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Study of Dexmedetomidine as an Adjuvant to Bupivacaine-Lignocaine with Adrenaline in Supraclavicular Brachial Plexusblock

Sandhya Gujar[1], Karuna Sidam[2]

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Abstract

Supraclavicular brachial plexus block is a very popular mode of anesthesia for various upper limb surgeries, as it is easiest and most effective and has good post-operative analgesia. It is carried out at the level of trunks *i.e*[7] at the middle of brachial plexus, resulting in homogenous spread of anesthetic agent throughout the plexus with a fast onset and complete block action.[9] Dexmedetomidine, is potent, highly selective α2-adrenoceptor agonist, has been used as an adjuvant during regional and local anesthesia because of rapid onset of action and relatively short half-life up to 2 hours.

Objective of Study: To compare the effects of addition of Dexmedetomidine to Bupivacaine-Lignocaine with Adrenaline combination for Supraclavicular brachial plexus block with regards to onset and duration of sensory block, motor block. Quality of anesthesia, analgesia and Adverse reactions if any after taking written informed consent.

Observations: Onset of sensory as well as motor blockade in Dexmedetomidine group was earlier when compared to plain bupivacaine-lignocaine adrenalin group. The duration of sensory and motor blockade was significantly increased ($p<0.05$) in Dexmedetomidine group when compared to another group.

With respect to hemodynamic parameters Dexmedetomidine group provided a higher Degree of cardiovascular stability with a lesser incidence of hypotension.

Result: There is earlier onset of action and longer duration of sensory; Motor block and Duration of analgesia (sensory block) was prolonged in dexmedetomidine group. Hence it is advisable to add dexmedetomidine as an adjuvant to local anesthetic combinations during supraclavicular block for prolonged anesthesia and to provide better analgesia

Keywords: 0.5% Bupivacaine; 2% lignocaine with adrenaline; Dexmedetomidine; Peripheral nerve stimulator (stimuplex DIG RC); Sensory and motor blockade.

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INTRODUCTION

Pain is "an unpleasant sensory or emotional experience associated with actual or potential tissue damage mainly during surgery and post-operative period. Regional anesthesia denotes interruption of pain impulse by physiological blockade at a certain point along with their pathway of transmission in peripheral nerves.

Regional anesthetic techniques have specific advantages both for anesthesia and surgery and it is also useful as analgesic supplements for intra-operative and post-operative period. Regional anesthesia serves an important role in facilitating ambulation by reducing immediate post-operative pain.[10] Uncontrolled pain, nausea and vomiting are common causes for delayed discharge and unanticipated hospital admission.[11,12]

Supraclavicular Brachialplexus blockade is a time tested technique for upper limb surgeries due to its effectiveness in terms of cost and performance, margin of safety and good post-operative analgesia.[1] The plexus is blocked where it is most compact[8] *i.e.* at the middle of brachial plexus, resulting in homogenous spread of anesthetic agent throughout the plexus with a fast onset and complete block action.[9]

An adjuvant is a pharmacological agent that modifies the effect of other agents. Numerous perineural adjuvants have been used with local anesthetics in regional anesthesia in an attempt to optimize block characteristics and improve clinical outcomes.[13,14] Dexmedetomidine is a selective α2-adrenoceptor agonist, Compared with clonidine, dexmedetomidine is about eight times more specific for alpha 2-adrenoreceptors with an α2:α1 selectivity ratio of 1600:1. These unique properties of dexmedetomidine make it an full agonist agent with sedative and anxiolytic effects.[11-13] The elimination half-life of dexmedetomidine is approximately 2 hours with a rapid distribution half-life being approximately 6 min. It has a rapid onset of action. It undergoes biotransformation in the liver, and the kidney excretes 95% of its metabolites.[26,27,28]

Duration and quality of motor and sensory blockade is dose dependant.[1] But increasing the doses of this hyperbaric Bupivacaine leads to increased incidences of hypotension, bradycardia and in some cases, respiratory difficulty and cardio-respiratory arrest. In cases of inadvertent intravascular injection of Bupivacaine, it was often fatal and responded poorly to conventional resuscitation methods.[21,22]

The duration of action of a local anesthetic is proportional to the time the drug is in contact with nerve fibres.[15] For this reason, epinephrine (1: 200,000 or 5 μg/mL) is added to local anesthetic solutions to produce vasoconstriction, which limits systemic absorption and maintains the drug concentration in the vicinity of the nerve fibres to be anesthetized. Thus, decreasing the possibility of systemic toxicity.[17] It is necessary to maintain doses of local anesthetic less and the same time increase duration and quality of anesthesia.

AIMS AND OBJECTIVE

To compare the effects of addition of Dexmedetomidine to Bupivacaine-Lignocaine with adrenaline combination for Supraclavicular brachial plexus block. The effects will be studied in terms of:

1. Onset of sensory and motor blockade
2. Duration of sensory and motor blockade
3. Hemodynamic effect
4. Quality of anesthesia
5. Vas score
6. Complications/side-effects if any

MATERIALS AND METHODS

Prospective, randomized, clinical study designed to evaluate and compare the effects of addition of Dexmedetomidine to Bupivacaine-Lignocaine with adrenaline combination for Supraclavicular brachial plexus block was conducted on 80 patients undergoing elective upper limb surgery.

Inclusion Criteria: All patients of ASA **grade I** and **II**, undergoing elective surgeries.

Exclusion Criteria: Patients having deformities and infection at the site of block.

Patients having bleeding disorders/coagulation abnormalities/raised intra cranial pressure.

Patients who fail to achieve desired sensory and motor blockade were excluded from the study.

The study protocol was approved by Hospital Ethics committee and Ethical Clearance was obtained from the institution for the study. Written informed consent was obtained. Pre-operative preparation and optimization of the patients were done as per protocol.

A prospective randomized comparative study was carried out in eighty patients of either sex

(18-60 yrs) allocated in one of two parallel groups containing 40 patients each.

Group I: Patients receiving 0.5% bupivacaine (15ml) + 2% lignocaine with Adrenaline (15ml) + normal saline (0.5ml).

Group II: Patients receiving 0.5% bupivacaine (15ml) +2% lignocaine with Adrenaline (15ml) + Dexmedetomidine (0.5ml).

0.5% bupivacaine, 2% lignocaine with adrenaline, dexmedetomidine, peripheral nerve stimulator (stimuplex DIG RC) were used in thestudy.

Pre-operative Preparation: Patients who are fulfilling all inclusion and exclusion criteria were explained about the study and were invited to participate in the study. All the patients underwent through pre-anesthetic evaluation on the day prior to surgery. A careful history and a through general and systemic examination were carried out including airway and the surface anatomy where the block was going to be given, and the procedure to be carried out was explained. They were informed about development of paresthesia and the motor twitch produced by nerve stimulator and vas score. Patients were reassured to alleviate their anxieties. They were investigated for routine blood and ECG, X-ray, chest. All Patients were kept nil per oral as per the fasting guidelines. All of them received Tab. Alprazolam 0.5 mg and Tab. Ranitidine 150 mg night before the surgery. Written informed consent taken. Operation theatre was prepared with routine equipment's as well as for emergency resuscitation in case of failed block or toxic reactions occurring during the procedure.

After shifting the patient to operation theatre, IV access was obtained on the forearm with 18 Gauge IV canula and IV infusion started with Ringer Lactate. Patients were monitored for heart rate (HR), non-invasive blood pressure (NIBP), percentage oxygen saturation (SpO_2). Patients were premedicated with Inj. Glycopyrrolate 0.004mg/ kg IV, Inj. Ranitidine 1 mg/kg IV, Inj. Ondansetron 0.08 mg/kg IV and Inj. Fentanyl 2 µg/kg IV.

Patient was made to lie supine with head turned opposite to side of intended block and arm adducted & pulled down gently. A small pillow or folded sheet was placed below the shoulder to make the anatomical landmarks more prominent like 1 cm above the midpoint of clavicle and pulsations of subclavian artery palpable in supraclavicular fossa.

Skin wheal is raised using 1ml of 2% lignocaine solution subcutaneously. The tip of index finger was rested in supraclavicular fossa directly over the arterial pulsation. A 22 gauge, 50mm (stimuplex, B Braun) needle attached to peripheral nerve stimulator was held in righth and and patient was instructed not to move as soon as he felt a "tingle" or "electric shock like sensation" going down his arm. The needle was inserted through skin and advanced slowly downward (caudal) rolled slightly inward (medially) and slightly backward (posteriorly). A nerve stimulator was used to locate the brachial plexus. The location endpoint was a distal motor response, *i.e.* the movement of the fingers and the thumb with an output current of 0.5 mA. During injection of the drug solution, negative aspiration done every 5 ml to avoid intravascular injection. Plexus block considered successful when at least two out of the four nerve territories (ulnar, radial, median, and musculocutaneous) effectively blocked for both sensory and motor block.

Sensory block (four nerve territories) assessed by pin prick test using a 3-point scale: 0=normal sensation, 1=loss of sensation of pin prick (analgesia), and 2=loss of sensation of touch (anesthesia).

Motor block determined by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion of elbow (Musculocutaneous nerve) according to the modified Bromage scale (18 scale) on a 3 points:

- **Grade 0:** Normal motor function with full flexion and extension of elbow, wrist, and fingers.
- **Grade 1:** Decreased motor strength with ability to move the fingers only.
- **Grade 2:** Complete motor block with inability to move the finger.

Both sensory and motor blocks assessed at 2, 5 minutes, then every 5 min for first 30 minutes and then at 45, 60, 90, and 120 min; and then hourly till the end of surgery even after the completion of surgery, until they had resolved.

Assessment of complete recovery of both sensory and motor blockade will be done for at least 12 hrs. post-operatively.

The time taken for the procedure, the onset of sensory blockade & motor blockade was noted. Intra-operatively, hemodynamic were monitored at regular intervals. Following completion of surgery, the patients were monitored to assess the quality and duration of post-operative analgesia. Thus, the patients were asked to classify analgesia as no pain, mild pain, moderate pain or severe pain every hour for the first 6 hours and then again at 8, 10 & 12 hrs. At the time of each subsequent assessment, patients were observed and/or questioned about any

subjective and/or objective side effects (sedation, nausea, vomiting or respiratory depression, neurological injury).

Defenitions of Parameters

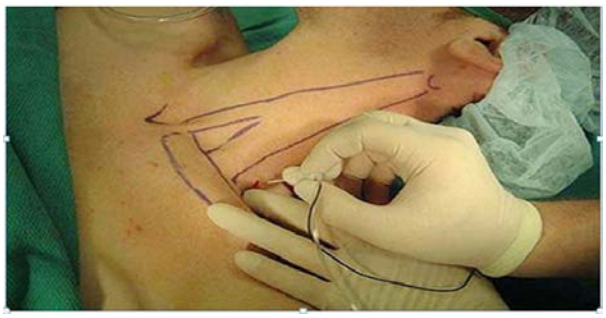

Fig. 1: Brachial plexus block with the help of nerve stimulator.

- *Onset of sensory blockade:* defined as interval between the time of injection of test drug to reduction of pain at the site of surgery or loss of sensation to cold at the site of surgery.
- *Onset of motor blockade:* defined as interval between time of injection of drug to development of motor weakness in the blocked limb.
- *Duration of analgesia:* defined as interval between onset of analgesia/sensory blockade to the time patient first complains of pain at wound site.
- *Duration of motor blockade:* defined as the interval between the onset of motor blockade to the time patient first experiences movement of the blocked limb.
- *Failure of block:* it is defined as inadequate or patchy analgesia even after 30 mins of the drug administration. Depending on the effectiveness of the block the patient was being administered sedative & analgesic in the form of IV midazolam & Injection Fentanyl. Incase of complete failure general anesthesia was administered.
- *Totally effective:* when the procedure is completed without the need of supplementation/analgesia.
- *Partially effective:* when there is need of supplementary analgesia. We administered injection fentanyl 1µg/kg.

STATISTICAL ANALYSIS

Results were statistically analyzed using association among study and control group with the chi-square and fisher exact test. Non-parametric values were analyzed using student t-test Qualitative data is presented with the help of frequency and percentage table. Quantitative data is presented with the help of Mean, Standard deviation, Median and IQR. p value less than 0.05 is taken as significant level.

OBSERVATION AND RESULTS

The prospective, randomized, comparative study was conducted on 80 patients aged between 18-60 years posted for upper limb surgeries. Patients are divided into two group to compare the effects of addition of Dexmedetomidine to Bupivacaine-Lignocaine with Adrenaline combination for Supraclavicular brachial plexus block in terms of onset & duration of sensory & motor blockade respectively, quality of block, hemodynamic changes & complications.

Group I: Patients receiving 0.5% bupivacaine (15ml) + 2% lignocaine with Adrenaline (15ml) + normal saline (0.5ml).

Group II: Patients receiving 0.5% bupivacaine (15ml) +2% lignocaine with Adrenaline (15ml) + Dexmedetomidine (0.5ml)

There were no clinical or statistically significant differences in the demographic profile of patients in either group. The average age was 36.93 ±10.413 yrs in group I, and 37.70±13.591 yrs in group II.

The average weights of the patients were 71.23±2.465 kgs in group I and 71.15 ±2.348 in group II respectively. There was no significant difference in age and weight between the two group with p > 0.05 which was statistically not significant.

Table 1: Comparison of age and weight distribution between the two groups

	Group	Group I	Group II	P Value
Age	Mean	36.93	37.70	0.775
	SD	10.413	13.591	
Weight	Mean	71.23	71.15	0.890
	SD	2.465	2.348	

Gender Distribution

Table 2: Gender distribution in two groups

-		Group I	Group II	P-Value
Sex	Male	22	26	0.361
	Female	18	14	

Both the study groups are comparable in terms of gender as shown in the table.

Onset of Motor blockade

Table 3: Onset of motor blockade in the two groups

Groups	Group I	Group II	P-Value
Mean (min)	16.23	9.98	0.000
SD	2.057	1.025	

The mean time of onset of motor blockade in **group I** was 16.23 ± 2.057 min. In **group II** it was 9.98±1.025 min.

There is significantly delayed onset of **Motor** blockade in **group I** which is statistically significant.

Table 4: Onset of sensory block in the two groups

-	Group	Group I	Group II	P Value
Onset of sensory blockade (min)	Mean	13.05	9.03	0.000
	SD	1.260	0.920	

Onset of Sensory blockade.

The mean time of onset of sensory blockade in **Group I** was 13.05 ± 1.260 mi. In **Group II** it was9.03±0.920 min. There is significantly delayed onset of sensory blockade in group I which is statistically significant.

Duration of Sensory blockade

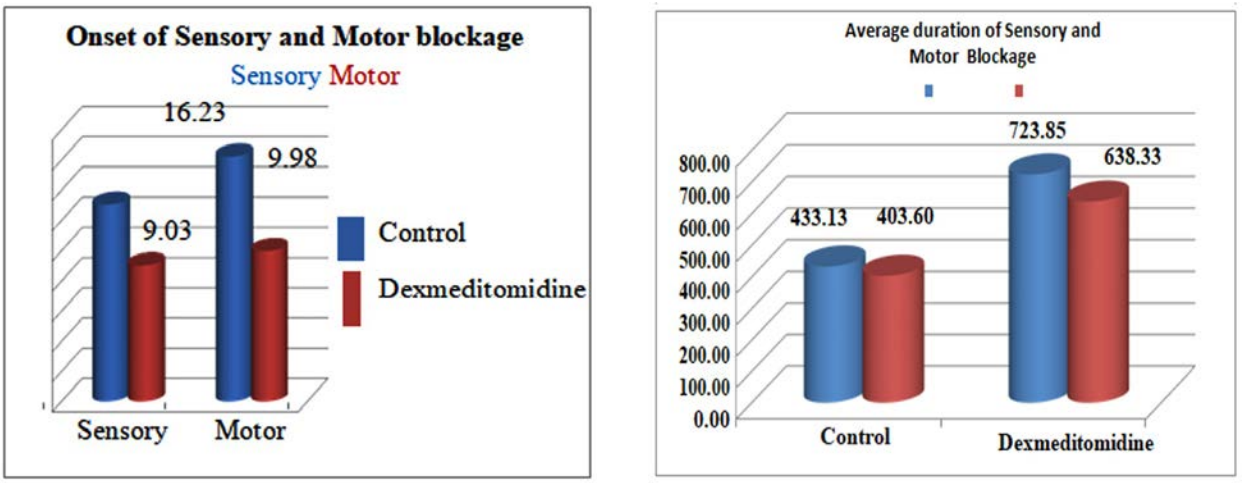

Fig. 2: BAR Diagram of Onset of sensory and motor block in Dexmedetomidine and control groups

Table 5: Duration of sensory blockade in the two groups

Group	Group I	Group II	P Value
Mean (min)	433.13	723.85	0.000
SD	16.396	11.021	

In **group, I** the mean duration of sensory blockade was 433.13 ± 16.396 min and in **group II** 723.85 ± 11.021 min. The duration of sensory blockade was shorter in **group I** when compared to **group II** and was statistically significant.

Duration of motor blockade

Table 6: Duration of motor blockade in the two groups

Group	Group I	Group II	P Value
Mean (min)	403.60	638.33	0.000
SD	15.979	13.327	

In **group I** the mean duration of motor blockade was 403.60±15.979 min where as in **group II** it was 638.33±13.327 min. The duration of motor blockade was shorter in **group I** when compared to **group II** & it was statistically significant

Haemodynamic Parameters

Table 7: Pulse Rate /Systolic BP /Diastolic BP in the two groups

Time	-	Pulse Rate	-	-	Systolic BP	-	-	Diastolic BP	-
Basal	82.28 ± 9.218	81.03± 9.088	0.543	130.73 ± 11.916	130.15 ± 11.939	0.83	84.08 ±7.043	83.08 ± 7.276	0.534
2 min	85.98 ±8.853	82.53±8.048	0.072	131.40 ±11.743	130.08 ± 10.166	0.591	84.33 ± 6.833	82.93 ± 6.203	0.34
5 min	88.38 ±8.860	80.10 ±7.675	0	131.50 ±10.715	127.30 ± 9.704	0.07	83.95 ±6.790	81.10 ±5.982	0.05
10 min	88.10 ±7.899	78.43 ±7.510	0	130.53 ± 10.175	124.15 ± 9.119	0.004	82.30 ± 6.446	78.08 ± 5.269	0.002
15 min	85.70 ±7.978	76.68 ±7.195	0	127.85 ± 10.88	121.50 ± 8.555	0.005	80.55 ± 6.950	76.28 ± 5.472	0.003
20 min	83.08 ± 7.188	74.90 ± 6.964	0	126.65 ± 10.561	119.68 ± 8.574	0.002	78.88 ±7.809	74.53 ±5.139	0.004
25 min	80.13 ±6.940	73.45 ± 6.935	0	126.03 ± 10.299	117.30 ± 8.456	0	78.53 ±7.500	72.75 ±4.824	0
30 min	77.53 ±7.016	71.65 ± 6.530	0	125.90 ± 11.043	115.13 ± 8.668	0	77.95 ± 8.025	70.98 ± 5.423	0
45 min	75.50 ± 6.965	70.18 ± 6.234	0.001	125.50 ±11.200	113.18 ± 8.348	0	78.33 ± 8.194	69.33 ± 4.96	0
60 min	74.58 ±6.621	69.18 ±6.365	0	126.35 ± 9.875	112.50 ± 7.978	0	78.73 ±7.977	68.83 ± 5.017	0
90 min	72.43 ±5.505	68.13 ±6.035	0.001	127.10± 10.553	113.28 ± 6.691	0	78.63± 7.645	69.33 ± 3.938	0
120 min	73.03 ±4.902	67.38 ±5.405	0	127.38 ±10.567	112.90 ± 6.234	0	79.85 ± 6.904	70.05± 3.063	0

Pulse Rate (Beats Per Min)/Systolic BP /Diastolic BP

Mean pulse rate decreases in both groups after 5 minutes, but decreases significantly in **group II** than **group I.** Respiratory rate in both groups are similar, there is no respiratory depression in both groups.

Mean systolic BP decreases in **group I** and **II.** After 20 minutes, there was statistically significant decrease in BP in **group II** compared to **Group I**.

Mean Diastolic BP decreases in **group I** and **II.** After 10 minutes, there was statistically significant decrease in mean diastolic BP in **group II** compared to **Group I.**

Table 8: Mean Respiratory rates in Dexmedetomedine and control groups

-	Group I	Group II	P value
Basal	12.58 ± 1.083	12.53 ± 1.086	.837
2 min	12.58 ± 1.083	12.58 ± 1.086	.837
5 min	12.60 ± 1.081	12.55 ± 1.085	.837
10 min	12.58 ± 1.130	12.53 ± 1.109	.842
15 min	12.13 ± 1.090	12.30 ± 1.114	.480
20 min	11.73 ±1.062	12.00 ±1.109	.261
25 min	11.55 ± 1.061	11.85 ± 1.075	.213
30 min	11.50 ± 1.038	11.68 ± 1.023	.450
45 min	11.45 ±1.011	11.50 ±1.038	.828
60 min	11.58 ± 1.130	11.58 ± 1.035	1.000
90 min	11.98 ±0.947	11.63 ± 1.079	.127
120 min	11.90 ±0.900	11.75 ± 1.104	.507

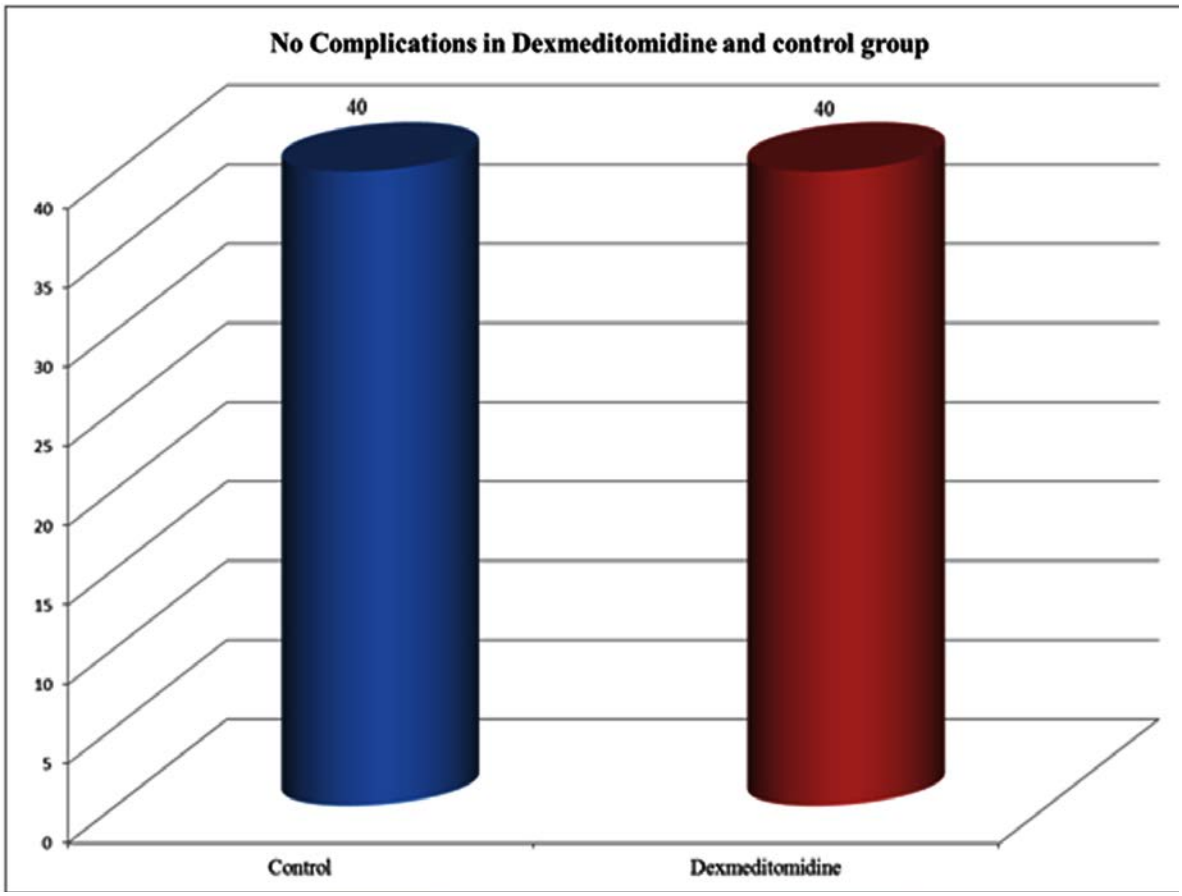

Fig. 3: Respiratory rate in both groups are similar, there is no respiratory depression in both groups

Mean Respiratory Rate (RR) in two Groups

COMPLICATIONS

Group **I** and **II** patients were observed for intra-operative and post-operative complications which include pain, nausea/vomiting, respiratory changes, SPO_2 less than 90%, hypotension, bradycardia. There is no significance difference, and no any complication in both groups.

DISCUSSION

Peripheral nerve blocks are cost effective anesthetic techniques used to provide good quality anesthesia and analgesia while avoiding airway instrumentation and hemodynamic consequences of general anesthesia. Patient satisfaction, a growing demand for cost effective anesthesia and a favourable post-operative recovery profile have resulted in increased popularity for regional techniques.

Brachia l plexus block is widely used in our practice for elective forearm and hand surgeries. It provides good intra-operative and post-operative analgesia. Various approaches like supraclavicular, interscalene, infraclavicular and axillary have been used for blocking the brachial plexus. Supraclavicular approach has rapid onset of action as compared to others.[1,2,3,8]

Evolution of Supraclavicular Brachial Plexus Block[24]

In 1911-1912, Kulenkampff described the first percutaneous supraclavicular approach.

He pointed out that above the clavicle the plexus lies under the skin as it passes over the first rib and accessible to a percutaneous technique. The midpoint of clavicle and the subclavian artery provided a constant landmark, most frequently at the point where external jugular vein intersects the clavicle.

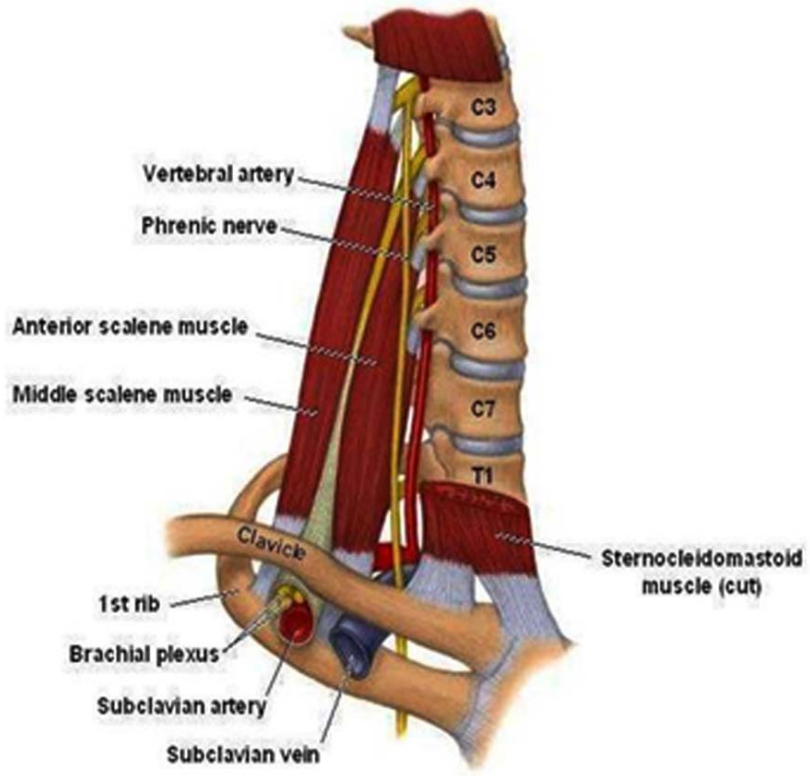

Fig. 4: Relations of brachial plexus

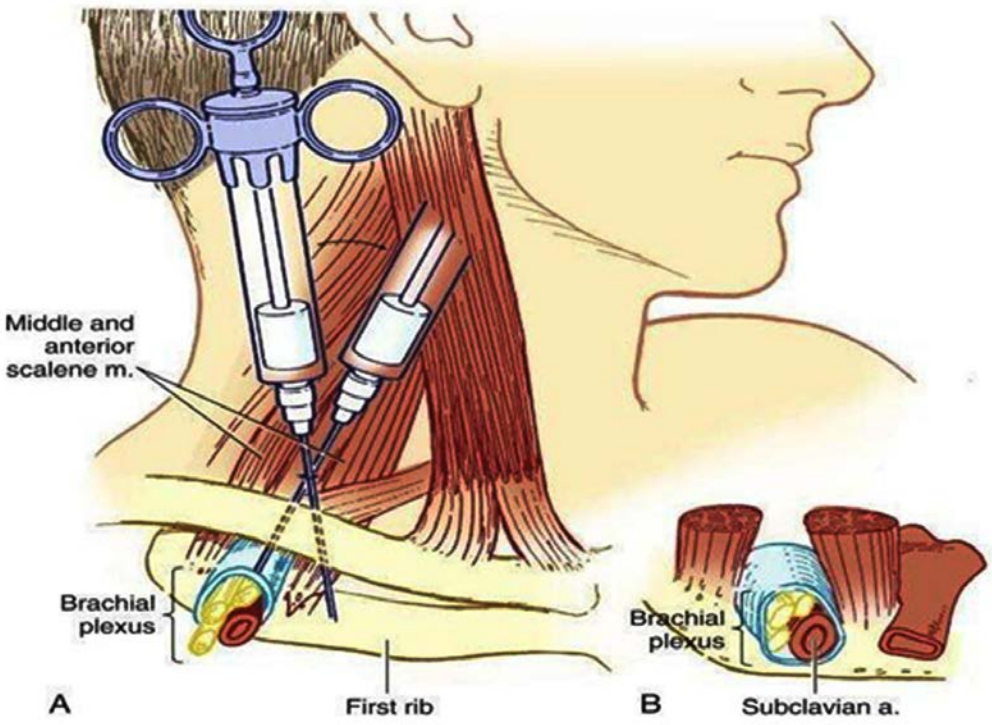

Fig. 5: Arrangement of nerve trunks in relation to landmarks used

In its passage from the cervical transverse processes to the first rib, the plexus is "sandwiched" between the anterior and middle scalene muscles and invested in the fascia of those two muscles. As the plexus cross the first rib, the three trunks are 'stacked' one on top of the other vertically This block is usually given after eliciting paresthesia and muscle twitch with peripheral nerve stimulator.[4-6] Paresthesia elicited serve two purposes.[24,25] (1) It indicates that the point of the needle is in contact with the nerve to be blocked *i.e.* an optimal position for injection of local anesthetic. (2) It also serves as a warning, indicating a risk for nerve injury.

Many substances have been added to local anesthetic agents in an attempt to prolong their duration of action. Among them addition of Dexmedetomidine is α2 receptor agonist and its α2/α1 selectivity is 8 times more than clonidine. Presynaptic activation of α2 adrenoceptor in central nervous system (CNS) inhibits the release of norepinephrine, terminating the propagation of pain signals and their post synaptic activation inhibits sympathetic activity, thereby decreasing HR and BP. High selectivity for α-2A receptors mediates analgesia, sedation, and anxiolysis.

We have investigated the effect of adding dexmedetomidine 9 to bupivacaine-lignocaine adrenalin for supraclavicular brachial plexus block, our primary end point was to find out the onset time and duration of motor and sensory blocks, hemodynamic stability and post-operative analgesia.[11-13] We conducted studies on eighty patients with demographic data in terms of age, weight and sex being similar in both the groups. The data collected was analyzed for statistical significance by student 't' test and Chi-Square test.

In our study the mean onset of sensory and motor blockade in Dexmedetomidine group was 9.03 and 9.98 minutes respectively. The mean time of onset of sensory and motor blockade in group I was 13.05 and 16.23 respectively the results of our study showed that addition of Dexmedetomidine as an adjuvant significantly enhanced the onset of both sensory and motor blockade.

The duration of sensory and motor blockade was significantly increased ($p<0.05$) in Dexmedetomidine group when compared to other group. In our study the mean duration of sensory and motor blockade in Dexmedetomidine group was 723.85 and 638.33 minutes respectively and in control group was 433.13 and 403.60 minutes respectively. It shows addition of dexem significantly enhanced duration of block. ($p<0.05$).

Effects on hemodynamic parameters including pulse rate, systolic BP (SBP), diastolic BP (DBP) monitored at 0, 2, 5, 10, 15, 20, 25, 30, 45, 60, 90 and 120 minutes. When the percentage changes in the HR, SBP, and DBP were compared from 0-5 min up to 0-120 min, they came out to be highly significant ($P <0.001$) in dexmedetomidine group. But there was no incidence of hypotension or bradycardia in both groups. There were no changes in respiration in both groups. **Group I** and **II**, patients were observed for intra-operative and post-operative complications which include pain, nausea/vomiting, respiratory changes, SPO_2 less than 90%, hypotension, bradycardia. There was no incidence of complications in both groups. Supraclavicular brachial plexus block is a very popular mode of anesthesia for various upper limb surgeries, due to its effectiveness in terms of cost and performance, margin of safety and good post-operative analgesia.[1]

Supraclavicular approach gives the most effective block for upper extremity and is carried out at the level of trunks of brachial plexus. The plexus is blocked where it is most compact *i.e.* at the middle of brachial plexus, resulting in homogenous spread of anesthetic agent throughout the plexus with a fast onset and complete block action.

A variety of adjuvants has been studied for brachial plexus blockade due to delayed onset of sensory and motor blockade, shorter duration of blockade for prolonged surgery and to increase post-operative analgesia. To overcome this drawback following were tried like, addition of enzymes, buffered and carbonated solutions, opioids, vasoconstricting agents, alkalization and warming up of local anesthetic solutions and potentiation of blockade by pain and muscular exercise. Dexmedetomidine, a selective α2-adrenoceptor agonist, has been used as an adjuvant during regional and local. Anesthetic procedures, suchas subarachnoid, epidural, and caudal injections.

CONCLUSION

Our study compared the effects of addition of Dexmedetomidine to Bupivacaine-Lignocaine with Adrenaline combination for Supraclavicular

brachial plexus block with plain Bupivacaine-Lignocaine with adrenaline from our study it was concluded that.

The onset of sensory and motor blockade was early in dexmedetomidine group when compared to control group:

- The duration sensory and motor blockade was prolonged in dexmedetomidine group when compared to another group.
- Duration of analgesia (sensory block) was prolonged in dexmedetomidine group.
- Enhancement of onset time and prolongation of duration and good post-operative analgesia, the lack of significant side effects and hemodynamic stability makes dexmedetomidine an attractive choice as an adjuvant for supraclavicular brachial plexus block.
- In this modern era of anesthesia which demands greater need of comfort, stress free anesthetic and surgical techniques, introduction of dexmedetomidine as an adjuvant to bupivacaine–lignocaine adrenaline solution might go a long way in the advancement of anesthetic procedures.

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Efficacy of Ultrasound Guided Bilateral Erector Spinae Block in Attenuating Pneumoperitoneal Stretch Response in Patients Undergoing Laparoscopic Abdominal Surg

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Abstract

Background: Increase in heart rate and blood pressure in response to pneumoperitoneum produced during laparoscopic abdominal surgeries is a challenging situation to anesthesiologists. Without adequate control of sympathetic response there is a chance of increase in morbidity of the patient during perioperative period. Aim of the study is to evaluate the efficacy of bilateral erector spinae plane block in attenuating pneumoperitoneal stretch response in patients undergoing laparoscopic abdominal surgeries under general anesthesia.

Methodology: A randomised control trial was conducted among 70 patients, who underwent laparoscopic abdominal surgeries. Under general anesthesia, 35 patients received ESP block with 20 ml of 0.25 levobupivacaine bilaterally (Group B) and 35 patients without block (Group R). This study compared requirement of opioids and response of hemodynamic parameters (Heart Rate, Mean Arterial Pressure) during pneumoperitoneal stretch. Statistical tests were applied.

Results: The intra-operative fentanyl requirement in Group B was 100.85+ and in Group R 119.375 + P value <0.001, statistically significant. The intra-operative heart rate at the time of port insertion, at 5 min, 10 min of pneumoperitoneum and at the end of surgery in Group B (91.97 ±12.09, 90.82±10.7, 92.08±10.9 and 90.857±12.5) were significant when compared to Group R (R 99.9±10.5, 94.48±13.08, 96.68±14.14 and 95.35±14.14 and same is proven statistically. ($p < 0.05$).

Conclusion: Ultrasound guided bilateral Erector spinae block with Levobupivacaine results in intra-operative hemodynamic stability during port insertion and pneumoperitoneum.

Keywords: Erector spinae; Laparoscopic Surgeries; Lneumoperitoneal.

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INTRODUCTION

Laparoscopic surgeries induce lower tissue damage than laparotomy, but still spark stress response affecting hemodynamic stability. Challenges to anesthesiologists is to lower the hemodynamic parameters in response to pneumoperitoneum during laparoscopic surgeries and also to attenuate the opioid consumption

perioperatively and to assist opioid affiliated side effects.[1] Hence regional anesthesia was often combined with general anesthesia as an alternative analgesia technique with many benefits; reduces intra-operative stress response, opioid consumption and as a means of providing post-operative analgesia[2] Analgesia provided by epidural and other peripheral nerve techniques like transverse abdominis block using local anesthetic agents was superior compared to the systemic opioids, use of these ways reduces morbidity and mortality.[3] Epidural anesthesia, a central neuraxial block was avoided compared to peripheral nerve block, to avoid unnecessary handling of central neuraxial and side effects of urinary retention.

Among peripheral nerve techniques, Erector spinae block represents a more straight forward, safer alternative to epidural anesthesia as the injection was into a tissue plane that was distant from the pleura, major blood vessels and discrete nerves.[4] Ultrasound guided erector spinae plane (ESP) block was originally reported in 2016 was used for thoracic neuropathic pain.[5] It provides thoracic analgesia when performed at the T5 level and relieves visceral abdominal pain when performed at the level of T8. Erector spinae block to be effective in providing extensive somatic and visceral abdominal analgesia in Laparoscopic abdominal surgeries by injection at lower thoracic level of T7-T9 level.[6] Local anesthetic injected into the fascial plane deep to the erector spinae muscle spreads in a craniocaudal fashion over several levels, also penetrates anteriorly through the inter transverse connective tissue and enters the thoracic para-vertebral space where it can potentially block not only the ventral and dorsal rami of spinal nerves but also the rami communicants that transmit sympathetic fibres.

Emerging research shows that erector spinae block can be employed as a simple and safe alternative analgesic technique for acute perioperative pain instead of using multimodal regimes.[7] This study aims to evaluate the efficacy of bilateral erector spinae plane block in attenuating pneumoperitoneal stretch response in patients undergoing laparoscopic abdominal surgeries under general anesthesia. The primary objective of this study was, total fentanyl consumption during pneumoperitoneum and secondary objective was hemodynamic stability during pneumoperitoneum.

METHODOLOGY

After taking institutional ethics clearance and CTRI number-CTRI/2021/12/038716, This Randomized control study design, which was a prospective, double blinded trial conducted on 70 cases undergoing laparoscopic abdominal surgeries for five months from November 2021 to March 2022. Patients were randomly divided into two groups based on random numbers generated by a computer program (www.randomizer.org).

- **Group B**: 35 patients, ESP block 0.25% levobupivacaine 20ml bilaterally.
- **Group R**: 35 patients receiving no blocks.

Allocation concealment was ensured by sequentially numbered opaque sealed envelope method. Patients who gave written informed consent, belonging to ASA I and II and patients undergoing laparoscopic abdominal surgery under general anesthesia were included in the study. Patients having hypersensitivity to the drugs used, having severe systemic illness similar asuncontrolled diabetes, hypertension and with bleeding diathesis were excluded from the study. Procedure was explained to the patients and an informed written consent was taken. Alprazolam 0.5 mg was given night before surgery. On the day of surgery patients were shifted to the operating room. Pulse oximeter, non-invasive blood pressure, ECG monitors were connected and baseline parameters recorded. An intravenous line with 18G IV cannula was obtained and IV infusion of 500 ml ringer lactate started. General anesthesia was induced with intravenous midazolam 1 mg and fentanyl 2mcg/kg as premedication, preoxygenation for 3 minutes duration with 100% oxygen at 6L/ min, inj. Propofol 2mg/kg as inducing agent. Inj. Vecuronium 0.1 mg/kg was employed as muscle relaxant. Trachea was intubated with appropriately sized cuffed endotracheal tube. Anaesthesia was maintained using isoflurane dial concentration to attain 1 MAC, in a mixture of oxygen and air. Patient was made to lie in the left side position under general anesthesia. Under strict asepsis, the T8 spinous process was located by counting down from the C7 spinous process. Ultrasound curvilinear array with low frequency 2-6 MHz, (Sonosite M Turbo) was placed transversely at this position to spot the tip of the T8 transverse process. The tip of the transverse process was centred on the ultrasound screen and also the probe was rotated into a longitudinal orientation to provide a parasagittal view, during which the following layers was visible superficial to the acoustic shadows of the transverse processes skin and subcutaneous tissue; trapezius; and erector spinae muscle. The rhomboid major muscle has its lower border at

the T5 – 6 vertebral position and its absence are going to be used as fresh evidence that the T8 transverse process is being viewed. An echogenic 23-G spinal needle (Quincke's) was inserted in-lane to the ultrasound ray in an exceedingly cranial-to-caudal direction until contact was made with the T8 transverse process. Correct position of the needle tip in the fascial plane deep to erector spinae muscle was verified by injecting 0.5-1 ml saline and seeing the fluid lifting the erector spinae muscle off the transverse process while not distending the muscle. An aggregate of 20 ml levobupivacaine 0.25% was fitted into the ESP. The procedure was repeated on the contralateral side.[4,5] Intra-operatively, intra-abdominal pressure was maintained ≤15 mm of Hg. Surgeons were asked to wait for 15 minutes to put incision (positioning of patient and aseptic precaution were followed meanwhile in that time); repeat fentanyl 0.5 to 1mcg/kg was given during carbon dioxide insufflation as rescue analgesia, if there was any increase in heart rate and mean arterial pressure by more than 20% from baseline. IV paracetamol 1gm was given to all patients in both the groups after intubation and repeated every 8th hourly. Isoflurane was stopped at the end of surgery case, was reversed with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.01 mg/kg. Case was extubated after surgery shifted to recovery. Total amount of fentanyl consumed during intraoperatively and the response of hemodynamic parameters (Heart Rate, Mean Arterial Pressure) during time of port insertion and at 5 min, 10 min, after pneumoperitoneum stretch, was recorded. Any side effects because of drugs or techniques like hematoma or vascular perforation was recorded and treated consequently. This was a double blinded study, among the two, one anesthetist involved in induction of general anesthesia along with drug loading and ESP block, another anesthetist was involved in recording the hemodynamic parameters after the block once the surgery started.

Sample size was calculated in line with the study conducted by Tulgar S *et al*[1]; grounded on the parameter fentanyl demand in group B (patients receiving ESPB) n=4 and group C (patients not receiving ESPB) n=11. The sample size calculation is $n = 2(Z_{\alpha}+Z_{1-\beta})^2\ \sigma^2/d^2$, Where Z_{α} = standard table value for 95%CI=1.96, Z1-β=standard table value for 80% power=0.84, Σ=standard deviation=3.72, D=effect size=2.5, n=35 in each group.

The statistical software namely SPSS 23 was used for the analysis of the data. Categorical data represented as frequencies. Discrete and continuous data represented as mean (SD) and used to analyse the pneumoperitoneal stretch response, fentanyl analgesia requirements intra-operatively. P value <0.05 is considered as statistical significant. Paired t test has been used to find the significance of hemodynamic parameters and opioid requirement between two groups (Inter group analysis).

RESULTS

The demographic profile about age, gender, ASA grade, and duration of surgery in the two groups were comparable. Totally 70 patients were enrolled for the study, out of which 44 were females and 28 were males with a mean age of 43.85 days duration of surgery was comparable in both the groups. There was no significant difference in status between two groups. (Table 1)

Table 1: Demographic parameters between the two groups.

Data	Group B	Group R	
Age in years	43.85±10.04	41.77±10.10	p value >0.0
Gender distribution	Male (16) Female (19)	Male (17) Female (18)	p value >0.05

The intra-operative fentanyl requirement during pneumoperitoneum in Group B was higher (100.85+2.34 mcg) compared to Group R (119.375+3.41 mcg) with P-value <0.001, statistically significant. (Table 2)

Table 2: ASA grade between the two groups

p = 0.832

		Group					
		Group B		Group R		Total	
		Count	%	Count	%	Count	%
ASA Status	1	25	55.56%	24	53.33%	49	54.44%
	2	20	44.44%	21	46.67%	41	45.56%

The hemodynamic parameters, heart rate (HR) and mean arterial pressure (MAP) was compared between the two groups. The p value on comparing baseline heart rate between the two groups was 0.128 and baseline values between two groups was comparable. The mean heart rate as beat per minute after port insertion, 5 min, 10 min of pneumoperitoneum and at the end of surgery was

stable in group B compared to group R, where the heart rate was increased on pneumoperitoneum, and the difference was statistically significant during port insertion (0.012*), at 5 min (0.043*), 10 min (0.037*) of pneumoperitoneum. (Fig. 1) The p value on comparing the baseline mean arterial pressure between the two groups was 0.342 and baseline values between two groups ware comparable. The mean arterial pressure after port insertion, 5 min, 10 min of pneumoperitoneum and at the end of surgery was stable in group B compared to group R, and the mean arterial pressure difference was statistically significant during port insertion, at 5 min, 10 min of pneumoperitoneum and at the end of surgery. ($p < 0.05$) Throughout the intra-operative pneumoperitoneum, hemodynamic parameters were stable in Group B. There was no hypotension due to sympathetic blockade or complications associated with the local anesthetic used in both the groups.

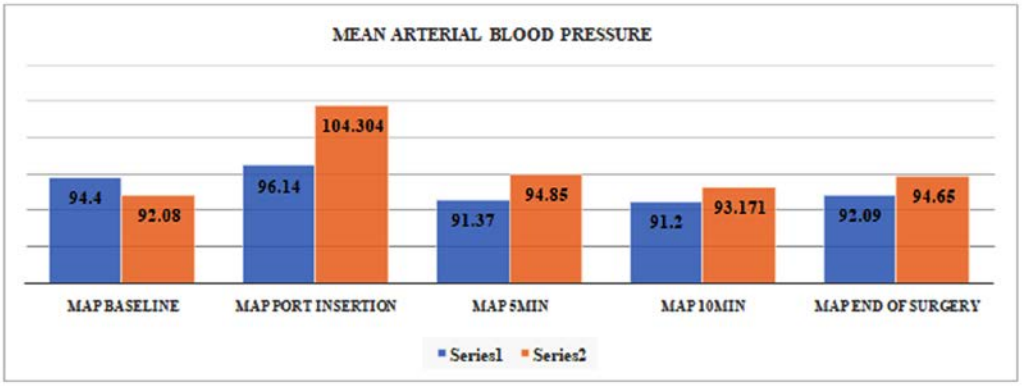

Fig. 1: Mean values of map in Group B (Series 1) and R (Series 2)
MAP = Mean arterial pressure

DISCUSSION

Erector Spinae Plane (ESP) block was first described having been used for the successful treatment of thoracic neuropathic pain. Later studies demonstrated that ESP block was an effective analgesic method in bariatric surgery, pneumothorax surgery, and major abdominal surgery when performed at the thoracic vertebral levels. The local anesthesia administered during ESP block spreads in the paravertebral space, which causes effective analgesia for somatic and visceral pain. When performed bilaterally, ESP block has similar effect as epidural analgesia.[1,2] It prevents the nociceptive signals from surgical area reaching central nervous system thus inhibits endocrine and metabolic response to surgery.[3]

Table 3: Mean value and p value of fentanyl consumption in both the groups.

	Group B	Group R	P Value
Fentanly Requrement	100.85	119.375	<0.001

Table 4: Heart rate comparison between two groups at different time intervals

bpm=beats per minute

Heart Rate at Intervals	Group B		Group R		P Value
	Mean	SD	Mean	SD	
Baseline	91.08 bpm	14.16 bpm	90.31 bpm	16.02 bpm	0.128
Port Insertion	91.97 bpm	12.09 bpm	99.9 bpm	10.5 bpm	0.012*
5 Min Pneumoperitoneum	90.82 bpm	10.7 bpm	94.48 bpm	13.08 bpm	0.043*
10 Min Pneumoperitoneum	92.08 bpm	10.9 bpm	96.68 bpm	14.14 bpm	0.037*
End of Surgery	90.85 bpm	12.5 bpm	95.35 bpm	14.14 bpm	0.043*

Laparoscopic surgery induces less tissue damage than laparotomy, the procedure will still activate stress response thus affecting hemodynamic stability. Pneumoperitoneum caused by CO_2

insufflation will increase intra-abdominal pressure (IAP) and affect hemodynamic secondary to mechanical and neurohormonal responses. Positive changes of IAP will increase MAP and systemic vascular resistance (SVR) and may decrease cardiac output (CO). Elevations in plasma norepinephrine, epinephrine, cortisol, vasopressin, renin and aldosterone levels were also demonstrated during pneumoperitoneum.[3] Pain during laparoscopic abdominal surgeries arises significantly from portsite incisions in the anterior sensory supply of the anterior abdominal wall, segmentally provided by nerves running in the fascial plane between transversus abdominis muscle and internal oblique muscle.[4] Other component of pain in upper abdominal laparoscopic procedures was the stretching of the peritoneum and peritoneal irritation caused by carbonic anhydrase response to the insufflations of parietal peritoneum with carbon dioxide.[5]

ESP block was a promising regional anesthesia technique as it had the ability to block both supra-umbilical and infra-umbilical dermatomes with a single level injection. Intra-operative fentanyl requirement during laparoscopic pneumoperitoneum would be high and it might result in side effects like vomiting, sedation, urinary retention, ileus, constipation, and respiratory depression. Patients with opioid allergy or history of serious nausea vomiting, or itching after opioid use, those who are not able to use NSAIDS, and those having low pain thresholds would appear to be good candidates for ESP block.[7,8]

In our study, ESP block the fentanyl was used to maintain intraoperative hemodynamic stability in turn to control pain, the consumption offentanyl in group B was 100.85+2.34 mcg and in group R was 119.375+3.41 mcg with p value <0.001, statistically significant. Similar results were found in Chin K J *et al*[4] and Peker *et al*[6] study, where there was reduced opioid consumption who received bilateral erector spinae block for laparoscopic surgeries. This was in concordance with the study conducted by Mohamed Ahmed Hamed *et al*,[9] total fentanyl consumption in the first 24 hrs was significantly higher in the control group compared to the ESP block group (485±20.39 mcg vs 445±67.49 mcg, respectively; P=0. 003. Heart rate the hemodynamic parameters in group B were stable. There was no variation from baseline compared to group R where there was statistical increase in heart rate, mean arterial pressure from the baseline showing stress response to pain. There was statistically significant difference during pneumoperitoneum in heart rate and mean arterial pressure (MAP) between two groups on comparing using p value, $p<0.05$. Similar findings were found in Besthadi Sukmono R[3] study, where the intraoperative mean arterial pressure in quadratus lumborum group had better stability in comparison with epidural groups, which may be caused by reduction of surgical stress responses in quadratus lumborum group.

ESP Block showed a relatively steadier intraoperative hemodynamic parameter and minimal fentanyl requirement during pneumoperitoneum, so we can use ESP block for most of the laparoscopic surgery instead of multimodal approach.[10] Limitation of our study was, we used only single shot bolus of drugs for only laparoscopic surgeries. Future scope is to evaluate the effects of unilateral catheter fixation for abdominal surgeries.

CONCLUSION

Bilateral erector spinae block was effective in attenuating pneumoperitoneal stretch response in patients undergoing laparoscopic abdominal surgeries resulting in significant decrease in opioids.

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Review Article

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Strychnine Poisoning: Literature Review

Ashish Nair

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Abstract

Strychnine poisoning is an quite unusual but a serious poisoning in which convulsions are the major threat to life. Convulsions are predominantly noted at the spinal level, and the key to recognizing this poison is observation of convulsive activity in the awake patient without a postictal phase. Successful treatment requires aggressive airway control and treatment of seizures with benzodiazepines or barbiturates. Neuromuscular blockade may be required. Gastrointestinal decontamination is usually indicated in recent acute ingestions but may precipitate convulsions. Recovery from strychnine poisoning is usually complete and rapid if treatment is aggressive.

Keywords: Strychnine; Poisoning; Convulsions.

INTRODUCTION

Strychnine was one of the most famous syntheses in the history by which two chemists won the noble prize (Robinson in 1947 and woodward in 1965) in the field of organic chemistry.[1] It was one, which has turned from medicine to poison through ges. Though it has been discovered in 1818 it has been used to kill dogs, cats and birds in Europe during 1600's.[1] During late 19th and early 20th centuries, it was popularly used as an athletic performance enhancer, recreational stimulant and believed to be cure for alcoholism addiction.[2] It has been familiar with murder mistries[3,4] (Alexander the Great, Jane Stanford cofounder of Stanford university and season 4 Game of thrones king Joffrey) and doping in olympics (1904 and 2016).

Strychnine is extracted from the plant strychnos nux vomica (genus – strychnos, family - loganiaceae) which are found in southern asia (India, Srilanka, East Indies) and Australia. At present, it is being used primarily as pesticide, particularly to kill rats. Devilishly, it was found mixed with street drugs such as LSD, Heroin and Cocaine.

Though strychnine poisonings are rare these days, ingestions (homicidal, suicidal and accidental) still occur in many places. So there is a dire need to know about varied presentations of strychnine toxicity and necessary treatment as this entity doesn't give much time between the consumption and irreversible damage to life. Hereby we would like to present a case series of poisonings and the review of literature regarding the famous rare poisonous substance: The Strychnine (Nux vomica).

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Etiology:

Strychnine poisoning can occur via ingestions, inhalational and intravenous routes. Earlier accidental ingestion were frequently seen as it has been used in many medicines (Used as a respiratory, circulatory and digestive stimulant). By the early 20th centuries, accidental ingestion has reduced a lot. Now a days, strychnine is being used in street drugs (white powder form heroin, cocaine), as pesticide (to kill rats), herbal medicines in some countries like china and combodia.[5,6,7]

MECHANISM OF ACTION

Strychnine acts by competitive inhibition of glycine receptor in the spinal cord. Uncontrollable muscle contractions occurs by negating the inhibitory effects of glycine at the postsynaptic junction predominantly in the spinal cord.[8] Due to these postsynaptic effects, patients will parade ''awake seizures'' in which patients suffer severe spasms and uncontrollable muscle contractions while maintaining clear mentation. The reason for this was relative sparing at the higher central pathways. Due to these rigorous contractions the sequelae like skeletal muscle rigidity, tachycardia, hyperthermia, respiratory failure, and potentially death through respiratory muscle paralysis occurs. Other complications which can occur include rhabdomyolysis, mixed metabolic and respiratory acidosis, hyperkalemia, and kidney failure. Strychnine is quickly absorbed from the gastrointestinal tract and symptoms can occur within 20 mins of ingestion.[9,10,11] The compound is mainly metabolized in the liver, with varying degrees of renal excretion depending on its serum levels.[9] So far reported median lethal dose of strychnine is approximately 1.5 mg/kg or 50 to 100 mg; Nevertheless, there have been case reports who survived with a serum level of 4700 ng/mL, which is >10 times the serum level seen in some case reports.[11,12,13]

The cause of death in most of the strychnine poisoning cases is due to asphyxia caused by prolonged contractions of respiratory muscles which is a sequalae of paralysis of respiratory centre in medulla.[14] The other proximate causes being cardiac arrest, multiple organ failure, or brain damage.[15]

Symptamatology and Health Effects[15, 16]

Strychnine poisoning has varied presentation from nausea to death depending on the dose/ amount of ingestion. It can take longer time to manifest at about 15 minutes by ingestion compared to inhalation or injection which can manifest within 5 min or less. Respiratory failure and brain death can occur within 15–30 mins with a very high dose and other signs and symptoms develop by ingesting lower doses.

System wise Health effects were tabulated below

S. No	System	Features
1	Central nervous system	Restlessness, apprehension, cold perspiration, heightened acuity of perception, hypervigilance, Tremor, violent repeated convulsions with opisthotonus, impairment of short and medium-term memory
2	Eyes	Exophthalmus, mydriasis, bilateral horizontal pendular nystagmus
3	Skin	Allergic response / Hypersensitivity
4	Respiratory tract	Spasmodic diaphragm movements, cyanosis, dyspnea, hypoxia, respiratory failure
5	Cardiovascular	Weak pulse, tachycardia, hypertension, cardiac arrest
6	Gastrointestinal tract	Vomiting
7	Renal and urinary tract	Myoglobinuria, acute renal failure
8	Musculoskeletal and smooth muscles	Stiffness of facial and neck muscles, hyper-reflexia Contractions of all voluntary muscles simultaneously, including chest and abdominal muscles, hypertonicity of the muscles, tonic twitching of the face and neck muscles, trismus, risus sardonicus, rhabdomyolysis
9	Metabolic	Hyperthermia
10	Laboratory findings	Lactic acidosis, hyperkalemia, elevations of AST, LDH, CPK, leukocytosis

Review of Literature

Literature on strychnine has been reviewed over a period of 100 years (1920-2020) which has been tabulated below.

Refer-ence	Year	Place	Age	No. of cases	Sex	Mode	Available form	Presentation	Drugs	Complications	Outcome
17	1928-32	Indiana, US	Apr-56	11	5 F 6 M	Suicidal (10) Accidental (1)	Tablets	Twitchings, convulsions	Sodiumamytal	Nil	Alive
18	1955	Newyork, US	15 Months	1	Male	Accidental	Laxativepills	Jerks, cyanosis convulsions	Sodium-benzoate	Respiratory failure	Expired
19	1963	Scotland, UK	18 Months	1	Male	Accidental	Tablets	convulsions	Sodiumthiopentone	Respiratory failure	Alive
20	1969	Portugal	1 year	1	Male	Accidental	Tablets	Convulsions, Opisthotonus	Notmentioned (NM)	Respiratory failure	Expired
21	1971	Portugal	13 Months	1	Male	Accidental	Syrup	Rigidity, convulsions	Diazepam	Respir atory failure	Expired
22	1971	Portugal	50 years	1	Male	Suici dal	Tablets	convulsi ons	Diazepam	Nil	Alive
23	1982	Dublin, ireland	Teens	8	NM	Accidental	Whitepowder, cocaine	Pain, stiffness, convulsions	Diazepam	Respiratory failure	7 Alive 1 Expired
24	1984	Portland	49 years	1	Male	Suicidal	Tablets	Unconsciousness, seizures	Diazepam	Respir atory failure	Expired
16	1985	Pittsburg, USA	56 years	1	Male	Suicidal	Rodenticide	Convulsions, bodyrigidity, opisthot onus	NM	Respiratory failure	Expired
25	1986	Portugal	42 yrs	1	Male	Suicidal	Notknown	Cramps, convulsions	Sedation, musclerel axation	Respiratory failure, AKI	Alive
26	1989	Newcastle, UK	32 years	1	Male	Accidental	Fumes, Fire	Convulsions, vomiting	Diazepam	Respiratory failure, AKI	Expired
16	1992	Toledo, USA	14 years	1	Female	Accidental	Syrup	Convulsions, Myalgia	Diazepam	AKI	Alive
27	1992	Malaysia	Teen	1	Male	Suicidal	Pills	Risussardonicus, opisthot onus	Notmentioned	Respiratory failure	Expired
28	1998	Spain	18 years	1	Female	Accidental	Ratbiscuits	Convulsions	Benzodiazepines	Chemicalpancr eatitis	Expired
16	2001	Manitoba, canada	50 years	1	Female	Treatment	Skinlotion	Musclespasms, cramps	Fluids, NAC	Nil	Alive
29	2002	Londoe, UK	42 years	1	Male	Suicidal	White powder	Muscle spasms	Midazolam Pancuronium	Respir atory failure	Alive
16	2003	Negev, Israel	6 years	1	Male	Accidental	Tablets	Cramps, disorientation	Diazepam, Vecuronium	Rhabdomyolysis	Alive
30	2004	Tehran, Iran	28 years	1	Male	Suicidal	Powder	Seizures	Midazolam, Sodiumthiopento ne	Respiratory failure, AKI	Alive
31	2004	Newyork, USA	46 years	1	NM	Suicidal	NM	Musclecramps, convulsions	Diazepam, midazolam, Pancuronium	Respiratory failure, AKI	Alive

32	2010	York, USA	49 years	1	Female	Suicidal	Syrup	Musclespasms	Lorazepam	AKI	Alive
33	2013	Portugal	87 years	1	Male	Suicidal	Powerform	Jerks, Convulsions	Diazepam, sodiumvalproate	Nil	Alive
34	2015	Dutch	47 years	1	Male	Suicidal	Powder	Seizures	Midazolam, Propofol	AKI	Alive
35	2016	Karnataka, India	23 years	1	Male	Suicidal	Seeds	Stiffness, Hyperreflexia	Diazepam	Nil	Alive
36	2016	Atlanta, US	40 years	1	Female	Accidental	Slangnut	Jawpain, Spasms	Barbiturates, Benzodia zepines	Nil	Alive
37	2016	Karnataka, India	36 years	1	Female	Tablets	Treebracts	Tonicclonicseizures, Teethclenching	Anti-convulsants	AKI	Alive
38	2017	Vellore, India	22-39 years	3	Males	Suicidal	Leaves	Vomitings, awake seizures	Lorazepam	Nil	Alive
39	2018	Tehran, Iran	22 years	1	Male	Suicidal	Pesticides	Serialseizures	Benzodiazepines	Respiratory failure	Alive

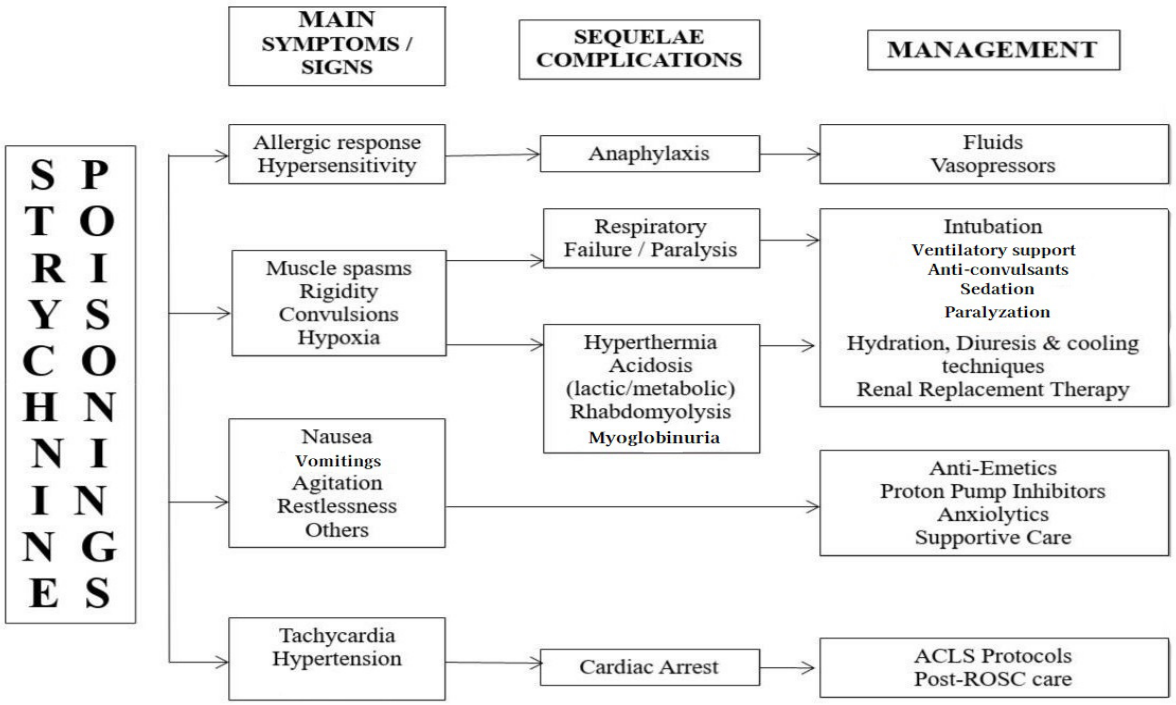
DIFFERENTIAL DIAGNOSIS

The diagnoses which need to be kept in mind includes epilepsy, tetanus, meningitis, rabies, phenothiazine overdose, cocaine and phencyclidine use and exposure to chlorinated hydrocarbons, isoniazid, cyanides, organophosphates or other substances that may cause myoclonus or seizures.[40, 41]

Treatment and its Progrssion From Decades

Observations through the above review of literature: Initially in the age olden days anticonvulsants used was sodium amytal, sodium benzoate and sodium thiopentone. Through the years it has changed to diazepam, lorazepam, midazolam and paralyzing agents like vecuronium, pancuronium etc., Early intubation was adopted now as a good outcome measure. Rapid cooling techniques for hyperthermia were also added some value for recovery of most of the cases. Improvement in renal replacement therapies has lead to avoid the AKI complications. ALL these improvements in care has shown improvement in mortality and morbidity of this toxic ingestion.

(**Antidote** for strychnine poisoning is cipher.) Strychnine poisoning is one entity which is deadliest without a specific antidote but can be treated with proper supportive care. Gastric decontamination can be done if presented early within 2 hrs though there are some evidences stating that it can lead to asphyxia, seizures. Early hospitalization with supportive care including hydration with fluids, medications for convulsions and spasms, cooling techniques and dantrolene sodium for hyperthermia, avoidance of complications like airway compromise, rhabdomyolysis, acidosis, AKI etc., are the mainstay of treatment which can save most of the poisonings of strychnine intoxication as in our case series described below. Regarding medications for convulsions barbiturates and benzodiazepines should be tried. If uncontrolled, sedation and paralysation with early intubation should not be delayed. Proper hydration can avoid rhabdomyolysis and impending AKI. Hyperthermia should be treated aggressively by ice water immersion, cooling blanket or cool mist. Symptoms, complications and management to avoid those were depicted in the below diagram.[16,42]



1: Strychnine Poisoning

Pearls to remember:

- Consider strychnine when history of illicit drug abuse is noted.
- Consider in any case with awake seizures, seizures not subsiding with barbiturates and benzodiazepines.
- Symptomatic treatment and avoiding complications with critical care during first 24–72 hrs can recover most of the cases of strychnine.

CASE SUMMARY AND DETAILS

This is a case series of four members who have consumed strychnine accidentally. Four members (One elderly male, two of them were husband and wife and one elderly lady who will be reffered from hereafter as case 1, 2, 3 and 4 respectively) have gathered at one fine night for a supper arranged at home of case 4 which has become the memorable event for their life time.

She had served the other three and had food later after 20 mins. after a while, case 1 started feeling agitated, nauseating, severe muscle spasms. Within a span of 10-15 minutes he started seizing. Case 2 and 3 were also feeling nausea and not normal. They were being taken to the hospital in the ambulance. Meanwhile case 2 started seizing in the ambulance itself. (Inj. lorazepam has been given in the ambulance for both of them). Just after reaching the hospital case 3 started seizing. We started preliminary treatment for all three cases, while one of us went to elderly lady to take history. The astounding feature about their presentation was awake seizures (conscious and aware of the things happening in the surrounding's during event). Most of the anticonvulsants were tried but seizures dint subside (we strived hard with most of the anticonvulsants but the results were lousy). To avoid complications we have no choice left other than to sedate, paralyze and intubate them. On the other side case 4 was about to finish the history and events happened. Suddenly to our surprise and giving hint, she also had seizures but got subsided with lorazepam. All of them were admitted in intensive care unit (ICU) and treated as per protocols. Treatment included mechanical ventilator (MV) support, anticonvulsants and other supportive care History and treatment part has been tabulated below.

Initial Resuscitation		
Components	**Features**	**Intervention**
A (Airway)	Frothing	Airway protection
B (Breathing)	30/min, decreased saturations, clear chest	ET Intubation, MV support, oxygen.
C (Circulation)	Tachycardia, MAP 65 to 90 mm of Hg	Nil required
D (Disability)	Not assessed	Antiepileptics, barbiturates, benzodiazepines, Sedatives, paralytics infusion.
E (Exposure and Environmental)	Hyperthermia	Cooling techniques
Sample History		
S (Signs & Symptoms)	Nausea, tachycardia, hyperthermia, agitation, restlessness, awareness of symptoms, breathing difficulty	
A (Allergy)	Nil Significant	
M (Medications)	Antihypertensives, antidiabetic medications	
P (Past History)	Nil Such events prior	
L (Last Meal)	Had food half an hour to fourty five minutes back	
E (Events)	Nil Significant	

2: Assessment

Three cases except the case 1 got recovered within a span of 3 days and got discharged within a week. All the details regarding the four cases were tabulated. Case 1 took more time as he had acute kidney injury (AKI) due to rhabdomyolysis caused by seizures.

S. No	Feature	Case 1	Case 2	Case 3	Case 4
1	Onset of symptoms	First	Second	Third	Last
2	Vitals	Stable	Stable	Stable	Stable
3	Drugs used	Benzodiazepines, Sedatives, paralytics	Benzodiazepines, Sedatives, paralytics	Benzodiazepines, Sedatives, paralytics	Benzodiazepines
4	Organ supports	MV support /Renal support	MV support	MV support	MV support
5	MV duration	7 days	3 days	3 days	0 days
6	Complications	AKI / Respiratory failure	Respiratory failure	Respiratory failure	None
7	Cause of complication	Rhabdomyolysis / more muscle mass / more food intake	Seizures	Seizures	None
8	ICU LOS	10	6	6	4

3. Case Wise Involvment

DISCUSSION

By the history at initial presentation provisional diagnosis was kept as Struchnine toxicity, Food poisoning, drug abuse mainly. Strychnine was kept by awake seizures, food poisoning as they had this feature in common before symptom onset and drug abuse as they all gathered for a party. After the recovery of the patients we were able to find the cause by taking detailed history including the examination of food materials used. The cause was found out to be due to seeds used to make dal which are surprisingly strychnine seeds (shown below in fig. 1) which were grown in their back garden. We examined the seeds and confirmed by the forensic team. Later all the events were correlated like symptom onset, complications, recovery with time of consumption of food, amount of consumption respectively.

Fig. 1:

Fig. 2:

In the present case series of four members, they have consumed accidentally Nux vomica seeds which were mix with other seeds from their garden. All the typical features were seen including nausea, awake seizures, hyperthermia, tachycardia etc., Nevertheless, there is no antidote, adequate supportive care along with avoiding complications can show good prognosis as in our case series. Death occurs mainly due to respiratory paralysis which was taken care by early intubation and MV support in our cases.

Point which has to be kept in mind and get cognizance about awake seizures so that mortality and morbidity can be reduced or avoided though strychnine poisoning due to drugs, nux vomica plant products consumption was few and far between.

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Case Report

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Topical Adrenaline Induced Ventricular Tachycardia in Craniopharyngioma Patient Posted for TNTS

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Abstract

Topical application of adrenaline is commonly used in ENT (Ear Nose Throat) surgeries and some neurosurgical procedures such as sellar or suprasellar mass excision by transnasal route. Cardiac adverse effects such as arrhythmias are not commonly reported complication of topical adrenaline application by nasal packs. Here we present a case report of topical adrenaline induced ventricular tachycardia in a Craniopharyngioma patient posted for Transnasal transsphenoidal excision of lesion (TNTS) excision of lesion.

Keywords: Adrenaline; Arrhythmias; Craniopharyngioma; Cortisol.

INTRODUCTION

Adrenaline soaked nasal packs are used routinely for most sellar and suprasellar lesion excision by transnasal route, and is a time tested procedure. Adrenaline is used with or without local anesthetic for its properties such as constricting capillaries, hemostatic agent and hence better visualization of surgical field.[1] There are reports of adverse cardiovascular effects during infiltration of adrenaline,[2] but rare encounter with the placement of Adrenaline soaked nasal packs used for nasal decongestion. Here we present a case report of induction of ventricular tachycardia in patient after placement of adrenaline soaked nasal pack for nasal decongestion for trans nasal transsphenoidal excision of Craniopharyngioma.

CASE PRESENTATION

A mid 20's man presented with history of blurring of vision since 1 month, was diagnosed as Craniopharyngioma and posted for TNTS excision of lesion. Routine pre-operative investigations were within normal limits. The endocrine workup revealed raised Serum cortisol of 28 mcg/dl and prolactin levels. Other investigations were normal. After confirming NPO, On the day the day of surgery, standard anesthesia monitors were attached and induction of anesthesia achieved with injection fentanyl 100 microgram, propofol 100 mg, vecuronium 6 mg intravenously (IV). Maintenance of anesthesia done with sevoflurane and boluses

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of fentanyl and muscle relaxants. Right subclavian vein and left radial artery cannulation was secured for fluid management and intra-arterial blood pressure monitoring respectively.

As a part of surgical preparation, 4 mg of adrenaline in 16 ml of saline soaked swabs were placed in nasal cavity for decongestion after anesthesia handover. Immediately there was tachycardia and hypertension followed by ventricular tachyarrhythmias with heart rate up to 170 bpm (beats per minute). During this period, the blood pressure went upto 290/160 mmHg. The steps taken were

- Immediately informed the surgeon to stop the stimulus and remove the nasal pack.
- Deepened the plane of anesthesia with fentanyl and propofol boluses.
- Injamiodarone 150 mg IV given by central line slowly over 10 minutes.
- Adequate fluids given to maintain the intravascular volume.

Arrhythmias settled eventually and heart rate returned to baseline after 5 minutes. There was persistent hypotension and hence blood pressure was maintained with noradrenaline infusion. There was significant ST elevation on ECG (Electrocardiogram) in lead V5 on monitor 12 lead ECG was taken and Cardiology opinion done. His 12 lead ECG showed broad QRS complexes and ST elevation in only lead V5 on monitor. Surgery was deferred in view of hemodynamic instability after consultation with the surgical team. Arterial blood gas analysis and was within normal limits. Patient was shifted to Neuro-Intensive care unit and smooth extubation was done after 2 hours. Hemodynamics were stable and later patient shifted to ward on the next day. His 2D Echo was done on the second day and follow up ECG were within normal limits.

DISCUSSION

Local application of adrenaline either infiltration or by nasal pack is commonly used to achieve hemostasis and improve the surgical field view. Although 4 mg of adrenaline with 16 ml of saline soaked nasal pack is routinely routinely by ENT Surgeons for better nasal decongestion and improved view and hence decrease the chances of bleeding during endoscopic surgeries. The studies have been done and it is advised to use adrenaline in such concentration for improved quality of surgical field and the associated hemodynamic changes could be controlled without much clinical consequences,[3] but in our case, we had ventricular tachycardia which is rare after topical adrenaline. Use of topical Moffett's solution, containing 1 ml of 1:1000 epinephrine, the peak levels of epinephrine were 2-4 times higher than the baseline epinephrine values. This increase is mainly by rapid absorption by nasal mucosa and the incidence of hypertension and tachycardia is dose dependent.[4]

It has been shown that acute low potassium levels can cause ventricular tachy arrhythmias even in the absence of cardiac disease and circulating adrenaline can induce hypokalemia in a dose dependent manner,[5] A case report of cardiovascular crisis with adrenaline infiltration and induced hypokalemia is available in literature.[6] However in our case, there was no hypokalemia as seen in arterial blood gas analysis and in post-operative period.

Inhalational anesthetics such as Isoflurane, Sevoflurane can increase the sensitivity and arrhythmogenic potential of adrenaline via synergistic effects between the adrenoceptors. It can also decrease the automaticity leading to atrial or ventricular arrhythmias.[7] In our case, we have used sevoflurane up to 0.8 MAC (Minimum Alveolar Concentration), which may be one of the contributing factor for adrenaline induced ventricular tachycardia. The role of raised serum cortisol levels and cardiovascular risk is documented.[8] Inour patient, there was only borderline elevation of serum cortisol of level 28mcg/dl which may or may not be the contributing factor.

A few factors were presumed to be contributing to the cardiovascular changes in this patient which includes:

- Increased sensitivity to adrenaline.
- Raised serum cortisol levels, though borderline elevation was present.
- Simultaneous Sevoflurane use in this patient which may increase the sensitivity of arrhythmogenic potential of adrenaline.

Considering the complexity of the individual variations of each patient and different underlying pathologies, further studies regarding the safety dose and route of administration of adrenaline for TNTS surgeries are warranted.

CONCLUSION

The sensitivity to adrenaline is different

for each individual, and may have severe cardiovascular consequences. Hence the clinician and anesthesiologist should be vigilant during the adrenaline administration even in normal doses and should be careful especially in patients with raised serum cortisol levels.

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Case Report

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Hypokalemic Periodic Paralysis: A Case Report

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Abstract

Introduction: Hypokalemic Periodic Paralysis is one form of Periodic Paralysis, a rare group of disorders that can cause sudden onset weakness. Although rare, Periodic paralysis must be differentiated from other causes of weakness and paralysis so that the proper treatment can be initiated quickly.

Case Report: A 72 year-old elderly male presented to the emergency room with sudden onset of paralysis. He had no respiratory or swallowing difficulty and was able to move his neck and facial muscles.

Neurologic exam revealed flaccid paralysis bilateral lower extremities which involved the proximal and distal muscles. Sensation was intact but deep tendon reflexes were slightly diminished to 3 out of 4 throughout. RFT deranged with serum creatinine level of 1.7 (0.6-1.2mg/dl), potassium level of 1.6 (3.5–5 mmol/L), magnesium level of 0.9 (1.3-2.1meq/l).

Electrocardiogram revealed bradycardia and left axis deviation. Two hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms started resolving.

The patient was diagnosed with Hypokalemic Periodic Paralysis and was started on calcium channel blocker for control of blood pressure. He was discharged home with an appointment to follow up.

Conclusion: Periodic Paralysis should be kept in mind when a patient comes with sudden onset weakness or paralysis, especially when other diseases have been ruled out. It can be life threatening if the treatment is improper, but intervention and subsequent correction of potassium abnormalities can clear the symptoms completely. The underlying etiology should be searched properly to avoid recurrence or persistence of the paralysis.

Keywords: Familial Hyperkalemic Paralysis; Hypokalemic Periodic Paralysis; Thyrotoxic Periodic Paralysis.

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Key Messages: The most frequent form of Periodic paralysis is hypokalemic periodic paralysis (HPP).

The abrupt onset of weakness, which can range in severity from mild, transient weakness to severe impairment leading to respiratory failure, is what makes the condition most distinctive.

Stress factors like viral fever or medications like beta-agonists, insulin, or

steroids can worsen it. Low potassium levels and muscular dysfunction are brought on by marked reduction in sodium and calcium ion channels.

In addition to potassium replacement, the heart rhythm and serum potassium are closely monitored during treatment. Prophylaxis can involve the use of spironolactone and acetazolamide.

INTRODUCTION

Hypokalemic Periodic Paralysis is one form of Periodic Paralysis, a rare group of disorders that can cause sudden onset weakness. A case of a 72 year old male is presented here. The patient presented with sudden onset paralysis of his extremities. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved upon repletion of his low potassium and he was discharged with no neurologic deficits. Further workup revealed there was Hypomagnesaemia in this patient.

CASE REPORT

A 72 year-old elderly male presented to the emergency room with sudden onset paralysis. The patient had gone to bed at 10 pm with no weakness and awoke at midnight unable to move his upper or lower extremities. The weakness was bilateral involving both the lower extremities. He was unable to walk and get up from sitting and squatting position. He had no respiratory or swallowing difficulty and was able to move his neck and facial muscles. He denied any pain or paresthesia. Prior to this episode, the patient had been healthy and denied any recent diarrheal, chest pain, shortness of breath, or weight change. He reported several episodes of dizziness and tiredness while working in his farm field. He did not take any other medications than his regular antihypertensives and denied use of alcohol or drugs, or significant changes in diet or activity levels. He is a known case of old CVA and hypertension on Tab Atenolol 50 mg and Amlodipine 5 mg. His parents and brother had no history of similar episodes and no other significant illnesses.

On physical exam, the patient's heart rate was 42 bpm and blood pressure was 110/80 mmHg, respiratory rate was 16com and Spo2 was 98% at room air. He was moderately built and nourished and normal in overall appearance His skin was cool and dry, and the oral mucosa was moist. No jugular venous distension, goiter or lymphadenopathy were appreciated. Cardiac evaluation revealed bradycardia with a regular rhythm and no murmurs. Examination of the lungs and abdomen were unremarkable. There were no deformities or enema of the extremities and distal pulses were present and equal bilaterally. Neurologic exam revealed flaccid paralysis bilateral lower extremities which involved the proximal and distal muscles. Sensation was intact but deep tendon reflexes were slightly diminished to 3 out of 4 throughout. Cranial nerve function was grossly intact. Fundoscopic examination revealed bilateral normal fundus study. 2D echo revealed grade 1 LV diastolic dysfunction, with LV EF=55%.

Routine chemistry, complete blood count were normal, RFT deranged with serum creatinine level of 1.7 (0.6-1.2mg/dl), potassium level of 1.6 (3.5–5 mmol/L), magnesium level of 0.9 (1.3-2.1meq/l)

Electrocardiogram revealed revealed bradycardia and left axis deviation.

CT brain plain study revealed a well circumscribed round soft tissue density lesion measuring ~ 2.5x2.4 cm with few specs of calcifications noted in the subcutaneous plane in high parietal region ~ likely trichelemmal cyst.

Inj. Potassium chloride 40 mEq in 500ml RL over 6 hours, Inj Magnesium 2 gm in 100 ml NS over 30 minutes was given. Two hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms started resolving. His heart rate was normal, however repeat electrocardiogram revealed a normal sinus rhythm with bradycardia. Follow up studies were performed to determine the etiology of the patient's hypokalemia. Urine sodium and potassium levels were measured to rule out adrenal involvement and were found to be normal. The patient was diagnosed with Hypokalemic Periodic Paralysis and was started on calcium channel blocker for control of blood pressure. He was discharged home with an appointment to follow up.

DISCUSSION

Weakness is the most common and general complaint both in the inpatient and outpatient

units. While there are several possible differential diagnoses for it (Table 1), the emphasis is significantly narrowed when a patient exhibits a clear decline in muscle strength on examination.[1]

Table 1: Causes of acute weakness

Neurologic
Stroke
Post-seizure Paralysis
Myasthenia Gravis
Cataplexy
Multiple Sclerosis
Inflammatory
Polymyositis
Dermatomyositis
Infectious
Polio
Diphtheria
Botulism
Metabolic
Porphyria
Alcohol/Opiates
Electrolyte disorder

Table 2: Causes of Hypokalemia

Potassium Depletion – Renal
Increased aldosterone
Diuretics
Hypomagnesemia
Renal Tubular Acidosis (Type I and II)
Metabolic alkalosis
Liddle's syndrome
Potassium Depletion – Extra renal
Decreased intake
Vomiting/Diarrhea
Zollinger-Ellison Syndrome
Fistulas
Potassium Shift into Cells
Increased insulin
Alkalosis
Thyrotoxic Periodic Paralysis
Familial Hypokalemic Paralysis

The possibility of death from nerve compression brought on by tumours and stroke should be ruled out first. Postictal paralysis or disorders of the motor neuron are some of the more frequent causes. For a diagnosis, a thorough history is required, paying close attention to the dates, durations, and different body areas affected.[1,2]

Periodic paralysis is a differential that is typically overlooked during the patient's initial work-up. Periodic paralysis comes in a variety of forms that are connected to problems in electrolytes and metabolism. The most frequent of these is hypokalemic periodic paralysis (HPP). Prevalence is 1 in 100,000 people. The underlying aetiology may affect the signs and symptoms. The abrupt onset of weakness, which can range in severity from mild, transient weakness to severe impairment leading to respiratory failure, is what makes the condition most distinctive. Stress factors like viral fever or medications like beta-agonists, insulin, or steroids can worsen it. Low potassium levels and muscular dysfunction are brought on by marked reduction in sodium and calcium ion channels.[1] Tendon reflexes may be absent, yet sensation is typically unaffected, because there is a defect with muscle contraction rather than nerve conduction. Although serum potassium levels are extremely low, total body potassium is within normal limits, and a change in serum level indicates a potassium shift inside cells.[2] Despite being present, electrocardiographic alterations do not match serum levels.[3] Because the patient may be clinically normal and the serum levels and electromyography may be within the normal range, it is challenging to diagnose it between two episodes. Since HPP can arise in a variety of circumstances, a thorough investigation is necessary to make a diagnosis. Familial Hypokalemic Paralysis (FHP), a form of HPP, can occur sporadically. It can be inherited either as autosomal dominant or spontaneously.[4] Because of defective sodium or calcium channel function, there is disrupted cellular potassium control in this form.[2,5] Defects in sodium channels caused by mutations in the CACNA1S and SCN4A genes result in aberrant potassium ion flow. In addition to potassium replacement, the heart rhythm and serum potassium are closely monitored during treatment. Prophylaxis can involve the use of spironolactone and acetazolamide.[2]

Conflict of Interest: Nil

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Case Report

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Ludwig's Angina in a Parturient: A Case Report

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Abstract

Introduction: Ludwig's angina is a rare but life threatening cellulitis of the soft tissue involving the floor of the mouth. Although Ludwig's angina is a rare condition, it must be kept as a differential in the clinical setup when a patient presents with a neck swelling.

Case Report: A 28 year old multigravida with 31 weeks 4 days gestational age presented with a swelling over the right side of the neck since 8 days. Patient also complains of toothache since past 5 days. The swelling was sudden in onset and progressed to attain the present size. It was associated with breathing difficulty since past 1 day. It was also associated with pain over the region. She had difficulty in eating solid food due the restriction in mouth opening due to the swelling. She was on liquid diet since past 7 days. She was diagnosed with hypothyroidism and was on Tab. Thyroxine 50 mcg.

On physical examination, she is a moderately built and nourished female, vitals were stable. Examination of the head and neck revealed a diffuse swelling of size 10x10 cm over the right side of face extending from the right mastoid till the right oral commissure. Superiorly it extended up to 1 cm below right eye and inferiorly till the sternocleidomastoid origin.

Total leucocyte count of 5.59 thousand/mm^3. After admission, antibiotics were started. Incision and drainage was planned for the patient. Intraoperatively, patient was tracheostomized due to restricted mouth opening and difficult intubation. After induction, vertical incision was made and about 100 ml of pus was drained from the swelling on the right side. Post-operative period was uneventful. The patient was discharged on antibiotics and tracheostomy care.

Conclusion: Ludwig's angina should be kept in mind with this type of presentation. It can be life threatening if the treatment is improper, but intervention and subsequent correction can clear the symptoms completely.

Keywords: Dental caries; Fiberoptic; Ludwig's angina; Periodontal abscess; Pregnancy; tracheostomy.

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Key Messages: In Ludwig's angina, the treatment is aimed primarily at protecting the airway the most common cause of death is asphyxiation from airway obstruction.

Airway management options include oral or nasal intubation, fiberoptic intubation or tracheostomy. Nasal intubation can be attempted by experienced personnel if the intraoral extension is minimal.

Flexible fiberoptic nasal intubation is the favoured method but arrangements for emergency awake tracheostomy must be in place before any airway intervention is attempted.

It can be life threatening if the treatment is improper, but intervention and subsequent correction can clear the symptoms completely.

INTRODUCTION

Physiologic changes in pregnancy may predispose pregnant women to oral health problems. However, most women are not counselled on oral health during pregnancy. Lack of proper oral health care predisposes pregnant women to odontogenic infections, which can lead to severe complications.

CASE REPORT

A 28 year old multigravida with 31 weeks 4 days gestational age presented to the ENT out-patient department with a history of swelling over the right side of the neck since 8 days. Patient also complains of toothache since past 5 days for which treatment was taken in a local hospital. The swelling was sudden in onset and progressed to attain the present size. It is associated with breathing difficulty since past 1 day. It is also associated with pain over the region. She doesn't give any history of fever, cough, or any other symptoms of upper and lower respiratory tract infection. There is no history of any discharge from the swelling or from the oral cavity. She had difficulty in eating solid food due the restriction in mouth opening due to the swelling. She is on liquid diet since past 7 days. Prior to this episode, the patient had been healthy and denied any recent weight loss, breathlessness or similar occurrence on the other side. She was diagnosed with hypothyroidism about 20 days prior to this episode and is on Tab. Thyroxine 50 mcg. She has a previous history of undergoing lower segment caesarean section 4 years back under spinal anesthesia which was uneventful. There is no other significant past or family history.

On physical examination, she is a moderately built and nourished female. The patient's heart rate was 84 bpm and blood pressure was 110/70 mmHg, respiratory rate was 16cpm and SpO_2 was 99% at room air. Her skin was warm and dry, and the oral mucosa was moist. No jugular venous distension, goitre or lymphadenopathy were appreciated. Cardiac evaluation revealed a regular rhythm and no murmurs. Examination of the lungs and abdomen and central nervous system were unremarkable. There were no deformities or enema of the extremities and distal pulses were present and equal bilaterally. Examination of the head and neck revealed a diffuse swelling of size 10x10 cm over the right side of face extending from the right mastoid till the right oral commissure. Superiorly it extended up to 1cm below right eye and inferiorly till the sternocleidomastoid origin.

Routine chemistry, complete blood count were normal with total leucocyte count of 5.59 thousand/mm^3. Renal function tests and Liver function tests were within normal limits.

Electrocardiogram revealed a sinus rhythm with no obvious changes in the waves.

After admission, Inj. Ceftriaxone 1gm intravenous BD was started. Incision and drainage was planned for the patient. The patient was taken up for the same on 01/07/2022. Intraoperatively, after discussion with the surgeons, patient was tracheostomized in view of restricted mouth opening, difficult intubation and the risk of rupture of the pus intraorally if intubation was attempted. After induction, vertical incision was made and about 100 ml of pus was drained from the swelling on the right side. Post-operative period was uneventful. The patient was discharged on antibiotics and tracheostomy care.

DISCUSSION

Ludwig's angina is a diffuse cellulitis of the soft tissue involving the floor of the mouth and neck. It involves three compartments of the floor of mouth, the sublingual, submandibular and submental. The infection is rapidly progressive leading to potential airway obstruction.[1]

The most common aetiology is a dental infection in the lower molars, mainly the second and third, which accounts for over 90% of the cases. Any recent infection or injury can predispose the patient to develop Ludwig's angina.[1,2]

Predisposing factors include diabetes, oral malignancy, dental caries, alcoholism, malnutrition, and immunocompromised status.

The infection begins in the subgingival pocket and

spreads to the musculature of the floor of the mouth. It then progresses below the mylohyoid line which indicates that it has moved to the sublingual space. The infection usually spreads lingually rather than buccally as the lingual aspect of the tooth socket is thinner. The disease is usually polymicrobial. The most common organisms include Staphylococcus, Streptococcus, Peptostreptococcus, Fusobacterium, Bacteroides and Actinomyces.

The treatment is aimed primarily at protecting the airway the most common cause of death is asphyxiation from airway obstruction controlling the infection with antibiotic therapy and in cases of well established infections, surgical draining.

Airway management options includes oral or nasal intubation, fiberoptic intubation or tracheostomy. Nasal intubation can be attempted by experienced personnel if the intraoral extension is minimal.

Flexible fiberoptic nasal intubation is the favoured method but arrangements for emergency awake tracheostomy must be in place before any airway intervention is attempted. In our patient, there was a risk of intraoral spread of the pus if oral or nasal intubation was attempted. There was no extension to the anterior part of the neck in this case and hence tracheostomy was chosen as the best option.[3]

Broad spectrum antibiotic intravenously are the first line of treatment. These should cover gram positive, gram negative bacteria and anaerobes. Most commonly used antibiotics are Ampicillin Sulbactam or Clindamycin. Dental extraction is recommended if the source of the infection is odontogenic. For patients who do not respond to initial antibiotics and develop a fluid collection on imaging, needle aspiration or surgical incision and drainage can be performed.[1,3]

Surgical drainage is indicated in cases of suppurative infections: purulent needle aspirate, crepitus, fluctuance and soft tissue air. The aim is to decompress the fascial compartments of the neck and evacuating the pus.[1,2]

CONCLUSION

Ludwig's angina should be kept in mind with this type of presentation. It can be life threatening if the treatment is improper, but intervention and subsequent correction can clear the symptoms completely. The underlying aetiology should be searched properly to avoid recurrence. Surgical intervention of any kind should be preplanned and proper counselling of the patient should be done.

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Case Report

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Intraoperative Anesthetic Management of Bilateral Intraventricular Hemorrhage Secondary to Anterior Communicating Artery Aneurysm Rupture in Hypertensive Patient

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Abstract

Introduction: Primary (IVH) is non traumatic intracerebral hemorrhage confined to the ventricular system which is relatively infrequent, but is more commonly caused by hypertensive (52%) hemorrhagic stroke, followed by trauma (15%), intraventricular arteriovenous malformation (7.5%), coagulopathy (7.5%), diabetic vasculopathy (7.5%), anterior communicating artery aneurysms (3%), and undetectable cause. Clipping of ruptured anterior communicating artery with hypertension possess anesthetic challenges in managing intra-operatively, and when it comes for an ideal neuroprotective agent, barbiturates induced anesthesia was planned.

Case Report: A 70 years old male came to our hospital with history of sudden loss of consciousness and a known case of systemic hypertension for 3 years and was on irregular medications, intubated in emergency medicine department in view of low GCS which was difficult intubation.

CT brain Angiogram was done and diagnosed as ruptured Anterior Communicating Artery Aneurysm and underwent Aneurysmal clipping of anterior communicating artery aneurysm under general anesthesia.

Emergency medications were kept ready. Intensive neuro monitoring, invasive blood pressure monitoring and to decrease the intraoperative intracranial pressure, to preserve autoregulation of cerebral blood flow, barbiturates induced anesthesia were planned (thiopentone as an induction agent and maintained with inhalational anesthetic agent). Patient was shifted to ICU for observation and extubated on next day uneventfully.

Conclusion: We present a successful anesthetic management of hypertensive patient with ruptured anterior communicating artery who underwent clipping of aneurysm. A detailed pre-anesthetic evaluation and proper planning is utmost important to encounter the risk of ischemic injury to brain while clipping of ruptured aneurysmal vessel.

Keywords: Barbiturates induced anesthesia; Hydrocephalus; Hypertension; Intraventricular hemorrhage.

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INTRODUCTION

Primary (IVH) is non traumatic intracerebral haemorrhage confined to the ventricular system which is relatively infrequent, but is more commonly caused by hypertensive (52%) haemorrhagic stroke, followed by trauma (15%), intraventricular arteriovenous malformation (7.5%), coagulopathy (7.5%), diabetic vasculopathy (7.5%), anterior communicating artery aneurysms (3%), and undetectable cause. Clipping of ruptured anterior communicating artery with hypertension possess anesthetic challenges in managing intra-operatively, and when it comes for an ideal neuroprotective agent, barbiturates induced anesthesia was planned.

CASE PRESENTATION

Here we present a 70 year old male who presented to Emergency Medicine Department with a history of sudden loss of consciousness following fall over the ground previous day with one episode of vomiting and no history of seizure.

Patient is a known case of systemic hypertension for 5 years and was on irregular medications (Tablet Amlodipine 5 mg twice a day) with no other comorbidities and no history of any previous surgeries. General physical examination revealed no pallor, icterus, cyanosis, clubbing, lymphadenopathy and edema with blood pressure of 190/120 mmHg, pulse rate of 80 bpm, respiratory rate of 20 cpm and saturation of 68% on room air. At arrival, patient had GCS of E1V1M1 with bilateral pupils 2 mm and reactive to light. Other systemic examination was within normal limits.

In view of low GCS, emergency intubation was done with the help of bougie due to presence of large tongue and CL grade 3 and then shifted to ICU. Patient was on Controlled Mechanical Ventilation mode with FiO_2 of 100%, Tidal volume of 450 ml RR = 12 bpm PEEP = 5 mm Hg.

Patient underwent bedside extra ventricular drainage to avoid the development of hydrocephalus following which he underwent CT Brain Angiography and was diagnosed with Bilateral Intraventricular Hemorrhage secondary to rupture of anterior communicating artery aneurysm.

Routine investigations done including complete hemogram and found to be within normal limit but coagulation profile was altered with increased prothrombin time of 20.3 secs, activated partial thromboplastin time of 33.2 secs and INR of 1.72 for which 4 FFPs were transfused. Renal function tests and liver function tests are done and within normal limits.

Electrocardiogram revealed a sinus tachycardia with tall peaked t waves (right atrial enlargement). Chest X-ray was normal. CT Brain Angiography revealed Saccular aneurysm arising from anterior communicating artery of 5x6 mm and hemorrhage was seen in lateral ventricles, 3rd and 4th ventricles.

On admission physician opinion was obtained for uncontrolled hypertension and was started on Tab. Amlodipine 5 mg 1-0-1, Tab. Telmisartan 40 mg 1-0-0, Tab. Nimodipine 60mg QID, Inj. Levetiracetam 500 mg IV 1-0-1, Inj. Mannitol 100 mg IV 1-1-1-1, Inj. Lasix 20mg IV 1-0-1.

After a thorough preoperative evaluation and written informed high-risk consent, patient underwent aneurysmal clipping for anterior communicating artery aneurysm.

Anesthetic Management:

Right femoral central venous catheterization done alongwith wide bore 18 gauge intravenous cannula. Arterial line was secured on right radial artery for invasive blood pressure monitoring. Strict neuromonitoring with electrocardiography, pulse oximetry and capnography monitoring was done.

Patient was pre-medicated with Inj. Glycopyrrolate 0.2 mg and Inj. Fentanyl 100 mcg. Induction with Inj. Thiopentone 250 mg IV over 5 minutes minutes followed by infusion dose was started at 4 mg/kg/hour it is titrated according to mean arterial pressure of the patient and facilitated with muscle relaxant Inj. Vecuronium. Anesthesia was maintained with isoflurane, oxygen, nitrous oxide and vecuronium.

Intraoperative blood pressure was 190/120 mmHg initially for which Inj. Nitroglycerin was commenced at 10 mcg/min (0.6 ml/hr) initially and titrated according to clinical response of the patient.

Blood loss was managed intraoperatively with crystalloids, 1 haemacel, 2 packed cells, 2 FFP and patient was hemodynamically stable throughout the intra-operative period and aneurysmal clipping was done successfully.

Patient was shifted with endotracheal tube in-situ to ICU for elective ventilation. Post-operative monitoring of vitals was done in ICU and ventilation was weaned down overnight. Post-

operative period was uneventful and hence patient was extubated the next day morning.

DISCUSSION

Intraventricular haemorrhage (IVH) is a recognized entity of intracranial haemorrhage.

Spontaneous also known as Primary (IVH) is non traumatic intracerebral haemorrhage confined to the ventricular system, occurs predominantly in men over 40 years of age. It is relatively infrequent, comprising only 3.1% of all spontaneous intracranial hemorrhages, but is more commonly caused by hypertensive (52%) hemorrhagic stroke, with high rates of death and disability. The appropriate treatment is not clear and the prognosis is variable with mortality rates reported as 40%-83%.

CT Angiogram or MRI Angiogram identifies the source of bleeding in about 60% of cases.

In the absence of specific treatment (*i.e.*, Extraventricular drainage (EVD), IVH is associated with a 78% risk of death and a 90% risk of poor outcome and so EVD is mandatory for the patients with IVH to reduce the risk of acute hydrocephalus.

The treatment is aimed primarily at neuroprotection and understanding the physiological implications of the surgical procedure.

Intraoperative thiopental administration significantly reduces the post-operative neurological complications in patients undergoing surgical clipping for aneurysmal rupture.

There are several proposed mechanisms for cerebral protection of thiopental.

Barbiturates (Thiopentone) induced anesthesia preserves autoregulation of cerebral blood flow by adose dependent reduction in CBF and $CMRO_2$ until the EEG becomes flat. At the point of isoelectrical EEG, no further $CMRO_2$ reduction occurs despite of further increase in barbiturate dose. The maximal thiopentone induced $CMRO_2$ decrease is 55 to 60%. Thus, with barbiturates, functional depression appears tobe coupled with reduction in CBF and $CMRO_2$. ICP is reduced by barbiturates, possibly because of reduction in CBF & CBV. This effect is used during the treatment of raised ICP in head injured patient as well as induction of anesthesia in patients with decreased intracranial compliance.

They mainly act at the post synaptic GABA receptors of the CNS synapses by keeping open the chloride channel which in turn produce conduction blockade due to the chloride ion flow producing hyperpolarization, barbiturates increase the duration of this open state of chloride channel whereas benzodiazepines increase the frequency of chloride channel opening. Secondly, patients with refractory elevated intracranial pressure (RICH) due to traumatic brain injury (TBI) may have improved long term outcome when barbiturate coma is added to their neurointensive care treatment. This phenomenon is also called an inverse steal or Robin Hood effect as cerebral perfusion to all parts of the brain is reduced (due to the decreased cerebrovascular response to carbon dioxide) allowing optimal perfusion to ischemic areas of the brain which have higher metabolic demands, since vessels supplying ischemic areas of the brain would already be maximally dilated because of the metabolic demand.[1-4]

Cardiac evaluation and liver function test was done to look for normal heart function as thiopentone is cardiotoxic which reduces the cardiac output and also it gets metabolized in liver.

The goal is to maintain ICP <25mmHg and to achieve therapeutic EEG response (burst suppression) or BIS value of 10-20 and SR of 60-80%.[5-6]

Continuous EEG monitoring, arterial blood pressure and ECG monitoring and plasma K+: Refractory hypokalaemia has been reported in patients receiving Thiopental infusions. The fall in serum K+ is thought to be due to metabolic changes within the brain. There is potential for severe rebound hyperkalaemia when the thiopental infusion is ceased, so supplement potassium cautiously, if ECG changes indicate is needed.

Nitroglycerin may reduce BP in both ischemic stroke and intracerebral hemorrhage in ultra-early, early, and subacute phase. NTG is a nitric oxide donor with other properties, including an antiplatelet effect and prevention of ischemia induced apoptosis. NTG leads to a decrease in mean arterial pressure and an increase in cerebral perfusion pressure (CPP) without any change in hemispheric CBF.

Post-operatively patient was shifted to Intensive care unit with tube in situ for elective ventilation.

CONCLUSION

In our case report we present a successful anesthetic management of a hypertensive patient who underwent aneurysmal clipping for anterior communicating artery aneurysm by intraoperative thiopentone administration which leads to

reduction in the post-operative neurological complications. A detailed pre-op planning and through understanding of hemodynamic changes is utmost important to overcome the risk of intraoperative events.

Conflicts of Interest: Nil

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The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

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