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EDITORIAL

Glycemic Control in Neurosurgical Patients

With the growing body of research on the adverse outcomes of hyperglycemia or hypoglycemia, the significance of maintaining glucose homeostasis in surgical patients cannot be overemphasized. Moreover, this has added impact in neurosurgical patients due to a difference in cerebral and systemic glucose metabolism as well as the impact of serum glucose levels on the surgical outcome. Studies have clearly demonstrated that hyperglycemia was associated with increased risk of poor outcome.[1] On the other hand; perioperative blood glucose control with intensive insulin therapy (IIT) in neurosurgical patients has not been associated with significantly improved outcome.[2] Moreover, it leads to an increased incidence of hypoglycemia with associated risks, including neurological deterioration.[3] Thus, a clinical consensus is required about the methodology of perioperative glucose control in the neurosurgical population.

Furthermore, the heterogeneity of pathologies in neurologic surgery suggests caution in the generalization of other study results to this patient population. The optimum level of blood glucose level in this group of patients is still debatable. Till any guideline is developed, the consensus statement of the American Association of Clinical Endocrinologists and the American Diabetes Association may be of help. It has recommended in critically ill patients, to start IV insulin therapy at a threshold of >9.9 mmol/L (180 mg/dL) and maintain the glucose level between 7.7 mmol/L (140 mg/dL) and 9.9 mmol/L (180 mg/dL).[4]

Greater benefit may be obtained at the lower end of this range. However, glucose concentrations <6.2 mmol/L (110 mg/dL) are not recommended.[2]

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GLYCEMIC CONTROL IN NEUROSURGICAL PATIENTS

LD Mishra[1], Nang Sujali Choupoo[2]

Abstract: Maintaining glucose hemostasis is of paramount importance in perioperative period particularly in neurosurgical patients.The unique pathophysiology of brain injury pauses tremendous challanges in doing so. This article reveiwes the physiology of glucose metabolism in brain, pathophysiology of hypo and hyper glycemia in neurosurgical patients and suggested optimum blood glucose level in such patient.
Key words: Hyperglycemia; Hypoglycemia; Neuro surgery

Introduction

Hyperglycemia and glucose intolerance are common manifestations of perioperative stress in many hospitalized patients. An increase of only 1.1mmol/L (20 mg/dL) in the mean intraoperative glucose was linked to an increase of more than 30% in adverse outcomes.[1] The deleterious effect of hyperglycemia includes delayed wound healing, increased cardiac morbidity, renal dysfunction, postoperative length of stay, and poor neurological outcomes etc. Although hyperglycemia induces poorer recovery of neurologic function, symptomatic cerebral vasospasm, and increased infarct size as well , it is uncertain whether plasma glucose level is primarily a surrogate marker of disease severity or a causative agent of damage exacerbation.[2]

Physiology of glucose metabolism in brain

The brain is critically dependent on a continuous supply of both oxygen and glucose for *normal* metabolic function. Glucose undergoes glycolytic breakdown to adenosine triphosphate (ATP) and pyruvate under aerobic conditions. Pyruvate is converted to acetyl-CoA via the tricarboxylic acid (TCA) cycle to generate ATP and reducing equivalents. When oxidative phosphorylation is impaired (lack of oxygen, mitochondrial dysfunction, brain injury), pyruvate is instead converted to lactate. Most of these biosynthetic reactions are under tight feedback control.

Experimental traumatic brain injury studies have shown that cerebral hyperglycolysis is a pathophysiological response to injury-induced ionic and neurochemical cascades[3]. The pyruvate generated, in the absence of functioning mitochondria, is predominantly converted to lactate causes an intracellular lactic acidosis. Episodes of cerebral hyperglycolysis may be linked to low extracellular brain glucose despite peripheral blood hyperglycemia. In this clinical situation, further lowering of blood glucose values could theoretically decrease brain glucose levels below critical values. Recent studies, both in vitro and in vivo, have demonstrated that lactate is an excellent aerobic energy substrate in the brain, and aerobic lactate utilization is crucial for neuronal recovery in the immediate post-ischaemia period. An alternative explanation tends to incriminate glucocorticoids for the aggravating effect of pre-ischemic hyperglycaemia. This hypothesis states that hyperglycaemia (or glucose loading) results in a transient elevation in the release of glucocorticoids, which is responsible for worse outcome.[4]

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Effect of hyperglycemia during brain injury: The effects of hyperglycemia on injured brain are multifold:

- It causes diuresis sufficient to produce hypovolemia can produce hypotension, decreased cerebral perfusion and electrolyte imbalances. Hyper osmolarity associated with hyperglycemia can alter mental status or produce seizures. Similarly, hyperglycemia may result in an autoregulatory decrease in CBF that could worsen brain ischemia.
- Chronic hyperglycemia, as occurs in diabetes mellitus causes cognitive impairment
- Hyperglycemia is associated with disruption of the blood brain barrier (BBB) in rodents which diffusion of excess calcium, lactate, and glutamate.
- pro-inflammatory effects of hyperglycemia are likely mediated by advanced glycation end products and there is secretion of tumor necrosis factor-α and interleukins.

Effect of hypoglycemic on brain:

➢ Effect of hypoglycemia is equally deleterious on normal brain function which exaggerates during any insult to brain.

➢ It is worth mentioning that cerebral hypoglycemia may occur without associated peripheral blood hypoglycemia. The fundamental pathology in hypoglycemia is necrosis; this differs from the injury that occurs in ischemic infarction

➢ Profound hypoglycemia induces a coma that, if maintained for >30 min, is associated with irreversible brain damage. There is isoelectric EEG and neuronal cell death when glucose concentration fall below 1.4mmol/L (25 mg/dL).

➢ Neuronal susceptibility to hypoglycemic injury is tissue specific. The hippocampus and cerebral cortex are highly susceptible to hypoglycemia-mediated damage, whereas the cerebellum, in particular, is relatively resilient

➢ Hypoglycemia increases cerebral blood flow and further worsen an already injured brain

Some recent studies showing effect of hyperglycemia in neurosurgical patients[2]

Author	Diagnosis	Year	Out come
Kimura et al[5]	ICH	2007	↑ Risk poor outcome/death
Frontera JA et al[6]	SAH	2006	↑ Risk poor outcome/death
Badjatia et al[7]	SAH	2005	↑ Risk symptomatic vasospasm
Kerner et al[8]	SAH	2007	↑ Risk poor outcome
Schlenk et al[9]	SAH	2008	↑ Risk symptomatic vasospasm
Stead et al[10]	Ischemic stroke	2009	↑ Stroke severity and poor outcome only in nondiabetes subjects
Poppe et al[11]	Stroke	2009	↑ Admission blood glucose associated with poor outcome after TPA
Woodworth et al[12]	Spinal cord tumour	2007	*Preoperative* ↑ blood glucose associated ↓ post-operative functional status

Glucose Control during Perioperative Period in Neurosurgery:

The benefits of improved blood glucose control after neurosurgery include a lower rate of craniotomy wound infections, reduced length of stay, and reduced hospital cost. Decreased bloodstream and nosocomial infections, acute renal failure, ventilatory support, blood transfusions, critical illness polyneuropathy, and duration of stay in the neurocritical intensive care unit have also been demonstrated.[13]

Clinical consensus is required regarding perioperative glucose control in the neurosurgical population. Furthermore, the heterogeneity of pathologies in neurologic surgery suggests caution in the generalization of other study results to this patient population: peripheral and cerebral glucose values do not correlate or correlate inversely; a normal cerebral glucose level is poorly defined, and the optimal level in the presence of anesthesia or brain pathology is unknown. In addition, no data exist to guide any type of differential management with intensive insulin therapy or fluid therapy in neurosurgical patients with or without preexisting DM.

Intensive Insulin Therapy and Outcome in Neurosurgical Patients[2]

Author	Target glucose	Outcome
Ho et al[14]	4.1 - 7.5	↓ ICP, ↓ L/P
Schlenk et al[9]	<7.7	↓ ECGlc; ↔ L/P
Theile et al[15]	4.9 - 6.6	No Δ mortality with IIT, small ↑ risk of death with hypoglycemia
Godoy et al[16]	3.3 - 8.3	↓ Mortality with IIT only after the first 12 h
Van den Berghe et al[17]	4.4 - 6.1	↓ ICP, ↓ vasopressors, ↑ seizures, ↑ neuro outcome 12 months

L/P, lactate/pyruvate; ECGlc, extracellular glucose

Careful preoperative evaluation is essential in patients with DM to identify previously unknown complications and to manage co morbidities. Before a planned surgical procedure, the patient's blood glucose should be as close as possible to that advocated by the American Diabetes Association. These targets include glycosylated hemoglobin (Hgb A1C) <7.0%, average (5.0 and 7.2mmol/L), and average postprandial plasma glucose <180mg/dL (10.0mmol/L)[18]

Hyperglycemia during neurosurgical procedures is best managed with a continuous IV insulin infusion. Following two regimen can be used:

Reactive regimen (according to monitorized values)[13]

Glucose value (mmol/L)	Insulin dose IU
<8.3	
8.3 - 11.1	5
11.1 - 13.8	10
13.8 - 16.7	15
16.7 - 19.4	20

Proactive regimen[13]

Glucose value (mmol/L)	Insulin infusion rate IU/hr
8.3-9.3	2
9.4- 11.0	3
11.1 -13.8	4
13.8 -16.6	6
16.7- 22.1	8
22.2+	10

Postoperatively, the insulin infusion is continued at the physician's discretion. In the presence of stable blood glucose, insulin drip is converted to a subcutaneous insulin regimen with a basal insulin dose and bolus/nutritional insulin dose.

When transitioning from IV to SC insulin, the drip should continue and overlap with the first SC dose of long-acting (basal) insulin for two to four hours. Failure to overlap IV and SC insulin can result in extreme hyperglycemia or diabetic ketoacidosis (DKA). Usually, Oral hypoglycemic agents are restarted after the first proper food intake if there are no contraindications.

Hypoglycemia can be managed by 25-50% dextrose infusion in the following way: (100 − glycemia) × 0.3 = mL in IV bolus Check plasma glucose every 30 minutes If glucose < 3.3mmol/L (60mg/dL), repeat the IV bolus stepTarget glucose level:

Therapeutic modality directed to keep blood glucose between 4.4mmol/L (80mg/dL) and 6.2 mmol/L (110mg/dL) called “intensive insulin therapy” (IIT). In 2001, Van Den Berghe showed significant decreased in mortality and morbidity in patient treated with IIT.[17]

But a recent study published in 2009, NICE-SUGAR trial showed contradictory result[19]

A consensus statement of the American Association of Clinical Endocrinologists and the American Diabetes Association has recommended in critically ill patients,to start treatment at a threshold of >9.9 mmol/L (180mg/dL), preferably with IV insulin therapy, and maintain the glucose level between 7.7 mmol/L (140 mg/dL) and 9.9 mmol/L (180mg/dL). Greater benefit may be obtained at the lower end of this range. Glucose concentrations <6.2 mmol/L (110mg/dL) are not recommended.[13]

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TRACHEAL GRANULOMA AS AN INCIDENTAL FINDING DURING ENDOTRACHEAL INTUBATION

Priyanka Patil[1], Vipul Patel[2], Ravi Jain[3], S.Choudhary[4], Priti Patel[5], Jayshree Thakkar[6], Bipin Patel[7]

ABSTRACT

Intubation granuloma of trachea is an iatrogenic condition which is induced by endotracheal intubation. The incidence has been reported to range from 0.01% to 3.5%. The endotracheal tube may rub the inside of trachea, leading to mucosal irritation and causing granulation tissue formation. In some cases granulation tissue may cause tracheal stenosis. It can be managed by conservative medical treatment with observation or surgical excision. A case of tracheal granuloma was found incidentally after administration of general anaesthesia in previously operated case of pituitary adenoma. Patient was managed by tracheostomy in intraoperative period and treated medically with steroids during post operative period.

Key words: Granuloma, Intubation

INTRODUCTION

Despite significant improvements in biocomatibility of materials used in translaryngeal tubes and efforts to decrease trauma associated with long-term intubation and tracheostomy, laryngeal and tracheal lesions continue to be problematic in patients requring prolonged airway support.[1] Abnormal healing of injured airway mucosa can result in granuloma formation. Granulation tissue may form in the trachea and dangerously block the airway due to tracheal narrowing. The body makes granulation tissue during the first stage of healing process. The tissue contains fibrous connective tissue and new blood vessels. This type of tissue forms usually over several days after trauma. Because of the angiogenesis granulation tissue can bleed easily, increasing the chances of aspiration. Eventually granulation tissue turns into scar and may cause permanent tracheal stenosis.[2]

CASE REPORT

A 60 year old, non diabetic, non hypertensive male patient of 60 kg weight with history of tuberculosis 20 years back had transnasal transsphenoidal pituitary adenoma resection two months back. Surgery was done under general anaesthesia. Endotracheal intubation was done with 40FG reinforced, nonkinkable Armoured tube. Endotracheal tube was kept for 10 hours following surgery. Patient had CSF rhinorrhea following surgery and was scheduled for CSF fistula repair surgery. He was premedicated with tab Diazepam 5 mg orally on the night before surgery. Intraoperative monitoring was done in the form of operation theatre Electrocardiogram, Pulse oximetry, Capnography and Noninvasive blood pressure were started. Anaesthesia was induced with intravenous fentanyl 100µg, thiopoentone 350 mg and vecuronium 6mg after 3 minutes of 100% pre oxygenation. IPPV seemed to be difficult and patient was intubated immediately with Armoured no 38 endotracheal tube and fixed at 21cm. Intubation was uneventful but there was resistance in bag ventilation. On chest auscultation bilateral air entry was minimal. With inspiratory pressure of more than 40 cm H2O was also it was difficult to ventilate the patient. Immediately endotracheal tube was changed and the patient was reintubated with red rubber no 10 endotracheal tube to rule out external pressure effects on the Armoured endotracheal tube. Mean while Injection deriphylline and dexona were given intravenously to rule out and correct bronchospasm if present. Resistance persisted even after change of tube. Saturation fell to 91%. As there was no improvement with all these measures, intratracheal pathology was suspected and immediate tracheostomy done. Immediately after tracheostomy, ventilation improved along with and bilateral air entry and oxygen saturation. Peak inspiratory pressure decreased to 10 cm H2O for ventilaing lungs effectively. Anaesthesia was maintained with Vecuronium 3.2 mg/hr, Sevoflurane 2-4%, Oxygen and Nitrous oxide 50% each. Anaesthesia along with reversal was uneventful during the rest of period. Patient was shifted to Neuro ICU. Patient remained stable maintaining his vitals.

Pulmonologist opinion was taken and patient was referred for bronchoscopy. Bronchoscopy report showed tracheal stenosis just above the tracheostomy tube with polypoidal mass. Right vocal cord paresis was present. Tissue was taken and sent for histopathological examination and CT scan neck was advised. CT scan of neck showed tracheal narrowing above tracheostomy tube measuring around 29x19mm sized heterogenously hypodense soft tissue density lesion involving trachea for a length of 20mm extending just below post cricoid region causing near complete obliteration of tracheal lumen. The lesion was abutting the oesophagus posterorly with focal loss of fat plane. Patient was kept on Ryle's tube feeding as oral feeding resulted in regurgitation through tracheostomy tube.

Histology report showed inflammatory granuloma with edematous interstitial tissue. In the post operative period patient was managed with steroids inj dexametasone 8mg tds iv, antibiotics and antiinflamatory medications. On the 20th postoperative period bronchoscopy was repeated, which showed no polyp/mass/granuloma proximal or distal to tracheostomy site with normal bilateral vocal cords movements. Fiberoptic oesophagoscopy showed no evidence of tracheooesophageal fistula. Granuloma resolution had occured. Tracheostomy tube was removed, oral feeding was attempted. There was no regurgitation and no difficulty in breathing also.

DISCUSSION

Airway granuloma is an uncommon complication arising from the irritation of mucosa and submucosa. First case of such case of intubation granuloma was reported by Clausen[(3)] in 1932. Since that time several reports and investigation of possible pathogenesis, prophylaxis and treatment has been discussed. Blanc and Tremblay[4] performed comprehensive study of the complications of tracheal intubation. Predisposing factors include age, female sex, anatomical characteristics and fragility of upper airway mucosa and gastro-esophageal reflux disease.[(5)] Other risk factor includes intubation duration and presence of nasogastric tube. The pathogenesis includes traumatic injury to larynx and trachea with destruction of unsupported surface epithelium and destruction of basement membrane resulting in hindered healing.[6] Shear stress from patient or tube movement can lead to deeper tracheal damage and possible colonization. The duration of endotracheal intubation required to result in granuloma is reported to be as short as 4.5 hours.[7] Nasal intubation provides stability to tube as compared to oral intubation where subtle movement may increase stress to larynx and trachea. Barton similarly studied the cases of airway trauma and found out both oversized tube or excessive cuff pressure can produce trauma.[8] Stanley et al [9] demonstrated that significant volume changes occur when cuff is exposed to N_2O and can result in glottis and sub glottic trauma. Even following minimum traumatic intubation infection can result in abraded tissue compounding the chances of decreased healing, ulcer and granuloma formation. Inflamed area becomes ulcerated secondarily and ultimately become a sessile granuloma. The granuloma then becomes pedunculated as peripheral fibrosis and healing occurs.

Symptoms develop on an average after 4-6 weeks of extubation as scarring and granuloma forms. Hoarseness is the most common symptom. Patient can present with exertional dyspnoe, husky voice quality, stridor and dry cough or heavyness on the throat. Our patient was not having any significant positive complaint of any of above symptoms and was maintaining normal spontaneous breathing. Size of granuloma, occupying tracheal lumen, associated nerve palsy alongwith remains determining factor as far as presenting clinical symptoms are concerned. In case of small granuloma patient may remain asymptomatic. Complete airway obstruction and aspiration of dislodged granuloma are the most dreaded complications.In a patient with positive history of prolonged intubation, indirect laryngoscopy, direct laryngoscopy or even fiberoptic bronchoscopy, laryngotracheal tomography and laryngeal electromyelography can be helpful to rule out possible airway pathology.[10]

Most granuloma would resolve by 8-14 weeks after extubation. Treatment therapy may include medical therapy with proton pump inhibitor, antireflux measures, systemic or topical steroids, antiinflammatory medication, antiallergic medication, cessation of smoking, botulinum toxin injection, antibiotic, voice rest and speech therapy. A recent study demonstrated 85% resolution of granuloma with inhalation budesonide within six months without

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with inhalation budesonide within six months without significant side effects. Surgical excision is warranted if the patient has airway obstruction, dyspnea on exertion, nocturnal dyspnea or histopathology specimen is required foe correct diagnosis. A combined micro surgical technique with CO_2 laser is most commonly used.[9] Care must be taken to remove the granuloma without damaging the normal mucosa and further exposing the perichondrium. Recurrence rate following surgical removal have been reported to be high.

CONCLUSION

Airway granuloma though rare complication of endotracheal intubation can be disastrous. Prophylactic measures are far more important than treatment. Avoidance of prolonged intubation and manipulation of tube in patient, proper sizing of tube and cuff pressure are the steps to ameliorate endotracheal anesthesia. Immediate recognition of the symptoms and detailed history of previous anesthesia with positively inquring about signs and symptoms of possible airway complications will further help the process. Lastly, steroids are quite effective in the management of such traumatic granulomas decreasing the need for surgery.

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INCIDENCE OF POST-DURAL PUNCTURE HEADACHE IN CAESAREAN SECTION : 23 VS 26 GAUGE QUINCKE'S SPINAL NEEDLE.

Parvaiz Ahmad[1], Ayaz Farooqi [2], Waqar-ul-Nisa[3]

ABSTRACT

100 female patients aged 20 – 40 years with ASA grade I and II, scheduled for elective caesarean section were divided into two groups of 50 each.Patients in group I recieved spinal anaesthesia with 23 gauge qunicke's spinal needle, whereas in group II, patients were given spinal anaesthesia with 26 gauge needle. The two groups were compared for incidence of post-dural puncture headache keeping all other variables constant.It was found that the incidence of post dural puncture headache was higher in group I (28%) as compared to group II (6%).It was therefore concluded that smaller gauge needles are associated with lesser incidence of post-dural puncture headache.

Keywords:

Aneasthesia: Obstetric, Anaesthetic technique: Spinal, Complication: Headache, Equipments: Quincke's spinal needle.

Introduction:

Spinal analgesia is being practiced clinically since 1889.It was first introduced by Augest Bier and since then the technique has been widely practiced to provide anaesthesia, particularly for surgery below umbilicus. The main advantage attributed to this technique are its simplicity, ease of performance, requirement of minimal apparatus, minimal effect on blood chemistry and optimum level of blood gases.Besides, patient remains conscious during surgery, maintains airway and requires minimal postoperative care and analgesia.Spinal anaesthesia is also not without disadvantages. Since the introduction of spinal analgesia, headache has remained the most distressing and a well known complication.[1, 2] A few decades ago less refined and thicker spinal needles were being used and the incidence of post dural puncture headache was high.But within the last 15 years more refined and thinner needles have been used more often and the incidence of postdural puncture headache is grossly reduced to less than 3-5%.[3,4] The incidence of post dural puncture headache is found more in females particularly after caesarean section in young parturient.[5,6]

The present study was undertaken to compare the incidence of postdural puncture headache, using 23 gauge and 26 gauge Quinke`s spinal needle in patients undergoing elective caesarean section under spinal anaesthesia.

Material and Methods:

This study was carried out in the Department of Anaesthesiology and Critical Care SKIMS Srinagar. 100 patients of ASA grade I and grade II aged 20-40 years, scheduled for elective caesarean section were studied.

Before including the patients to the study, detailed written informed consent was taken. Patients were divided with two groups of 50 each.

Group I : Patients who received spinal anesthesia with 23 gauge Quincke's Spinal Needle.

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Group II: Patients who received spinal anesthesia with 26 gauge Quincke's spinal needle.

Patients with history of hypertension, migraine, chronic headache, coagulopathy or any other contraindication to lumbar puncture were excluded from the study. Patient who required more than one lumbar puncture were also excluded from the study.

All the patients were clinically evaluated and routine investigations, including coagulation profile was done. No premedication was given to any patient. On arrival in operation theater, an 18 gauge cannula was secured in the peripheral vein and an infusion of 500ml RL was given prior to spinal anaesthesia. Patients were connected to ECG monitor, pulse oximeter and non-invasive blood pressure monitor. Baseline reading of heart rate, blood pressure and oxygen saturation were taken 5 minutes before giving spinal anaesthesia and thereafter monitored every 3, 5 and 10 minutes after giving the spinal anaesthesia.The haemodynamic variables were monitored thereafter regularly at five minutes interval until completion of surgery. Under strict aspectic precautions lumbar puncture was done at L2-L3 or L3-L4 interspace using either 23 gauge or 26 gauge Quinke's spinal needle in the sitting position using midline approach and 2.5ml of 0.5% hyperbaric bupivacaine was injected. After withdrawal of the needle patients were turned to the supine position with left lateral displacement.

The patients were interviewed post-operatively on day 1, 2, 3 4, 5, 6 and 7 for the incidence of postdural puncture headache.

Headache was labeled as postdural puncture headache as per following criteria:

- Aggravated by erect or sitting position and coughing, sneezing or straining.
- Occurred after mobilization
- Relieved by lying flat
- Mostly localized on occipital, frontal or generalized.

At the end of the study data was compared and statistically analyzed using appropriate parametric and non-parametric tests which include student's t-test, Mann Whitney test and chi-square test.

Observations

Comparison of incidence of postdural puncture headache between the two groups.

	Group I		Group II			
Incidence	N	%	N	%	P value	Remarks
Yes	14	28.0	3	6.0	0.004	Sig

Of the 50 patients in group-I, 14 were complicated by postdural puncture headache. The overall incidence of postdural puncture headache in group I was 28%. In group II only 3 patients complained of headache and over all incidence of postdural puncture headache was 6%.

The difference observed in the incidence of postdural puncture headache between the two groups was statistically significant with a p value of (0.004).

Discussion :

The incidence of post spinal headache varies from 0.4% to 37.2% as reported by various authors.[3,4,5] The most important factor contributing to the higher incidence of post dural puncture headache has been reported to be the gauge and type of needles used. Thicker the needle, and more traumatic the type of needle (cutting type), more will be the incidence of the post spinal headache.[7]

Evidence suggests that the post dural puncture headache is due to a low cerebrospinal fluid pressure consequent upon seepage of cerebrospinal fluid through the dural puncture hole and choroid plexus is unable to secrete sufficient fluid to maintain the cerebrospinal fluid pressure. Moreover the negative pressure in the epidural space may draw cerebrospinal fluid from subarachnoid space. Cerebrospinal fluid leakage from the dural hole produces cerebrospinal fluid hypotension, which in turn leads to intracranial venous dilation resulting in an increase in brain volume in the upright position. There occurs a difference in cerebrospinal fluid volume and also pressure differential between the intracranial and intra-vertebral part of the subarachnoid space. Venous dilation and compensatory increase in brain volume will result in brain sag which in turn will exert traction and stimulate pain sensitive anchoring structures like dural vessels, basal dura and tentorium cerebella, causing post spinal headache. Larger the hole in dura mater, more will be the leakage of Cerebrospinal fluid and longer the time required for repair. The number of holes in the dura also makes a difference in the loss of cerebrospinal fluid. It takes about two weeks or more for the holes to seal.[8]

The incidence of post dural puncture headache in our study was 28% in group I, and 6% in group II. The difference is statistically significant.

The incidence of headache is more common among women than men, particularly prone are parturient, because of the reduction of both, the intra- abdominal and epidural pressures after delivery, thereby promoting extra leakage of cerebrospinal fluid than usual. Sex bound difference is caused by emotional and hormonal factors also(5),(6).

Spielmann (1982) mentioned the factors responsible for an increased incidence of post dural puncture headache in obstetric patients which include stress of labour, changing hormonal level, and dehydration. It is because of this reason, that this study was conducted in patients undergoing caesarean section.

Age of the patients did not play any significant role in our study since all the patients in two groups were of the like age groups. However the incidence was found to be lower in older patients. In older patients an altered pain sensitivity of vascular pain receptors and narrowed route of escape of cerebrospinal fluid from the epidural space are assumed to be the explanation for lower incidence.[9]
We conclude that the ease and certainty of dural puncture with a 26 gauge quincke's spinal needle combined with low incidence of postdural puncture headache makes 26 gauge needles a suitable choice for spinal anaesthesia.

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A RANDOM PROSPECTIVE STUDY TO EVALUATE POST-OPERATIVE NAUSEA AND VOMITING IN CHILDREN

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SUMMARY

The risk factors studied for association with PONV were age>5 years, female gender, previous history of PONV/motion sickness, type of surgery and duration of anaesthesia >45 min. All 100 ASA grade I and II patients of either sex aged between 2-12 years underwent similar anaesthesia protocol and received two antiemetic agents (ondansetron 0.05mg.kg^{-1} and dexamethasone 0.15mg.kg^{-1}) in premedication. The patients were observed for 24 hours post-operatively for the incidence of vomiting and adverse reaction to antiemetic. Overall 34% patients developed PONV of which 26 had only one episode and 8 had 2 episodes during first 24 hours. Incidence of PONV was 13% in first 4 hours whereas it was 29% in the late post-operative period. In early post-operative period, PONV was not associated significantly with any predicted risk factors. However, age >5 year, duration of anaesthesia >45 minutes and history of motion sickness/PONV were significantly associated in late post-operative period (4-24hr.). Female gender and type of surgery were not associated with increased PONV. The combination of antiemetic effectively prevented PONV in early post-operative period (0-4Hr) only but not in late post-operative period (4-24hr.).

Key Words
Paediatric anaesthesia; vomiting; Post-operative, Ondansetron;Dexamethasone

INTRODUCTION

Post-operative nausea and vomiting (PONV) is most common and unpleasant side effect after surgery. Incidence of PONV has decreased now a days as compared to past. However, incidence of PONV in children remains higher.[1]

PONV not only distresses the child but also decreases parental satisfaction and is a major cause of unanticipated admission.[2] Uncontrolled vomiting may cause fluid and electrolyte imbalance, severe dehydration and wound dehiscence. PONV makes the child irritable and repeated PONV might even cause behavioural changes.

Causes of PONV are multifactorial and involve anaesthetic, surgical and patients risk factors. Several scoring systems have been developed for adults[3,4] but their use is limited in paediatrics population.

Various antiemetics are available but till date, no single drug has been found as an effective antiemetic agent that can inhibit the pathway of vomiting reflex and antagonize all receptor sites involved in emetic response.

Prophylaxis in children at moderate to high risk of emesis should include a combination of $5HT_3$ antagonist and a second drug This combination is effective than single drug for prophylaxis of PONV.[5]

We conducted this study to identify the risk factors associated with PONV in paediatric population undergoing common surgeries. Ondansetron and dexamethasone combination was used as prophylaxis of PONV and the anaesthetic technique used was same for all patients so as to identify risk factors investigated in this study for association with PONV were age>5 years., female gender, previous history of PONV, type of surgery and anaesthesia for more than 45 min duration.

METHOD

We conducted the study after approval by hospital ethics Committee. One hundred ASA grade I/II patients of either sex aged between 2-12 years undergoing elective surgical procedures were randomly selected.

All patients were premedicated with midazolam 0.1 mg.kg^{-1} IM/0.03 mg.kg^{-1} IV, tramadol 2mg.kg^{-1} IV, glycopyrrolate 0.01 mg.kg^{-1} IV, ondansetron 0.05mg.kg^{-1} IV and dexamethasone 0.15 mg.kg^{-1} IV.

Following preoxygenation for 3 min with 100% oxygen, patients were induced with Propofol 2.5mg.kg^{-1} IV and vecuronium 0.1 mg.kg^{-1} was given as neuromuscular blocker. Intubation was done with appropriate size endotracheal tube.

Anaesthesia was maintained with mixture of sevoflurane and oxygen. Ventillation was controlled manually using Mapleson F Circuit. Pulse rate, NIBP, SpO_2, temperature monitoring was done. At Conclusion of surgery, residual paralysis was reversed with neostigmine 0.04mg.kg^{-1} and glycopyrrolate 0.01mg.kg^{-1}.

Parameters recorded in postoperative period

1. Vomiting episodes in first 24 hours were assessed using vomiting scale:
   <2 episodes = mild vomiting
   2 vomiting = moderate vomiting
   > 2 episodes = Severe vomiting

2. Adverse effects during 24 hours i.e. headache, sedation, diarrhoea and abdominal pain were recorded.

RESULTS

All the data are expressed as mean ± SD. The vomiting incidence in various groups were compared using Chisquare test. Values were taken as significant when $p<0.05$.

The mean (SD) age, weight, duration of anaesthesia and male: Female ratio of the patients was 6.4(2.9) years, 19.4(6.6) kg, 71.5(32.3) min and 62:38 respectively (Table 1). These patients were operated for various surgical procedures (Table-2) and assessed for various risk factors for PONV (Table-3)

Table-1 Demographic profile of patient population

Variable	Mean ± SD
Age (yrs)	6.4 ± 2.9
Weight (Kg)	19.4 ± 6.6
Sex Ratio (M:F)	62:38
Average duration of anaesthesia (min)	71.5 ± 32.3

Table -2 Distribution of patients according to the type of surgery

Type of Surgery	No. of patients (%)
ENT	24 (24%)
Ophthalmological	10(10%)
Abdominal	21(21%)
Plastic	30(30%)
Orthopaedic	15(15%)

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Table-3 Distribution of patients according to various risk factors

Variable	Risk Factors	No. of Patients (%)
Age	< 5 years	37(37%)
	> 5 years	63(63%)
Sex	Male	62(62%)
	Female	38(38%)
Duration of Anaesthesia	<45 min	39(39%)
	> 45 min	61(61%)
History of Previous PONV/motion sickness	Present	20(20%)
	Absent	80(80%)

A total of 34 patients suffered from post-operative vomiting episodes in 24 hours (34%), of which 26 had only one episode and 8 patients had 2 episodes. No patients suffered from severe vomiting (>2 episodes). Only 13 % patients suffered from vomiting in first 4 hours whereas it was 29 % in late post-operative period (4-24 hr.)

As evident in Table 4, none of the predicted risk factors was found to be statistically significant for causing PONV during first 4 hours. However, in the late post-operative period, vomiting was present in 37% patients aged more than 5 years whereas it was present in 16% patients aged < 5 years. The difference in the incidence was statistically significant (P=0.03).

Whilst both gender groups were similar in the incidence of vomiting, the incidence of PONV was higher in patients with duration of anaesthesia exceeding 45 minutes. PONV was present in 38% patients when the duration of anaesthesia exceeded 45 min while it was 15% when duration of anaesthesia was short (<45min.). The difference was statistically significant. (P=0.02)

Out of one hundred patients, twenty patients had either a positive history of PONV or motion sickness (Table 3). As evident in Table 4, 25% patients (5/20) with a positive history of PONV/motion sickness had vomiting in first 4 hours whereas 10% patients (8/80) without history of PONV or motion sickness had vomiting. The difference in the two groups was not statistically significant. Also evident in Table 4, the incidence of vomiting was not associated with any particular surgical procedure.

Four patients developed abdominal cramps and two patients developed diarrhoea as an adverse reaction to antiemetic prophylaxis. None developed headache, excess sedation or erythema at injection site. (Table 5)

Table - 5 Observed adverse effects

Adverse	No. of patients (%)
1. Headache	0(0%)
2. Sedation	0(0%)
3. Diarrhoea	2(2%)
4. Abdominal pain	4(4%)
5. Local erythema	0(0%)

Discussion

Post-operative vomiting remains a major cause of patient distress, delayed hospital discharge, unanticipated hospital admission and increased use of resources leading to increased cost of care both in adult and children.[2]

Unremitting vomiting leads to dehydration, fluid & electrolyte imbalance and unwarranted side effect like pulmonary aspiration in children. Despite incidence of PONV hanging around 35-40% in children, the benefit of routine prophylactic antiemetic treatment has been questioned because antiemetics have their own side effects (sedation, lethargy & extrapyramidal). However, totally neglecting the potential of prophylactic antiemetic is by no mean acceptable but cost of prophylaxis can be contained by identifying “at high risk” patients.

The overall incidence of vomiting in our study was 34% in 24 hours period with a lower incidence of 13 % during first 4 hours but higher incidence (29%) during 4-24 hour time period. Rowley et al[1] found similar incidence of PONV in children. Lower incidence of PONV during early post-operative period signifies extended action of antiemetic prophylaxis given as premedication. Thus lower incidence of PONV during first 4 hours as compared to 4-24 hours (13% v 29%) signifies that combined premedication of ondansetron 0.05mg.kg^{-1} and dexamethasone 0.15 mg.kg^{-1} IV is highly effective in preventing PONV in paediatric patients undergoing surgery under anaesthesia.

Younger age is a risk factor that is identified in adults[(6)] however, in our study we found young children (>5years) are less susceptible to emetic stimuli. 16% (6/37) patients of age <5 years developed vomiting whereas 37% (23/63) aged >5 years had vomiting episode in 4-24 hours period. The comparison was found to be statistically significant.

Our findings are similar to those of Cohen et al.[7] Eberhart et al[(8)] also found that risk factor for developing PONV increases dramatically in patients aged more than 3 years. The difference in findings of two studies may be because of selection criteria. We studied children aged 2-12 years whereas Eberhart had studied PONV in children aged 0-14 years.

No statistical difference in the incidence of PONV in the two gender groups was found. This was comparable to findings of Rowley et al.[1] Increased emesis observed in adult female patients is probably due to hormonal changes occurring post-puberty. Most of the girls in our study were in pre-pubertal age group so the incidence was similar in both gender groups.

Our study reveals significantly different incidence of PONV in patients exposed to >45 min of anaesthesia when compared with<45 min duration. Similar observations have been made in adult PONV scoring studies. [4,9,10] In the paediatric risk study Eberhart et al[8] however found higher emetic episodes in surgeries longer than 30 min duration.

History of motion sickness/PONV has been associated with increased PONV in paediatric as well as adult patient studies.[4,7-10] Positive history of PONV/motion sickness was found to be statistically associated with increased PONV in our study.

Paediatric operations considered to be at high risk for PONV are strabismus surgery, adenotonsillectomy, hernia repair, orchidopexy, penile surgery and middle ear procedures [11-13] However, Apfel et al[9] did not find any association between type of surgery and the risk of PONV. In our study also, association of PONV with any particular surgical procedure was not found. We, however, could not study association of strabismus surgery with PONV as none of the patients underwent strabismus surgery.

Certain other risk factors like use of opioid, volatile anaesthetics and use of regional anaesthesia have been found to influence vomiting. However, the anaesthesia technique was kept common to all patients in this study. By identifying these “at risk” children and selectively premedicating them we can decrease incidence of PONV, improve parental satisfaction and reduce overall cost.

Another major finding in our study was the incidence of PONV in high risk group. It remained high in late postoperative period (4-24 hours) despite the use of combination therapy. Therefore, in high risk patients, not only combination antiemetic prophylaxis should be used but also the anaesthetic technique modified to further reduce PONV. Total intravenous anaesthesia (TIVA) with propofol is another option in these patients. Propofol infusion will not only preclude use of volatile anaesthetic but also has additional antiemetic properties. Post-operative pain is again associated with PONV. Use of regional blocks whenever possible, will not only minimize need of parenteral opioids but also provide longer post-operative pain free period.

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PROPOFOL AND THIOPENTONE SODIUM ADMIXTURE - EFFECT ON PAIN ON INJECTION, PULSE, BLOOD PRESSURE AND RECOVERY

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Bipin. M. Patel[4], Gaurav Sharma

Abstract

Aim:

This study was carried out to compare the dose requirement, induction time, haemodynemic changes, pain on injection and recovery characteristics with Thiopentone-Propofol admixtures, Propofol - Lignocaine and Thiopentone sodium alone in Day Stay Unit patients under total intravenous anaesthesia.

Methods

This study had examined two admixtures of Propofol and Thiopentone sodium. 100 patients of ASA I & II were divided in four groups. Group P_{50} Propofol 1% (5 ml) + Thiopentone sodium 2.5% (5 ml), Group P_{75}: Propofol 1 %(7.5 ml) +Thiopentone sodium 2.5 % (2.5 ml), Group P_{100}: Propofol 1% (09 ml) + Lignocaine hydrochloride 2% (1 ml), Group T: Thiopentone sodium 2.5 % (10 ml).

Results

Thiopentone sodium resulted in more rapid induction (45.64+/-7.4 seconds) of anaesthesia than any other group .The Group P_{50} (Thiopentone+ Propofol 50%) was found to be superior to Group P_{100} in reducing pain on injection. The fall in systolic blood pressure was significantly less in P_{50} (10%-20%) group compared to group P_{75} (20%-30%) and Group P_{100} (25%-40%). Recovery was early in P_{100} group (2.92+/-1.1 min).

Conclusions

Admixture of Thiopentone sodium + Propofol 50% in group P_{50} results in additive hypnotic effect, a less pain on injection and reduced hypotensive response. Group P_{50} was found more economic compared to group P_{100} alone.

Keywords: - Propofol , Thiopentone sodium, Admixture, Day Stay Unit

Introduction

Smooth induction and rapid awakening are the anesthetic prerequisites for the ambulatory surgery and both are satisfied with the induction agent like Propofol and Thiopentone sodium. Thiopentone sodium is the most widely used intravenous induction agent in practice, however a long half-life makes it less ideal agent for day stay unit. It is said, that Propofol, the newer induction agent has high lipid solubility, short half-life and inactive metabolites producing rapid onset of anesthesia, quick return of psychomotor function but has potential disadvantages like pain on injection and marked hypotension [1].

Pain on injection can be reduced by Propofol admixture with Lignocaine[2] or less conveniently by prior administration of Thiopentone sodium[3]. Hypotension with Thiopentone sodium induction is less frequently observed and when it occurs is of shorter duration[1]. In recent study some degree of synergism of hypnotic effects of Thiopentone sodium and Propofol was demonstrated. Propofol- Thiopentone admixture are physio-chemically compatible, have some degree of hypnotic synergism if they are administered sequentially[1]. Barbiturates acts by enhancement of Gamma Amino Butyric acid (GABA) receptor mediated inhibition in CNS[4]. Propofol probably acts by modulation of GABA neurotransmission although exact mechanism is unknown[4].

The aim of our study were to compare the dose requirement, induction time, haemodynemic changes, pain on injection and recovery characteristics with Thiopentone-Propofol admixtures, Propofol -Lignocaine and Thiopentone alone in DSU patients under total intravenous anaesthesia.

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METHOD

After obtaining Institutional Ethical committee approval and informed consent from patients, 100 patients of ASA I and II of Carcinoma oral cavity aged 38- 50 yrs, undergoing direct laryngoscopy and biopsy (DLB x) were included in this study. Detailed explanation about procedure, anesthesia technique and complications were given to all patients and their relatives. We excluded patients of known hypersensitivity to used drugs, aged >60 yrs, predicted difficult intubation patients and patients at risk of regurgitation.

Proper preoperative examination including examination of oral cavity to rule out difficult intubation and routine investigations (Hb, x-ray chest, urine routine, HIV and HbsAg) were done in all patients. Inj.Glycopyrolate 0.2 mg intramuscular was given half an hour before the procedure. Patients were divided in four groups.

Group P_{50} - receive mixture of Propofol 1%+ Thiopentone sodium 2.5 % in ratio of 5 ml to 5 ml.

Group P_{75}- receive mixture of Propofol 1 % +Thiopentone sodium 2.5 % in ratio of 7.5 ml to 2.5 ml.

Group P_{100} - receive mixture of Propofol 1% 09 ml + Lignocaine hydrochloride 2% 1ml

Group T -Thiopentone sodium 2.5%, 10 ml.

pH of all the induction admixtures had determined by pH meter at room temperature. A 20 gauge cannula was inserted in a vein and Ringer lactate Solution was started. The patient was asked to hold water filled syringe between thumb and middle finger of the dominant hand and Pre oxygenation was done for 6 minutes. Patient was asked to count one to hundred numbers. The drug was injected slowly over 20 seconds. The patient was asked for any forearm discomfort and, we also noted if the patients grimaced, withdrawn their hand or complained of pain[1]. When the water filled syringe dropped from patient's hand, the infusion was stopped and the volume of induction agent delivered was recorded. Induction time was assessed by noticing when the patients stopped counting. Than Injection Succinyl chloride 1mg/kg was given. Patients were ventilated till fasciculation disappeared and then surgeon was allowed to do laryngoscopy. Top up doses of drugs was given as and when required during direct laryngoscopy (DLB). The total doses of drug required and procedure time was noted.

procedural stimulation (Table 5A). But pulse rate was relatively stable in P_{50} group.

In our study recovery was faster in Group P_{100} than all other group. Recovery to discharge was also good in Group P_{50} than Group P_{75} and Group T.

Involuntary movements were presented in 5 patients in P_{100} group while it was present in 1 patient in P_{75} and group T. None of the patients experienced involuntary movements in P_{50} group. No other complications like nausea, vomiting noted in any other groups.

CONCLUSION

Addition of Thiopentone with Propofol as 50% admixture resulted in reduction in pain on injection, a less hypotensive response, rapid induction and rapid and full recovery than Propofol alone. Added to these advantage, it is also found that P_{50} admixture is economical comparable to Propofol alone.

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A RANDOMIZED, DOUBLE BLIND, PLACEBO-CONTROLLED STUDY OF PERI-OPERATIVE PREGABALIN FOR POST-OPERATIVE PAIN RELIEF IN PATIENTS UNDERGOING LOWER LIMB ORTHOPAEDIC SURGERY UNDER SPINAL ANAESTHESIA.

Pratik Tantia[1], S. Bano[2], Ruchi Saini[3]

SUMMARY

Introduction: Spinal anesthesia is the preferred technique for lower limb orthopaedic surgeries providing excellent operative conditions but with limited post-operative analgesia. Pregabalin is being used in the chronic pain arena for quite some time now but role in acute pain is not yet well defined.

Method: A randomized double-blind placebo controlled study including a total of 60 patients was designed to find the efficacy and safety of Oral Pregabalin as a protective analgesic.

Results: The time to rescue analgesic (VAS > 3) was 372.33±100.46 minutes in study group and 278.2±69.19 minutes in control group ($p < 0.0001$). The total dose of analgesics required (mg of Diclofenac) in study group (125 ± 49.57 mg) was less than the control group (162.5 ± 39.8 mg) but not statistically significant.

Conclusion: Oral Pregabalin was effective in increasing the duration of post-operative analgesia and reducing the dose of rescue analgesic required.

KEY-WORDS: Pregabalin, protective analgesia, post-operative analgesia

INTRODUCTION

"All is well that ends well". The reverse is also true as far as anesthesia and post-operative analgesia is concerned "Nothing is well if the end is not well". Acute post-operative pain (APOP), if untreated or inadequately treated can result in problems that can be a night-mare both to the patient and the attending doctor.

In an attempt to assert its significance, the Joint Committee for Accreditation of Healthcare Organization has declared "pain as the fifth vital sign"; keeping in view the benefits of aggressive management of APOP. Out of the desire to have a comfortable and pain-free patient in the post-operative period, efforts have been made and are going on throughout the world.

One such effort is the development of the concept of 'Preemptive analgesia', which aims at preventing the sensitization of the nervous system.[1, 2] This can be achieved by providing analgesia prior to the pain stimulus and continuing the same in the intra- and post-operative periods.

'Protective analgesia' is a technique that has grown as a part of preemptive analgesia wherein the drugs used are not primary analgesics but adjuvant drugs used in non-acute pain arena. Gabapentin and Pregabalin are being explored as protective analgesics.[1,2]

Pregabalin is a synthetic analogue of GABA, acting as the $\alpha_2\delta$-subunit calcium channel ligand. Clinically it has analgesic, anti-convulsant, anxiolytic and sleep-modulating activities. By reducing the hyper-excitability of dorsal horn neurons that follows surgical trauma, Pregabalin does pose as an effective protective analgesic.[3,4]

Majority of lower-limb orthopaedic surgeries are conducted under spinal anesthesia that provides profound analgesia. Untreated APOP may develop into chronic persistent neuropathic pain.

This study, therefore, was designed to evaluate the efficacy of peri-operative Pregabalin as protective analgesic in patients undergoing lower-limb orthopaedic surgeries under spinal anesthesia.

MATERIAL AND METHODS

After approval by the Institutional Ethics Committee, 60 ASA I/II adult (18-60 years) patients of either sex were enrolled in this prospective, randomized, double-blind, placebo-controlled study. They were to undergo elective lower-limb orthopaedic surgery under spinal anesthesia. Informed consent was obtained from all patients. All patients were examined in the PAC clinic/Bedside as per the Department's protocol.

Exclusion criteria for the study included patients having neurological or coagulation disorder, abnormal liver or renal function tests, valvular heart disease, hypotension, diabetes mellitus, Body mass index (BMI>30), emotional instability, unwillingness and any anticipated difficulty in regional anesthesia along with other contraindications of central neuraxial blockade.

Patients were then assigned to either of the two groups of 30 patients each with computer-generated randomization. They were to receive either study drug (Pregabalin 150 mg) or matching placebo at 1 hour before the spinal anesthesia and 7 hours after the first dose.

Anesthesia technique was standard for both the groups and started with pre-loading (500 ml of Ringer's lactate) over 15-20 minutes. Inj. Ondansetron 0.1 mg/kg was administered intravenously. Technique for spinal anesthesia included aseptic preparation and draping of the lower back. After identification of L_3-L_4 intervertebral space, lumbar puncture was done using 26 or 23 G Quincke spinal needle in sitting position. After confirmation of CSF flow, Inj. Bupivacaine 0.5% heavy, 2.5 ml was injected in subarachnoid space. Patient was then turned supine and monitors were attached (Pulse-oximeter, ECG and NIBP) and vitals recorded.

Parameters recorded

1. Pulse - Bradycardia was considered when pulse rate < 50/minute and was treated with Inj. Atropine 0.5 mg, if

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FIGURE – 2:

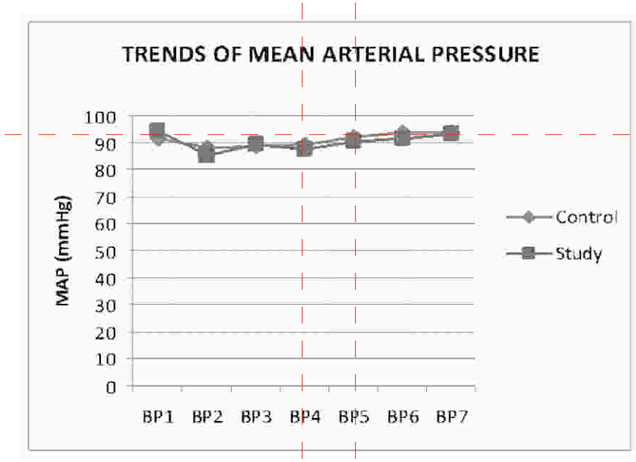
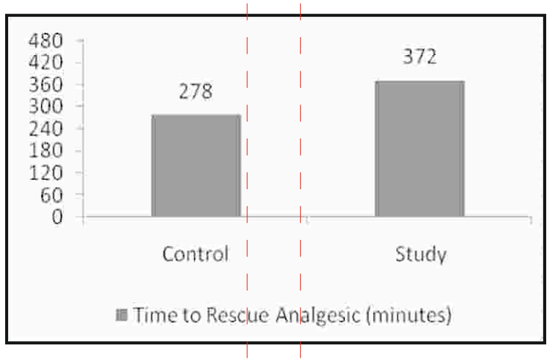


FIGURE – 3:



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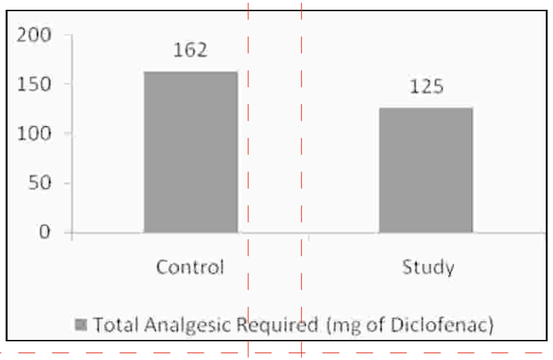

CONCLUSION

We conclude that peri-operative Pregabalin has been effective in increasing the duration of post-operative analgesia and decreasing the total dose of analgesics required in early post-operative period without any significant changes in hemodynamic parameters or side-effects.

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accompanied with hypotension.

2. BP - Hypotension was considered when systolic blood pressure showed a fall of more than 20% of the pre-operative level and was treated with Inj. Mephentermine 6 mg i.v. and crystalloid infusion.

3. SpO_2

4. Level of Sedation - This was noted as 4 point verbal rating scale

0 – No sedation
1 – Drowsiness
2 – Asleep but arousable
3 – Unarousable with loss of verbal contact

5. Total i.v. fluids and vasopressor required were noted.

6. VAS - In the post-operative period, pain was assessed using the Visual Analogue Scale at 30 minutes for the first 2 hours and then at 2 hourly intervals and next morning at 8 a.m.

7. Rescue analgesic - The time when the patient first complained of pain was noted and VAS score was assessed. VAS score more than 40 was considered significant and patient was given Rescue analgesic in the form of Inj. Diclofenac Sodium 1 mg/kg intravenous infusion.

8. Total analgesic required in the first 24 hours was noted.

9. Side Effects – Patients were asked for the following complaints: Nausea, Vomiting, Somnolence, Diplopia, Confusion and Urinary retention.

Statistical analysis was done using SPSS version 15. Since no previous data was available, sample size of 30 was taken as the minimum so as to ensure that the results were statistically significant (Central Limit Theorem). Student's t-test and Fisher's exact test were employed to find the significance of the results obtained. p-value < 0.05 was considered statistically significant. Power analysis was done post-hoc using the power and sample size calculator using the time to rescue analgesia and maximum pain scores. Cut-off limit for power of test was 80% (β= 0.8).

Aims of this study were to evaluate:

1. The duration of post-operative analgesia
2. Total dose of analgesics required in first 24 hours
3. Effect on hemodynamics
4. Side effects, if any, attributable to the drug

RESULT

Sixty patients were analyzed for the study in two groups of 30 each.

There was no statistically significant difference amongst the two groups with respect to age, sex or duration of surgery. (TABLE -1)

There was no difference amongst the two groups with respect to the site of surgery. (TABLE -2)

The hemodynamic parameters (intra and post-operatively) and the fluid and vasopressor requirement were comparable in the two groups. (FIGURES - 1 & 2)

The time to rescue analgesic (VAS > 3) was 372.33±100.46 minutes in study group and 278.2±69.19 minutes in control group (p < 0.0001). The difference was extremely significant. (FIGURE – 3)

The total dose of analgesics required (mg of Diclofenac) in study group (125 ± 49.57 mg) was less than the control group (162.5 ± 39.8 mg) but not statistically significant. (FIGURE – 4)

Of the noted complications; nausea (3 patients in control group and 1 patient in study group), vomiting (1 in control), confusion (none), diplopia (0 in control, 1 in study), and urinary retention (1 in control, 2 in study), the two groups did not differ significantly. However, the incidence of Somnolence (2 patients in control group and 10 patients in study group) was significantly higher in study group.

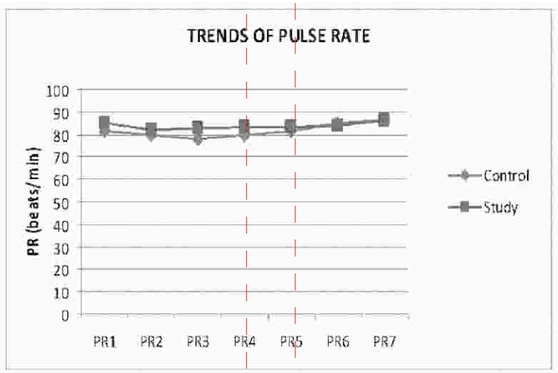
TABLE - 1: DEMOGRAPHIC DATA

Parameter	Control group	Study group	p-value
Age (years)	33.47 ± 13.66	32.03 ± 11.35	0.5145
Sex	M-22/F-8	M-20/F-10	0.7787
Duration (minutes)	78.17 ± 23.76	79.67 ± 30.51	0.4119

TABLE- 2: DISTRIBUTION OF PATIENTS ACCORDING TO SITE OF SURGERY

Site of surgery	Control group	Study group
Hip & thigh	16	17
Knee & leg	10	9
Ankle & foot	4	4

FIGURE – 1:



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Acknowledgements

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The references of the article are the foundation on which the work of the study is built. They provide the scientific background that justifies your study, including the methods used. AAAR follows 'Vancouver style' of quoting the references as superscripts in which references are numbered consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by Arabic numerals in parentheses. References cited only in tables or in legends to figure should be numbered in accordance with a sequence established by the first identification in the text of the particular table or figure. Use the style of the examples below, which are based with slight modifications on the formats used by the U S National Library of Medicine in Medline database. The titles of journals should be abbreviated according to the style used in Medline. The references must be verified by the authors(s) against the original documents. Restrict references to those that have a direct bearing on the work described, preferably less than 25 for general articles and 6 for short communications.
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6. Irrelevant and extra tables must not be included

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6. Legends and explanatory material appear in the accompanying caption and not no the figure itself.

7. Legends are typed together on one page. Legends for photomicrographs include information regarding stain and magnification.

8. Nothing is written on the back of the figures. An adhesive label, designating the top, with the first author's name and number of the figure, is attached firmly to the back of the illustration.

9. Figures are placed in a labeled envelop. No glue, paper clips or tape has been used on art.