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A Comparative Study Between Ultrasound Guided Quadratus Lumborum Block Versus Ultrasound Guided Transversus Abdominis Plane Block In Laporoscopic Bariatric Surgery

Thesis
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Contents

List of Abbreviations

ABG	**:**	Arterial blood gases
AMPA	**:**	α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
ANOVA	**:**	Analysis of variance
ASA	**:**	American Society of Anesthesiologists
ASIC	**:**	Acid-sensing ion channels
BDNF	**:**	Brain-derived neurotrophic factor
BMI	**:**	Body mass index

CD	**:**	Cluster of differentiation
C_m	**:**	Minimum concentration
CNS	**:**	Central nervous system
CO_2	**:**	Carbon dioxide
COX	**:**	Cyclooxygenase
CPP	**:**	Cerebral perfusion pressure
CXR	**:**	Chest x-ray
CYP	**:**	Cytochrome P450
ECG	**:**	Electrocardiogram
EEG	**:**	Electroencephalography
EO	**:**	External oblique muscle
ERK	**:**	Extracellular signal-regulated kinases
GABA	**:**	Gamma-aminobutyric acid
GDNF	**:**	Glial-derived neurotrophic factor
H_1 and H_2	**:**	Histamine receptors
IBW	**:**	Ideal body weight
iNOS	**:**	Inducible nitric oxide synthase
IO	**:**	Internal oblique muscle
IV	**:**	Intravenous
Ki	**:**	Inhibitory constant
L/M	**:**	Lateral/medial
L1	**:**	First lumbar vertebra

LBW	**:**	Lean body weight
LD	**:**	Latissimus dorsi muscle
M3G	**:**	Morphine-3-glucuronide
M6G	**:**	Morphine-6-glucuronide
MAO	**:**	Monoamine oxidase
MHz	**:**	Megahertz
MKP	**:**	Mitogen-activated protein kinase phosphatase
MRI	**:**	Magnetic resonance imaging
MRP2	**:**	Multidrug resistance-associated protein 2
NGF	**:**	Nerve growth factor
NGs	**:**	Neutrophilic granulocytes
NIBP	**:**	Non-invasive blood pressure
NT	**:**	Neurotrophin
OSA	**:**	Obstructive sleep apnea
PABA	**:**	Para-aminobenzoic acid
$PaCO_2$	**:**	Partial pressure of carbon dioxide in arterial blood
PACU	**:**	Post-anesthesia care unit
PCA	**:**	Patient-controlled analgesia
PFTs	**:**	Pulmonary function tests
P-gp	**:**	P-glycoprotein
pK_a	**:**	Acid dissociation constant
PM	**:**	Psoas major muscle

P-value	**:**	Probability value
QL	**:**	Quadratus lumborum muscle
QLB	**:**	Quadratus lumborum block
RA	**:**	Rectus abdominis muscle
RSD	**:**	Reflex sympathetic dystrophy
SC	**:**	Subcutaneous tissue
SpO_2	**:**	Peripheral capillary oxygen saturation
T½	**:**	Half life
T7-T12	**:**	Thoracic vertebrae
TA	**:**	Transversus abdominis muscle
TAP	**:**	Transversus abdominis plane
TLR	**:**	Toll-like receptor
TNF	**:**	Tumor necrosis factor
UGT	**:**	Uridine diphospho-glucuronosyltransferases
VAS	**:**	Visual analogue scale
Vd	**:**	Volume of distribution
X^2	**:**	Chi-square

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Introduction

Obesity has been associated with an increased hazard ratio for all-cause mortality, as well as significant medical and psychological co-morbidity. Nonsurgical management can effectively induce 5%-10% weight loss and improve health in severely obese individuals resulting in cardio-

metabolic benefit. Bariatric surgery procedures are indicated for patients with clinically severe obesity; currently, these procedures are the most successful and durable treatment for obesity. The best choice for any bariatric procedure (type of procedure and type of approach) depends on the individualized goals of therapy (e.g., weight loss and/or metabolic [glycemic] control), available local expertise (surgeon and institution), patient preferences and personalized risk stratification. At this time, there is still insufficient evidence to generalize in favor of one bariatric surgical procedure for the severely obese population. In general, laparoscopic bariatric procedures are preferred over open bariatric procedures due to lower early postoperative morbidity and mortality ***(Jeffrey et al., 2013).***

In the obese patient, the goal of postoperative pain management is provision of comfort, early mobilization and improved respiratory function without causing inadequate sedation and respiratory compromise. The pathophysiology of obesity, typical co-morbidities and the high prevalence of obstructive sleep apnea (OSA) amongst obese patients make safe analgesic management difficult. In particular, pain control after bariatric surgery is a major challenge. Although several reviews covering anesthesia and analgesia for obese patients are published, there is mainly expert opinion and a paucity of evidence-based recommendations. Advice on general management includes multimodal analgesic therapy, preference for regional techniques, avoidance of sedatives, non-invasive ventilation with supplemental oxygen and early mobilization ***(Schug and Raymann, 2011).***

In the past few years, transversus abdominis plane (TAP) block, which was first described by Rafiin in 2001, has been increasingly used for postoperative pain relief after laparoscopic surgery. As a part of multimodal analgesic regimen, TAP block results in less analgesic consumption and less pain at two hours and slightly at six hours after laparoscopic surgery in comparison with the usual opioids alone ***(Xiang et al., 2014).***

The quadratus lumborum block (QLB) was first described by Blanco in 2007. The main advantage of QLB compared to the transversus abdominis plane (TAP) block is the extension of local anesthetic agent beyond the transversus abdominis plane to the thoracic paravertebral space. The wider spread of the local anesthetic agents may produce extensive analgesia and prolonged

action of the injected local anesthetic solution. Previous studies showed that both TAP block and QLB might reduce morphine requirements in the postoperative period. However, studies comparing between trans-muscular quadratus lumborum and transversus abdominis plane blocks are of limited number ***(Rafa et al., 2015).***

Aim of the Work

The aim of this work is to study the analgesic efficacy of ultrasound-guided trans-muscular quadratus lumborum block compared with transversus abdominis plane (TAP) block and intravenous opioid drugs during laparoscopic bariatric surgery and in the early postoperative period regarding pain relief, provision of comfort, early mobilization and improved respiratory functions.

Pathophysiology of Pain

Definition of pain

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage ***(Harold et al., 2012).***

Classification of pain

Pain can be classified according to the neurophysiological mechanism, temporal aspects, etiology, or region affected. Neurophysiological mechanism of pain has been categorized as nociceptive and non- nociceptive pain ***(Khalida, 2016).***

Nociceptive pain is presumed to be maintained by continual tissue injury, it results from the activation or sensitization of nociceptors in the periphery, which transduce noxious stimulus into electrochemical impulse. These impulses are then transmitted to the spinal cord and higher rostral centers in the central nervous system. Nociceptive pain is sub-divided into somatic pain and visceral pain ***(Massieh and Karen, 2013).***

Somatic pain results from excitation and sensitization of nociceptors in tissues; such as bone, peripheral soft tissue, joints and muscles. It can be intermittent or constant, and is described as aching, stabbing, gnawing or throbbing. ***(Khalida, 2016).***

There are five physiological processes involved in the somatic nociception:

- Transduction: noxious stimuli (mechanical, chemical and thermal) act on peripheral nociceptors and are converted into electrical activity; hence, culminating in an action potential. This is carried as a nerve impulse.
- Conduction: nerve impulse travels through the length of first order neurons to reach the synapse with the second order neuron.
- Transmission: synaptic transfer of information takes place at the synapse between the first and the second order neurons in the dorsal horn of spinal cord.
- Perception: the actual conscious experience of pain, both sensory (localization, character and discrimination) and affective (emotional) aspect.
- Modulation: pain experience is not a direct and proportionate mechanical response to the noxious stimuli. A multitude of factors modulates the stimulus–response pathway ***(Clifford, 2004).***

Visceral pain has five important characteristics. Visceral organs are not sensitive to pain. It is not always linked to visceral injury (cutting the intestines causes no pain, but stretching of the bladder causes pain). It is diffuse and poorly localized. It is referred to other locations. It is accompanied by motor and autonomic reflexes; such as nausea and vomiting ***(Khalida, 2016).***

Non-nociceptive pain can be sub-divided into neuropathic and idiopathic pain. Neuropathic pain can result from injury to neural structures within the peripheral and central nervous systems. It is believed to be caused by aberrant somatosensory processing in the central and the peripheral nervous systems. Neuropathic pain is usually sharp and burning ***(Khalida, 2016).***

There are three subsets of neuropathic pain:

- Peripherally mediated pain involves the peripheral nerves and brachial plexus.
- Central pain syndrome involves the nervous system.
- Sympathetically mediated pain that can be generated centrally and peripherally; like Reflex Sympathetic Dystrophy (RSD) symptoms ***(Joachim and Clifford, 2007).***

Acute Postoperative Pain

Acute pain after surgery has a distinct pathophysiology that reflects peripheral and central sensitization as well as humoral factors contributing to pain at rest and during movement. This can impair functionality and often culminates in delayed recovery. Surgical tissue trauma leads to nociceptor activation and sensitization. As a result, individuals suffer from ongoing pain at rest and increased responses to stimuli at the site of injury (primary hyperalgesia). Different surgical procedures (including debridement for acute burn care) involve distinct organs and specific tissue within and adjacent to them, creating a variety of patterns of nociceptor sensitization and differences in the quality, location and intensity of postoperative pain ***(Timothy et al., 2017).***

Mediators released locally and systemically during and after surgery that contribute to nociceptor sensitization include; prostaglandins, interleukins, cytokines and neurotrophins (e.g. nerve growth factor [NGF], glial-derived neurotrophic factor [GDNF], neurotrophin [NT]-3, NT-5, and brain-derived neurotrophic factor [BDNF]). Decreased tissue pH and oxygen tension, and increased lactate concentration, persist at the surgical site for several days. These responses may contribute to peripheral sensitization (e.g., muscle C-fibers) and spontaneous pain behavior following an incision. Acid-sensing ion channels (e.g. ASIC3) likely transduce this ischemic-like signal. Peripheral neutrophilic granulocytes (NGs) contribute to peripheral sensitization and pain after surgical incision. Endogenous CD14+ monocyte responses (e.g., via the TLR4 signaling pathway) are associated with differences in the time course of postsurgical pain ***(Gabriela et al., 2015).***

Nerves may be injured during surgery and hence discharge spontaneously. Spontaneous action potentials in damaged nerves may account for qualitative features of neuropathic pain that may be present early in the postoperative period and can evolve into chronic neuropathic pain ***(Valeria et al., 2012).***

Noxious input during and after surgery can enhance the responses of nociceptive neurons in the CNS (central sensitization); thereby amplifying pain intensity. The magnitude of central sensitization depends on many factors, including the location of the operative site and the extent of the injury. α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)-receptor mediated

spinal sensitization contributes to pain and hyperalgesia after incision. Other molecules involved in central sensitization after surgical incision involve phosphorylated extracellular signal-regulated kinases (ERK), BDNF, Tumor necrosis factor TNFα, iNOS, mitogen-activated protein kinase phosphatase (MKP) 3, monoamine oxidase (MAO) B, toll-like receptor (TLR) 4 receptor and cyclooxygenase (COX) 2 (among others) ***(Peter et al., 2005).***

Spinal inhibitory mechanisms may be able to prevent central sensitization after surgery, for example via spinal α-adrenoceptors, γ-Aminobutyric acid (GABA) -receptors, or enhanced Glutamate transporters, among other mechanisms ***(Timothy et al., 2017).***

Anatomy of the Abdominal Wall

The abdominal wall is a continuous cylindrical myofascial structure that attaches to the thoracic cage superiorly, the pelvic girdle inferiorly, and the spinal column posteriorly. It forms a sturdy, protective musculofascial layer that protects the visceral organs and provides strength and stability to the body's trunk. The boundaries include an anterior, two lateral, and one posterior abdominal walls ***(Ki-Jinn et al., 2017).***

Anatomy of the anterolateral abdominal wall:

The anterolateral abdominal wall extends between the posterior axillary lines on either sides. The superior boundaries are the costal margin of the 7th to 10th ribs and xiphoid process of the sternum, and the inferior boundaries are the iliac crests, inguinal ligament, pubic crest, and symphysis ***(Ki-Jinn et al., 2017).*** The anterolateral abdominal wall is composed of five paired muscles: two vertical muscles (the rectus abdominis and the pyramidalis) and three layered flat muscles (the external abdominal oblique, the internal abdominal oblique, and the transversus abdominis muscles) ***(Sanjay, 2016).***