken this explanation. First the mother of Julius Caesar lived for many years after his birth in 100 BC, and as late as the 17th century, the operation was almost invariably fatal. Second, the operation whether performed on the living or dead, is not mentioned by any medical writer before middle ages [2].

3.2. Incidence:

Cesarean section (CS) is one of the most common major surgery performed, million women who undergo this operation per year [3].

The Egypt Demographic and Health Survey obtained information about the frequency of cesarean deliveries (CD). More than one-half of deliveries in the five-

year period before the review were by CD. The likelihood of CD increased with the age of the mother and decreased with the child's birth order. CD was more common in urban areas than in rural areas (60 percent and 48 percent, respectively). CD was less common in Upper Egypt, especially in rural areas, and in the Frontier Governorates than in the Lower Egypt and the Urban Governorates [4].

3.3. *Technique of cesarean section:*

A caesarean section be made of several individual surgical steps based on the anatomical layers to be incised before and closed after the baby is extracted. The surgical techniques used in each step of the caesarean section may affect both short- and long-term maternal morbidity [5].

3.4. *Preoperative preparation:*

In the case of a planned process, the preoperative assessment should include a full history and physical examination, past medical and surgical history, current medications, drug allergies, consent, and indication for cesarean section. In the uncomplicated patient checking a full blood count. In more complex cases preoperative consultation with an anesthetist, or other relevant specialist should be considered on an individual basis. The obstetrician should usually highlight women who are at high risk of anesthetic complications during the antenatal period. The risks should be documented in the medical notes and communicated with the anesthetist nearer the time [6].

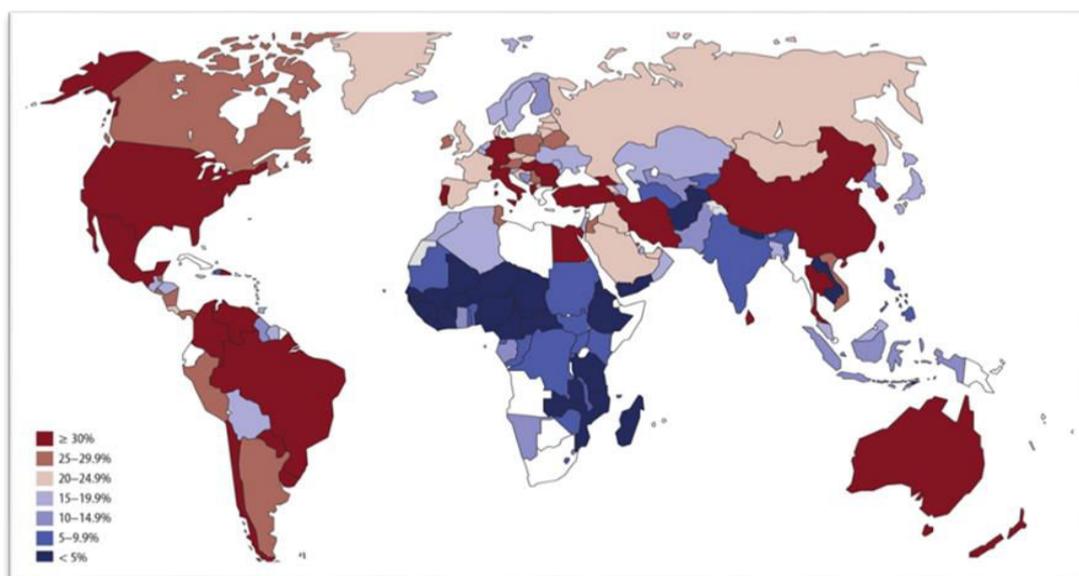


Figure 1: Country variation of CS rates according to the latest nationally representative reported data [7].

3.5. *Postoperative complications:*

Hemorrhage is more likely to be atonic in the early postoperative period. Atony usually results in revealed bleeding and necessitates blood replacement and evacuation of clot from the uterus and cervix, and uterotonic agents must be used. Unidentified trauma often results in abdominal signs with circulatory decompensation and requires surgical exploration together with circulatory resuscitation [8].

Later bleeding is usually associated with endometritis and requires broad-spectrum antibiotics. The possibility of retained placental tissue should be extremely low, and uterine exploration in the presence of a scar should be avoided. Ultrasound assessment of the uterus is rarely helpful in the early puerperium and may be misleading in mistaking blood clot for retained tissue [8].

Bowel dysfunction: Postoperatively, some patients may experience a slow return of bowel function. Postoperative narcotics may delay return of normal bowel function in a few patients. Most respond to conservative therapy, but a small portion may require decompression. In those with a slow return of bowel function, assessment of fluid and electrolyte status needs to be a priority [9].

Postpartum endomyometritis: This is increased significantly in patients who have had a cesarean section. The rate of endomyometritis is up to 20-fold higher than with a vaginal delivery, with a reported mean of 35-40% occurrence after a cesarean section. Major risk factors include whether the cesarean section was the intended (primary) procedure and the socioeconomic status of the patient. Other major risk factors include duration of membrane rupture, duration of labor, number of pelvic examinations, and the presence of chorioamnionitis prior to initiating cesarean section. Blood cultures are positive in approximately 10% of patients with postoperative febrile morbidity, and broad-spectrum antibiotics should be used. Postcesarean rate of endomyometritis is decreased to 5% with the used prophylactic antibiotics [10].

Wound infection: Following a cesarean section, the risk of a wound infection ranges from 2.5% to higher than 15%. According to the study of (Tran et al., [11]).

Placenta previa: Theoretically, scarring of the endomyometrium secondary to hysterotomy may lead to later low implantation and placenta previa in the following pregnancy. Numerous studies have confirmed the increased risk of placenta previa following cesarean section [12].

4. MANAGING POSTOPERATIVE PAIN IN C-SECTION

Postoperative pain is not simple due to tissue injury alone but is the final result of various neurophysiological interactions. This makes efficient postoperative pain management much more difficult and an ideal pain management programmed is still abstract. A step-up method to post-operative pain management will provide adequate analgesia while minimizing exposure to adverse events. Thus, post-operative pain relief is important for decreased morbidity and mortality [13].

For effective post-surgical analgesia, interventions at the levels of peripheral sensitization, mediators and central sensitization requires a multimodal approach to pain relief. In addition to local anesthetics, other interventions that may be used as part of a multimodal approach include the following [14].

a) **Opioids:** Post-operative pain management is centered on opioids, although side-effects are common. Administration is best via infusion pump as patient-controlled analgesia.

b) Non-steroidal anti-inflammatory drugs: As diclofenac, NSAIDs inhibit prostaglandin synthesis. The platelets and gastrointestinal side effects are affected by cyclooxygenase isoenzymes (COX-1).[14].

c) TAP block as multimodal analgesia lowered postoperative severity of pain scores, reduced opioids consumption and complications as well as prolonged time for the first analgesic requests[15].

4.1. Paracetamol

Paracetamol work through central cyclooxenase (COX 2) inhibition, with a reduction in central nervous system prostaglandin E2 production and activation of descending serotonergic pathways [16].

Munishankar et al.,[17] revealed that the combination of paracetamol and diclofenac resulted in significantly less morphine consumption.

4.2. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Pain after cesarean delivery may have at least two components: postoperative (somatic) pain from the wound itself and visceral pain arising from the uterus. Although somatic pain may be relieved by opioids, visceral pain may be more difficult to treat. NSAIDs are effective for relieving pain related to menstrual cramping and, as a result, there has been interest in the use of NSAIDs to treat a component of pain after cesarean delivery. Unfortunately, NSAIDs alone are inadequate to effectively treat post-cesarean delivery pain.

However, inclusion of NSAIDs in a multimodal approach to pain relief after cesarean delivery has been very successful both in improving the quality of analgesia resulting from systemic or neuraxially administered opioids and reducing side effects [18].

For instance, use of IM diclofenac 75 mg results in a morphine-sparing effect and a decrease in side effects related to morphine. These benefits also apply to women having regional or general anesthesia and to women having intraspinal opioids for pain relief [19].

The disadvantages to using NSAIDs relate to the probable for gastrointestinal side effects and platelet dysfunction. In this regard, use of cyclooxygenase (COX-2) inhibitors may be better because they do not inhibit platelet function. However, COX-2 inhibitors are secreted in the breast milk and there is little experience using these drugs in breast-feeding women[17].

4.3. Diclofenac

They appear to have anti-inflammatory, antipyretic, and analgesic properties. These are thought to be mediated via inhibition of prostaglandins. This inhibition of prostaglandins is itself mediated via the inhibition of the cyclooxygenase (COX) enzyme. There are two forms of the COX enzyme. COX-1 is involved in 'housekeeping' activities, such as mediating normal platelet function, regulating renal blood flow and providing cytoprotecting of the gastric mucosa. COX-2 is involved in

the response to tissue damage and mediates inflammation and pain. The COX-2 inhibitors are more selective in their inhibition of COX-2 relative to COX-1. The COX-2 inhibitors have been associated with higher rates of cardiovascular adverse events and it is hypothesized that this effect is a result of relative COX-2/COX-1 inhibition. While diclofenac is a traditional NSAID, it does display a preferential inhibition of COX-2 compared to COX-1 [20]. While the in vivo effects of paracetamol are similar to those of the selective COX-2 inhibitors [21].

Diclofenac sodium as a NSAID mediator has anti-rheumatic, anti-inflammatory, pain control and some other properties. Regardless of the fact, it causes platelet, renal and gastrointestinal dysfunction [22].

4.4. Contraindications

a) Absolute contraindications to PCA include

The patient is unable to understand the concept behind PCA, Systemic infection, or infections at the preferred site of PCA placement, Allergic reactions to the designated medication, Burns or trauma on the area of PCA placement, Previous neural deficits in the area of a planned indwelling nerve catheter, and Increased ICP for epidural catheter placement [23].

b) Relative contraindications to PCA include

Chronic renal failure, the patient is on antithrombotic therapy, the patient has a documented bleeding disorder, and Sleep apnea [23].

4.5. Pharmacology of Opioids Used in PCA

a) Nalbuphine

Nalbuphine is an opioid, that blocks the μ receptor, activates the κ receptor, causing analgesia and sedation [24], Use of nalbuphine carries a lower risk of the respiratory depression, nausea, vomiting, pruritus, constipation, PONV (postoperative nausea and vomiting) and urinary retention, when compared to morphine [25]. Nalbuphine is an opioid has a ceiling effect in respiratory depression hence, it is considered to be safer than morphine, being mu antagonist and kappa agonist, it has lower incidence of adverse effects in comparison with morphine [26].

b) Morphine

Morphine and its metabolites act as agonists of the mu and kappa opioid receptors [27].

The mu-opioid receptor is integral to morphine's effects on the ventral tegmental area of the brain. Morphine's activation of the reward pathway is mediated by agonism of the delta-opioid receptor in the nucleus accumbens (Kim J et al. [28]) while modification of the respiratory system and addiction disorder are mediated by agonism of the mu-opioid receptor [29].

4.6. Transversus Abdominis Plane Block

Introduction:

TAP block was first clear by Rafi in 2001 [30]. In this technique, two facial nerve clicks are felt while passing through the external and internal oblique muscles benefiting from the 'triangle of Petit,' and local anesthesia is set at this area. In 2007, this technique was defined again with ultrasound (USG) guidance.

Ultrasound-guided TAP block is performed by observing the region between the internal oblique muscle and transversus abdominis muscle, called 'TAP', for blocking the frontal branches of T6-L1 nerves and administering local anaesthetic agents [11].

Applied anatomy:

abdominal wall supplied by various spinal nerves and identifying fascial planes of abdominal muscles within which these nerves lie after originating from transverse foramina of the vertebral column [31].

The skin and muscles of the abdominal wall are supplied by spinal nerves originating from T6 to L1 level. A typical spinal nerve originates and divides into anterior and posterior divisions, called anterior and primary rami. The anterior rami supply the muscles and skin of the anterolateral abdominal wall. The spinal nerves can be clubbed into:

A. Thoracoabdominal nerves: These are anterior rami of the spinal nerves of T6–T11 [31]. They divide into lateral and anterior cutaneous branches. Lateral cutaneous branches arise in the neurovascular plane between the internal oblique and transversus abdominis muscle, near the angle of the rib, and supply the skin after piercing the external oblique and internal oblique muscle. The anterior cutaneous branch arises also at the lateral border of the rectus sheath and it pierces the rectus abdominis muscle before supplying the skin. They supply the muscles and skin of the upper anterolateral abdominal wall, between the umbilicus and coastal margin.

B. Subcostal nerve: This is the anterior rami of the T12 spinal nerve, which follows the course similar to thoracoabdominal nerves and ends in similar lateral and cutaneous branches. It innervates muscles and skin of the lower anterolateral abdominal wall, between the umbilicus and inguinal ligament.

C. Ilio-hypogastric and ilioinguinal nerves: Terminal branches of the anterior rami of the L1 spinal nerve.

The dermatomal distribution of the abdominal wall closely correlates with the pathway of spinal nerves and their branches because there is no plexus formation at paravertebral level [31].

all branches further connect at multiple locations, including large branch communications on the anterolateral abdominal wall (intercostal/upper TAP plexus) and plexuses that run with the deep circumflex iliac artery (DCIA) (lower TAP plexus) and the deep inferior epigastric artery (DIEA) (rectus sheath plexus). Since

these segmental nerves communicate just above the transversus abdominis muscle, the subfascial extent of local anesthetic can provide anterolateral abdominal wall analgesia [32].

Muscles:

The anterolateral abdominal wall is formed by bilaterally paired three flat muscles and two vertical muscles):

Flat muscles (from outside to inside): External oblique muscle, Internal oblique muscle, and Transversus abdominis muscle.

Vertical muscles: Rectus abdominis and Pyramidalis muscle.

Nomenclature of TAP block:

A TAP block basically contains deposition of local anesthetic in the plane between the internal oblique and transversus abdominis muscles to object the nerves passing through them. Since the transversus abdominis plane is spread over a large area crossing dermatomes, it forms a huge bull's eye for anesthesiologists to target. Hence, the TAP block has evolved considerably from the "Classical" TAP block introduced by Rafi, to other variants which try to approach the "Transabdominis Plane" [30]. There is no standardized classification and nomenclature of TAP block [33].

Classical TAP block:

The classical TAP block is the landmark based TAP style which was first introduced by (Rafi et al. [30]) The "triangle of petit" is the landmark from where the TAP is move toward. The triangle of petit is situated between the iliac crest and subcostal margin. The base of the triangle is formed by the iliac crest, and it is bounded anteriorly by the external oblique muscle and posteriorly by the lattissimus dorsi muscle. According to the technique described by (McDonnell et al., [34]) the iliac crest is palpated in anterior to posterior direction, and insertion of the lattissimus dorsi is identified.

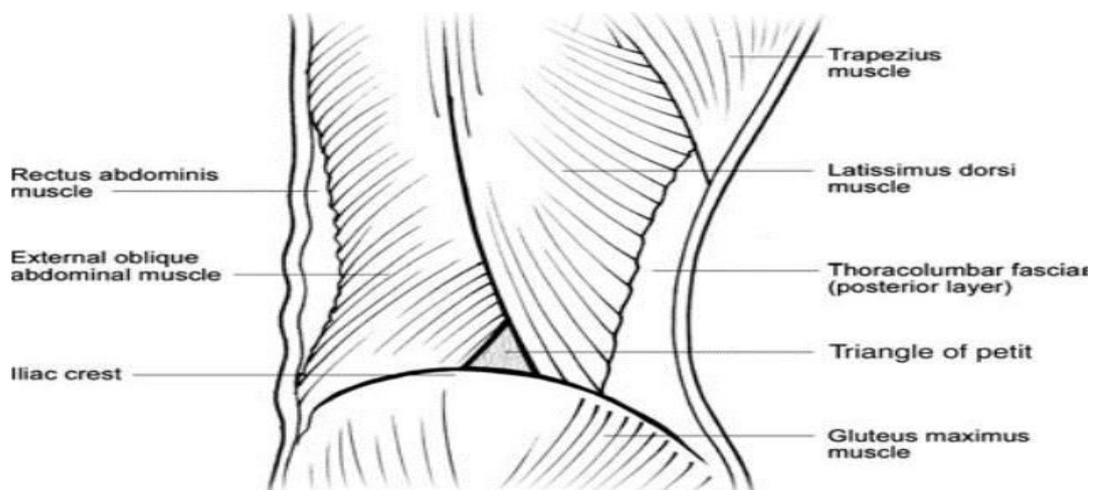


Figure. 2: The lumbar 'triangle of Petit' [35]

Ultrasound TAP blocks:

With ultrasound, the site of injection will be confirmed by giving a test dose of 0.5–1 mL 0.9% NaCl into the internal oblique and transversus abdominis muscles (figure3), and (when swollen muscle fascia will be observed) local anesthetic agents will be added into TAP (figure4) [36].

Sonographic landmarks:

The first step in performing TAP blocks with ultrasound guidance is to detect the muscles of the anterolateral abdominal wall. The external oblique is usually the most echogenic muscle of the anterolateral abdominal wall. The external oblique and internal oblique muscles typically extend farther posteriorly than the transversus abdominis muscle, Retroperitoneal fat (hypoechoic appearance on ultrasound scans) lies under the posterior feature of the transversus abdominis muscle. The layers underneath the transversus abdominis muscle are (in order) the transversalis fascia, extraperitoneal fat, and peritoneum. The quadratus lumborum muscle is hypoechoic and therefore difficult to visualize on ultrasound scans (as is the retroperitoneal fat) [37].



Figure 3: The three layers of muscles forming the anterior abdominal wall, from superficial to deep: external oblique, internal oblique, and transversus abdominis [38]

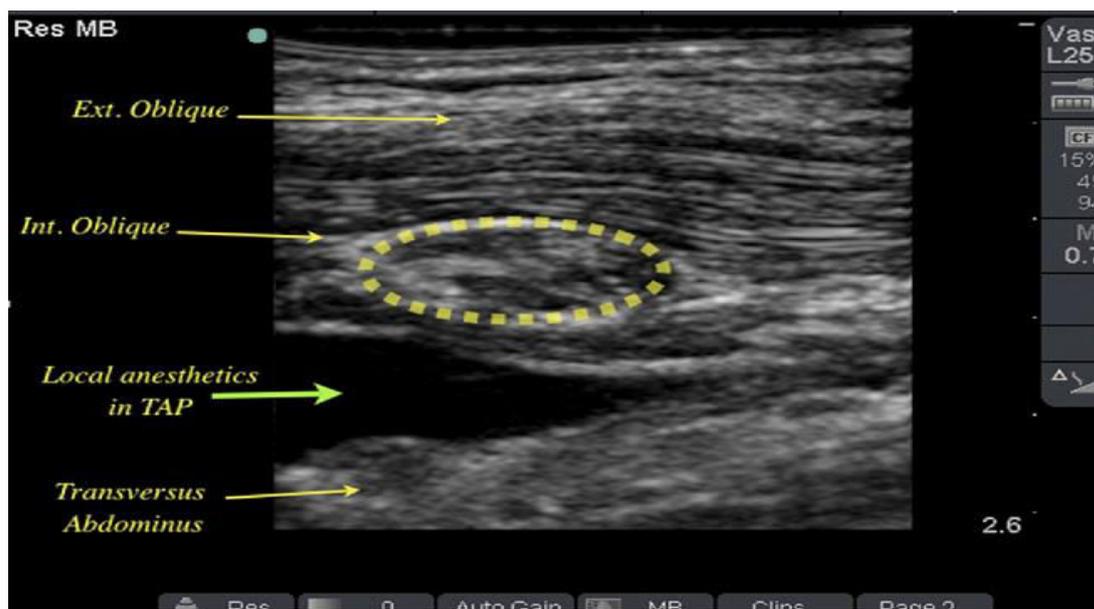


Figure 4: Local anesthetics installed in the appropriate TAP between the internal oblique and the transversus abdominis muscles. Notice the initial intramuscular injection within the internal oblique muscle (circle) [38].

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