Pre-Emptive Analgesia for Reduction of Postoperative Pain

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ABSTRACT:

BACKGROUND:

Pre-emptive analgesia is important for reduction of post operating pain in all surgical procedure including inguinal herniorraphy under, general anaesthesia.

OBJECTIVE:

To compare the effectiveness of pre-emptive analgesia for reduction of post-operative pain in inguinal herniorrhaphy under general anaesthesia.

PATIENT AND METHOD:

30 adult ASA1 male patients undergoing inguinal herniorraphy under general anaesthesia were included in randomized prospective clinical study. The patients classified into two groups. Group 1 (n=15) control group receive 20 mg Ketamin Hcl at induction of anesthesia before operation. Group 2(n=15) receive 75 mg diclofenac sodium 30 min.I.M before induction and1 microgram/Kg Fentanyl citrate at induction of anesthesia. Then 20 ml of 0.25% Bupivacaine infiltrated in tissue under abdominal sheath at surgical area after induction of general anesthesia with another 20 ml of 0.25% Bupivacain was deposited in subfascial area after skin incision and. VAS (visual analog scale) score were all recorded.

RESULT:

In control group (group1) showed mark increase in stress response to post-operative pain as evidence by high visual analogue scale and in stress response to pain as increase in heart rate and mean arterial pressure during 1st 24hr post-operative. While group II patient (study group) mark decrease in post-operative pain as evidence by low VAS and insignificant change in heart rate and mean arterial blood pressure. It shows that pre-emptive analgesia reduces severity of post-operative pain and analgesic requirement in post-operative period.

CONCLUSION:

Our result indicates that pre-emptive analgesia is effective in reduction of post-operative period.

KEY WORDS: pre-emptive, analgesia visual analogue scale.

INTRODUCTION:

pain defines as unpleasant sensory and emotional experience associated with actual or potential tissue damage. These definitions recognize the interplay between the objective, physiologic sensory aspects of pain and its subjective emotional and psychological components. The response to pain can highly variable between individuals as it in the same individual at different times. (1)

tissue damage. It's most common forms include post traumatic post-operative pain.

Nociception: This term used to describe the neural response to traumatic or noxious stimuli. All nociception produces pain. But not all pain results from nociception. Clinically useful to divide pain into:

(1) **Acute pain:** This is primarily due to nociception.

(2) **Chronic pain:** which may be due to nociception but psychological and behavioral factors often play a major role (2)

Acute pain caused by noxious stimulation due to injury, disease process. It's typically associated with neuroendocrine stress that's proportional to intensity. It is serve to detect, localize and limit

Acute pain usually self-limited or resolved with treatment in a few days or weeks. When acute pain fail to resolve because of inadequate treatment or abnormal healing it will become chronic.

Neurophysiology of pain:

Stimuli generated from thermal, mechanical or chemical trauma.Tissue damage may activate nociceptors, which are free afferent nerve ending of myelinated Adelta and unmyelinated C fibers. These peripheral afferent never endings send axonal projection in dorsal horn of spinal cord where they synapse with second order afferent

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neurons. Axonal projections from second order neurons cross to contralateral side of spinal cord and ascend as afferent sensory pathway (spinothalamic tracts) to the level of thalamus. Second order neurons synapse with third order neurons, which send axonal projections to sensory cortex. [3, 5]

Modulation of Nociception

Can occur at several levels of the afferent sensory pathway prior to perception of pain at sensory level. Peripheral: occurs by liberation of endogenous mediators of inflammation in the vicinity of nociceptor. These mediators sensitize nociceptor in tissue subjected to trauma or inflammation. ***Aspirin*** and ***NSAIDS*** block Nociception at peripheral sites. [5]

Spinal Cord:

Modulation result from effect of excitatory or inhibitory neurotransmitters in the dorsal horn or by spinal reflexes.

Supraspinal:

Occur through descending efferent inhibitory pathway that originate at the level of brainstem and synapse in substantial gelatinosa of dorsal horn. An opioid descending inhibitory pathway release endorphins and encephalons, which act pre synaptically by prevent generation of action potential to next synapse and subsequent release of neurotransmitter. Opioids act as agonist in this site.

Adverse Physiological Effects Of Pain:

Respiratory: decrease lung volume, increase skeletal muscle tension, atelectasis, and ***V/Q*** mismatch, increase PaCo2, decrease $Pa0_2$,

Cardiovascular: increase ***Bp***, increase ***P.R***, cardiac dysrhythmia, myocardial ischemia.

Endocrine system: Hyperglycemia, Sodium and water retention, protein catabolism.

Immune System: decrease Immune response.

Coagulation System: decrease platelet adhesiveness, increase fibrinolysis, Hypercoagulation, deep venous thrombosis [4]

Factors Modifying Postoperative Pain:

1.Site, nature, duration of surgery.
2.Type and extent of incision.
3.Physiological, pharmacological, preparation of patient before surgery.
4.Complication, which are related to the surgery.
5.Anesthetic management.
6.Quality of postoperative care.
7. Pre-emptive analgesia, this type of management pharmacologically induces an effective analgesic state prior to surgical trauma. This may involve infiltration of wound with local anesthetic or administration of effective doses of opiods, ***NSAIDs***. Pre-emptive analgesia attenuate peripheral and central sensitization to pain, also reduce analgesic requirements post operatively [5 – 12]

Pain measurement

Reliable quantitation of pain severity helps to determine therapeutic interventions and evaluate the efficacy of treatment. The visual analog scale *(VAS)* most commonly used. It's a [10] cm horizontal line labeled "no pain" at one end and "worst pain" at other end. The patient is asked to mark on this line where the intensity of pain. The distance from "no pain" to the patient mark numerically quantities the pain.[6]

PATIENTS AND METHOD:

Our study was conducted at the Baghdad Teaching hospital and alshaffa private hospital, thirty healthy adult male patients *ASA I* and *II* undergoing elective inguinal herniorraphy. Their ages ranged from (20-50) years and their weight ranged from (50-90) Kg. They were divided randomly into two equal groups (15 patient each):

Group I (Control Group):

This group received 20mg IV Ketamin Hcl at induction of general anaesthesia and postoperative analgesia was accomplished by I.V morphine (0.1) mg/kg started after recovery from general anaesthesia when *VAS* exceed 4

Group II (Study Group):

This group received *I.M Diclofenac Sodium* (75 mg) 30 minute before surgery and 1 microgram/kg *I.V* Fentanyl citrate at induction of general anesthesia. And after induction of general anaesthesia infiltration of skin at surgical area with 20 ml of (0.25%) Bupivacaine done. Incision of skin was done 5 minutes later. And after skin incision another 20 ml of (0.25%) *Bupivacaine* was deposited in the sub-fascial area.

General Anaesthesia:

Both groups induced by 5-7 mg/kg *Thiopenton Sodium* and 1 mg/kg *Succinylcholine* to facilitate endotracheal intubation. Anaesthesia was maintained in both groups by (100%) 0_2 and *Halothane* (2.5% ± 0.5%) with spontaneous breathing throughout surgical procedure. The following data were recorded:

1.Pulse rate and mean arterial blood pressure were observed before induction, (5) minute after induction, (20) minute after induction, immediate postoperative (6hr, 12hr, 18hr, 24hr) postoperative.
2.The efficacy and duration of analgesia was determined according *VAS* (30) minute after fully awaking (1hr, 2hr, 3hr, 4hr, 5hr, 6hr, 12hr, 18hr and 24hr) postoperative.

3.The time between end of surgery and first analgesic requirement.
4.Total amount of *I.V* morphine administrated in the postoperative period.
Data were tabulated and analyzed by use of paired "T" test for compares within the same group. Unpaired "T" for compares between the two groups "T" was considered statically significant it corresponds to *P>O.05.*

RESULT:

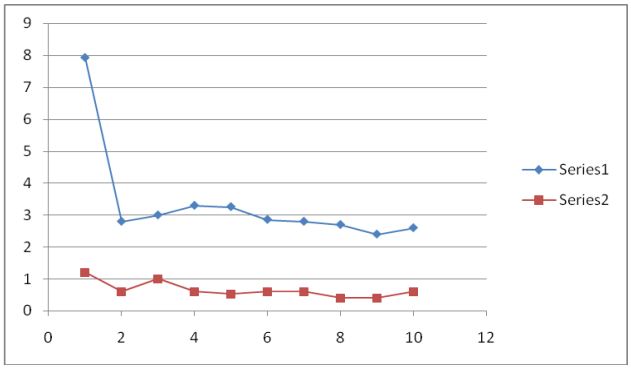
(Control Group) 15 *Patients:*

- Age range: (20-50) years (mean 33.46 ± 8.79).
- Body weight range: (55-90) kg (mean 73.8 ± 14.35).
- Morphine consumption ranged: (5-9) mg (mean 7.3 ± 2.25).
- First request for analgesia (30) minute after recovery.
- Pain intensity measured by *VAS* (7.93 ± 1.2) (30) minutes after recovery and decline to (2.8 ± 0.6) after administration of morphine.
- *H.R* and *MAP* (73 ± 5.41) and (91 ± 2.58) before induction.
- *H.R* and *MAP* (90.8 ± 1.57) and (99.33 ± 2.93) in immediate postoperative period.

Study Group (15) *Patients:*

- Age range: (21-50) years (mean 36.06 ± 8.36).
- Body weight range: (60-90) kg (mean 76.73 ± 10.09).
- No morphine consumption in postoperative period.
- No request for analgesia after recovery and 1[51] 24hr postoperative.
- Pain intensity measured by *VAS*(0.6 ± 0.18) (30) minutes after recovery and remains close to (1) during (24) hr postoperative period.
- *H.R* and *MAP* (71 ± 1.71) and (90.86 ± 1.71) before induction and remains unchanged all over (24) hr.

There was significant unchanged in *H.R* and *MAP* in study group when compared to control group postoperative



Series 1 Control group	30 min after recover	1hr	2hr	3hr	4hr	5hr	6hr	12hr	18hr	24hr
	1	2	3	4	5	6	7	8	9	10
	7.93	2.8	3	3.3	3.26	2.86	2.8	2.7	2.4	2.6
series 2 study group	30 min after recover	1hr	2hr	3hr	4hr	5hr	6hr	12hr	18hr	24hr
	1	2	3	4	5	6	7	8	9	10
	1.2	0.6	1	0.6	0.53	0.6	0.6	0.4	0.4	0.6

Fig 1: Visual analogue scale of control and study group.

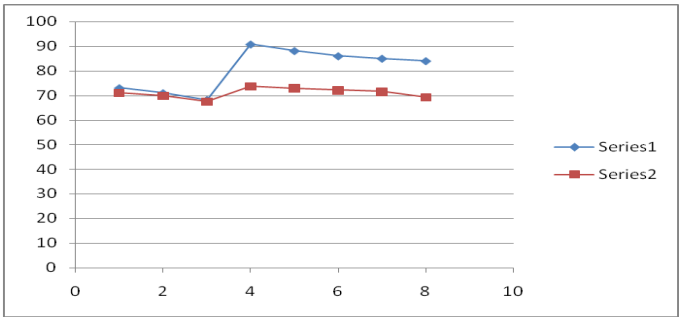
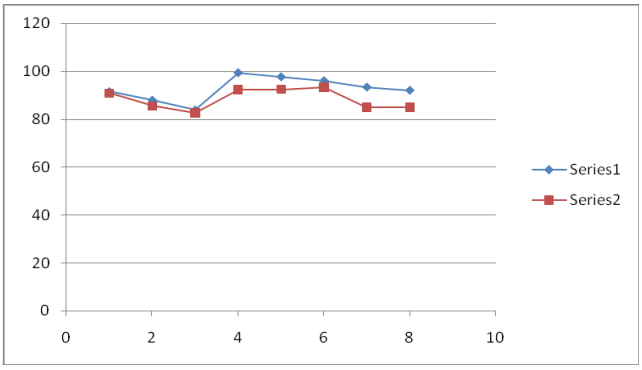


Fig 2: Heat rate (beat/min) of the control and study group.

Series 1 Control group	Before induction	5 min after induction	20 min after induction	Immediate Post O.P	6hr post O.P	12hr post O.P	18hr post O.P	24hr post O.P
	1	2	3	4	5	6	7	8
	73	70.93	68.13	90.8	88.13	86	85	84
series 2 study group	Before induction	5 min after induction	20 min after induction	Immediate Post O.P	6hr post O.P	12hr post O.P	18hr post O.P	24hr post O.P
	1	2	3	4	5	6	7	8
	71	69.8	67.53	73.66	72.93	72.13	71.53	69.3



30 min after recover	1hr	2hr	3hr	4hr	5hr	6hr	12hr	18hr	24hr
Mean 0.6	0.73	0.86	0.86	0.86	0.86	0.86	1.53	1.55	1.26
± S.D	0.2	0.26	0.26	0.26	0.26	0.26	0.6	0.6	0.33
T1 21.63*	22.3*	12.7*	11.5*	8.3*	7.8*	7.3*	6.9*	6*	5.5*

Fig 3: Mean arterial blood pressure (mmHg) of the control and study group.

Table 1: Comparison of VSA in both Groups.Postoperative.

Group I

Series 1 Control group	Before induction	5 min after induction	20 min after induction	Immediate Post O.P	6hr post O.P	12hr post O.P	18hr post O.P	24hr post O.P
	1	2	3	4	5	6	7	8
	91.4	87.9	83.8	99.3	97.6	96	93.3	92
series 2 study group	Before induction	5 min after induction	20 min after induction	Immediate Post O.P	6hr post O.P	12hr post O.P	18hr post O.P	24hr post O.P
	1	2	3	4	5	6	7	8
	90.8	85.46	82.6	92.2	92.33	93.3	85	85

Group II.

30 min after recover	1hr	2hr	3hr	4hr	5hr	6hr	12hr	18hr	24hr
Mean 1.93 ± S.D 1.2	2.8	3	3.3	3.26	2.86	2.8	2.7	2.4	2.6
	0.6	1	0.6	0.53	0.6	0.6	0.4	0.4	0.6

DISCUSSION:

The concept that analgesia given before the painful stimulus has effects that long outlasts the presence of the analgesic in body created the basis for pre-emptive treatment of pain. The aim of such treatment is to prevent the spinal cord from reaching hyperexcitable state in which it responds excessively to afferent inputs.

It has been shown that administration of opiods before induction of anaesthesia may decrease the need for paranteral analgesic postoperatively. This is consistent with the concept that activation of afferent pain pathway, especially in lightly anaesthetized patient, produces changes in ***C.N.S*** that subsequently lead to amplification and prolongation of postoperative pain. Indeed, the dose of opioid require to prevent **C** fiber induced excitability changes in the spinal cord is lower than that require to suppress these changes once it occurs. So opioid given ***I.V***just before induction may be more logical.

Our study showed very mark in decrease postoperative pain. There is no morphine consumption in postoperative period in study group who received (75) mg I.M diclofenac Sodium (30) minute before induction, (1) Mg /Kg Fentanyl citrate LV just before induction of anesthesia, (20) ml of (0.25%) Bupivacaine infiltrated in surgical area. Before and after skin incision, also in study group there is decrease stress response to postoperative pain as showed by insignificant change in heart rate and mean arterial pressure at immediate postoperative period and 1st24hr postoperatively.

The result of our study agrees with study of ***Mogensen*** *(3),* ***Bugedo***(4), ***Woolf*** (5), ***Campbell***(6). Our study was done in healthy patient ***ASAI*** and ***ASAII*** for those patients with underlying cardiovascular diseases such as hypertension, coronary artery disease.

Provision of analgesia postoperatively by pre-emptive treatment of pain the decrease stress response to surgery by prevention of sympathetic out from ***C.N.S*** that lead to stabilization of heart rate and mean arterial blood pressure and prevent tachycardia and hypertension and ischemic attack. Also provisions of analgesia improve wound healing and ensure early mobilization and prevent thrombus formation.

McQuay(7), ***Yeager***(8) had studies coincides with our result.

Their clinical date, suggests that preventing painful impulses from being processed in the ***CN.S*** gives better results than treating pain after it has been perceived in the ***CN.S***.

Reuben and colleagues have also investigate several novel routes of administration of NSAIDS

and other for postoperative analgesia (e.g. intrarticular, local, and regional analgesia). One of the controversial area of research related to potential adverse
effect of NSAIDS and COX-2 inhibitors on bone healing, Reuben reported that perioperative (short term) administration of celecoxib had no apparent effect of nonunion at time of 1 year follow up evaluation(19).

CONCLUSION:

pre-operative administration of opioids , ***NSAIDs***. *And* Local infiltration of the surgical area with local anesthetic markedly decreasethe degree of postoperative pain, decreasestress response to pain and decrease analgesic requirement postoperatively.

Recommendation:

I recommended that given preoperative analgesia with NSAIDS, opioids and infiltration of surgical area with local anesthetic agent will decrease postoperative pain and decrease postoperative analgesic requirement

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