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Thoracic Epidural Analgesia versus Dexmedetomidine Infusion in Traumatic Flail Chest

Ahmed Abdelaal Ahmed Mahmoud[1*], Mohamed Adly Elramely[2], Hatem Elmoutaz[1]

[1]Faculty of Medicine, Beni-Suef University, Beni-Suef, Egypt
[2]National Cancer Institute, Cairo University, Cairo, Egypt
Email: *carnitin7@yahoo.com

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Abstract

Background: Traumatic flail chest is a serious injury that can impair ventilation and affect patient outcome. Thoracic epidural analgesia is the gold standard to provide adequate analgesia in flail chest, however, it may be unavailable in some patients due to coagulopathy, failure or difficult insertion. We compared between parenteral dexmedetomidine and thoracic epidural block with plain local anesthetic in flail chest cases. Patients and methods: fifty eight trauma patients with flail chest randomly allocated into either Group E (n = 29): epidural group, patients received mid-thoracic epidural analgesia using 6 ml mixture of 0.125% bupivacaine and 2 µg/ml fentanyl, which followed by continuous infusion of 6 ml/hour; Group D (n = 29): dexmedetomidine group, patients received loading dose of dexmedetomidine 1 µg/kg over 30 min, after a continuous infusion at a rate of 0.5 µg/kg/hr. The primary outcomes were to assess the effect of analgesic type on ventilation (PaO_2/FIO_2 ratio, $PaCO_2$). The secondary outcomes were to compare analgesic effect, hemodynamics, the need for ventilation and ICU stay. Result: PaO_2/FIO_2 ratio was significantly higher in epidural group and $PaCO_2$ was significantly lower in epidural group (p value < 0.05). The incidence of mechanical ventilation was significantly lower in epidural group than in dexmedetomidine group (6 patients group versus 13 patients, p value < 0.04). Mean arterial blood pressure was significantly lower in dexmedetomidine group than in epidural group (94.3 ± 6.84 mmHg versus 102 ± 5.72 mmHg, p value < 0.001). Moreover, heart rate was significantly lower in dexmedetomidine group than epidural group (89.97 ± 6.22 bpm versus 96.07 ± 9.3 bpm, p value = 0.004). VAS was significantly lower in epidural group (p value < 0.001). Throughout different measuring points, RAMSAY score was significantly higher in dexmedetomidine group. Conclusion: Epidural analgesia is more effective than parenteral dexmedetomidine in flail chest, but dexmedetomidine can represent a good alternative if epidural is not possible.

*Corresponding author.

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Keywords

Dexmedetomidine, Thoracicepidural, Flail Chest

1. Introduction

Rib fractures occur in up to 39% of patients with blunt chest trauma [1] and can result in a mechanically unstable chest wall or "flail chest" which is defined as three or more consecutive rib fractures, in two or more locations, creating a flail segment [2]. Flail chest can impair ventilation with subsequent prolonged mechanical ventilation, barotrauma and increased the risk for pneumonia, sepsis, long ICU stay, and death [3]-[5].

The current treatment of flail chest injuries consists of non-surgical management via intubation and intermittent positive pressure ventilation, analgesia, pulmonary toilet, and chest physiotherapy [1] [6]. According to National Trauma Data Bank (NTDB), surgical fixation of the chest wall was performed in only 0.7% and epidural catheters for patients with flail chest injuries were seldom used in only (8%). The optimal non-operative treatment of patients with flail chest includes adequate pain management, via the use of epidural catheters, intercostal nerve blocks, or patient-centered analgesia [7]. The use of epidural catheters seems to be the most preferred method, with improved outcomes and lower complications compared with other methods [8].

Conventional epidural techniques continue to be plagued by failure rate which is owed to utilize inspection and palpation only. The epidural technique relies on surface anatomical landmarks, and loss-of-resistance (LOR). Anatomical landmarks are useful but are surrogate markers, while LOR technique is also a blind technique, and the needle may be inadvertently placed intradural or intravascular or may not even in the canal [9]. Correctly identifying the location of the space not only determines the success or failure of the epidural space procedure but may also influence the onset of action and rate of absorption of the drugs [10].

The thoracic epidural space presents an additional anatomic challenge in that the spinal cord, which usually terminates between T12 and L2 lies beneath the dura mater and is closer to the ligamentum flavum laterally than it is in the midline. Accidental dural puncture at this level carries the very real risk of spinal cord injury. Keep in mind that at the midthoracic level, the interlaminar foramen through which the epidural needle must pass lies 1 to 3 cm cephalad of this interspace and the tip of the cephalad spinous process lies over the lamina of the inferior vertebra. Also, the interlaminar foramen extends laterally several millimeters wider than the spinous process [11]. Inadequate analgesia or anesthesia experienced with epidural technique may have a variety of causes including placing the epidural catheter at an inappropriate level or mal-distribution of drugs delivered into the epidural space [12].

Dexmedetomidine is a highly selective α-2 adrenoceptor agonist. It has sympatholytic, sedative, amnestic, and analgesic properties [13]. It provides a unique analgesia, without respiratory depression best described as opioid-sparing [14]. The analgesic properties of α-2 agonists are mediated by Supraspinal (locus ceruleus) and spinal (dorsal horn) mechanisms. Also it decreases sympathetic outflow through a central action in a dose-dependent manner, and these presynaptic sites of action are clinically significant because they modulate the release of norepinephrine. This inhibitory effect on neurotransmitter release is mediated by the blockage of calcium entry into nerve terminals [15] [16].

Systemic administration of the α-2 agonists has been tested in the perioperative period. The reduced opioid requirement was a feature of the administration of such group of medications [17]. This feature is indirect evidence that these drugs have analgesic action. However, the perioperative period involves many conflicting factors. Clearly, the sedative effect may be the reason behind the reduced opioid requirements [18].

2. Aim of Study

This work aims at comparing midthoracic epidural analgesia with continuous dexmedetomidine infusion in trauma patients with flail chest.

3. Methods

This monocentric un-blinded randomized controlled study was carried out in the surgical ICU at Beni-Suef University hospital, faculty of medicine, Beni-Suef University, a tertiary hospital with an average yearly admis-

sions of trauma cases is 780 cases per year. The study was carried out between June 2013 and June 2015, after obtaining approval of the departmental scientific and ethical committee.

Ttrauma patients admitted to our surgical ICU over a period of two years (June 2013 and June 2015) were assessed for eligibility for inclusion in the study, fifty eight patients of both sexes, 18 - 60 years old, with isolated chest trauma, trauma survey only detected flail chest without haemothorax or pneumothorax were included in the study after obtaining a written informed consent. The exclusion criteria were body mass index more than 30 kg/m^2, the need for mechanical ventilation on admission, hemodynamic instability, haemothorax or pneumothorax, long-term use of B-blockers, psychiatric illness and the contraindications for performing epidural insertion as Coagulopathy, Vertebral column deformities, local infection, and trauma survey include abdominal or head injury. Patients were randomly allocated into two groups.

Group E: Epidural group, 29 patients, received mid-thoracic epidural analgesia with a loading dose of 6 ml mixture of 0.125% bupivacaine and 2 μg/ml fentanyl, which was followed by continuous infusion of 6 ml/hour.

Group D: Dexmedetomidine group, 29 patients, receivedinitial loading dose of dexmedetomidine of 1 μg/kg over 30 min followed by a continuous infusion at a rate of 0.5 μg/kg/hr.

The Primary outcome of this study was assessment of parameters of adequate ventilation and oxygenation; PaO_2/FIO_2 ratio and $PaCO_2$. The secondary outcomes were the need for mechanical ventilation, Visual Analogue Scale (VAS) score, changes in heart rate (HR) and changes in arterial blood pressure (ABP).

All patients were subject to systematic assessment including history taking, physical examination, and review of the results of routine investigations. Visual analogue pain score (VAS) was explained to all candidates where zero corresponds to no pain and 10 is indicative of the worst unbearable pain.

In the Group E, patients were preloaded with 500 ml Ringer's solution. The procedure was done in sitting position. The interspinous space was identified by utilizing anatomic landmarks and palpation. Paramedian access plane is for needle insertion with loss of resistance technique [19]. Local anesthetic was injected into the skin and interspinous ligament after sterilization. The epidural space was located using a manual "loss-of-resistance" technique. A Tuohy needle was inserted into the interspinous ligament and advanced very slowly until there was no longer any resistance felt, indicating that the tip of the needle was in the epidural space. A catheter was threaded through the needle upwards for 3 cm more than the distance between skin and epidural space and remains in the epidural space during needle removal [20]. After that, epidural analgesia was activated using 6 ml 0.125% bupivacaine and 2 μg/ml fentanyl, which followed by continuous infusion of 6 ml/hour for 48 hours.

In Group D, patients received an initial loading dose of dexmedetomidine one μg/kg over 30 min. followed by a continuous infusion at a rate of 0.5 μg/kg/hr. for 48 hours.

All patients in both group received paracetamol (Perfalgan®) 1 gm IV drip every 8 hours, diclofenac sodium (Voltaren®) 75 mg IM every 12 hours and pethidine 0.5 mg/kg IV if needed.

In both groups these parameters were recorded for 48 hours:

- Heart rate, and mean arterial pressure (MAP) were recorded at 10, 20, 30, 40, 50, 60, 90 and 120 min. then at 6, 12, 16, 18, 24, 36, and 48 hr;
- PaO_2/FIO_2 ratio and $PaCO_2$ were recorded at 1, 2, 3, 4, 8, 16, 24, 36, 48 hours;
- Visual Analog Scale (VAS) were recorded at 1 hour and every 6 hours;
- Sedation scores every 8 hours, Ramsay score was used to evaluate sedation. Score 1 represents agitated and uncomfortable patient. Score 2 represents cooperative and orientated patient. Score 3 represents a patient who can follow simple directions. Score 4 represents an asleep patient with a strong response to stimulation. Score 5 represents an asleep patient with a slow response to stimulation. Score 6 represents an asleep patient with no response to stimulation;
- The incidence of mechanical ventilation, nausea, vomiting, urine retention, failure of epidural technique, dural puncture, intravascular catheterization or subarachnoid catheterization.

3.1. Statistical Analysis

The statistical analysis was performed using a standard SPSS software package version 17 (Chicago, IL). Data were expressed as mean values ± SD, ratio and median (IQR). Student's *t*-test was used to analyze the parametric data, Mann Whitney test for non-parametric data and categorical variables were analyzed using the χ^2 test, with p values < 0.05 considered statistically significant.

3.2. Sample Size Calculation

Using PASS for sample size calculation, it was calculated that a sample size of 29 per group will achieve 85% power to detect a difference between the two groups with a significance level (alpha) of 0.05 using a two-sided two-sample t-test.

3.3. Results

Both groups consist of 29 patients. Mean age in epidural group was 34.81 ± 10.867 years and in dexmedetomidine group was 35.9 ± 10.8 years. Epidural group has 22 males and 7 females while dexmedetomidine group has 20 males and 9 females.

PaO_2/FIO_2 ratio was significantly higher in epidural group (Table 1) and $PaCO_2$ was significantly lower in epidural group (Table 2). Mean arterial blood pressure (Table 3) was significantly lower in dexmedetomidine group after 20 minutes (94.3 ± 6.84 mmHg versus 102 ± 5.72 mmHg) (p value < 0.001). Moreover, Heart rate (Table 4) was significantly lower in dexmedetomidine group after 20 minutes (89.97 ± 6.22 bpm versus 96.07 ± 9.3 bpm) (*P* value = 0.004) and (81.77 ± 5.96 bpm versus 91.23 ± 7.79 bpm) (p value < 0.001) after 30 minutes. Clearly, Incidence of mechanical ventilation was significantly lower in epidural group than in dexmedetomidine group (13 patients versus 6 patients, p value < 0.04) and VAS (Figure 1) was also significantly lower in epidural group after 1 hour [1.5 (1 - 3) versus 5 (4.75 - 6)] (p value < 0.001). Throughout different measuring points,

Table 1. PO_2/FiO_2 in epidural and dexmedetomidine groups.

	Group E (n = 29)	Group D (n = 29)	p-value
Baseline	171.9 ± 51.3	172.76 ± 62.99	0.954
1 h.	188.47 ± 41.28	162.38 ± 61.29	0.04*
2 h.	181.13 ± 38.68	155.1 ± 59.57	0.032*
3 h.	179.97 ± 41.02	148.07 ± 60.09	0.023*
4 h.	185.67 ± 37.4	140.28 ± 63.35	0.001*
8 h.	178.6 ± 56.1	139.37 ± 63.62	0.017*
16 h.	181.7 ± 62.07	136.6 ± 63.79	0.014*
24 h.	175.5 ± 65.9	140.5 ± 64.28	0.025*
36 h.	177.37 ± 66.77	138.97 ± 73.46	0.02*
48 h.	174.07 ± 72.2	134.86 ± 52.48	0.03*

Data are presented as mean ± SD. *: p value < 0.05 is considered statistically significant. Po_2: arterial oxygen tension measured in mmHg. FiO_2: fraction of inspired oxygen. Group E: Epidural group. Group D: Dexmedetomidine group. h.: hour.

Table 2. PCO_2 in epidural and dexmedetomidine groups.

	Group E (n = 29)	Group D (n = 29)	p-value
baseline	42.37 ± 5.94	44.87 ± 5.26	0.53
1 h.	40.57 ± 3.4	44.13 ± 4.81	0.002*
2 h.	41.13 ± 4.24	43.9 ± 5.15	0.027*
3 h.	40.33 ± 4.66	43.97 ± 5.78	0.01*
4 h.	40.13 ± 3.75	44.3 ± 7.09	0.006*
8 h.	39.47 ± 3.42	44.45 ± 7.089	0.001*
16 h.	40.83 ± 4.23	45.18 ± 7.88	0.012*
24 h.	41.17 ± 4.89	44.8 ± 7.55	0.036*
36 h.	48.3 ± 2.73	42.3 ± 7.36	0.003*
48 h.	48.07 ± 3.49	41.32 ± 5.86	0.019*

Data are presented as mean ± SD. *: p value < 0.05 is considered statistically significant. PCO_2: arterial carbon dioxide tension measured in mmHg. Group E: Epidural group. Group D: Dexmedetomidine group. h.: hour.

Table 3. Mean arterial blood pressure in epidural and dexmedetomidine groups.

	Group E (n = 29)	Group D (n = 29)	p-value
Baseline	106.57 ± 5.33	109.13 ± 5.72	0.0777
10 min.	104.2 ± 5.8	100.8 ± 4.9	0.779
20 min.	102 ± 5.72	94.3 ± 6.84	<0.001*
30 min.	100.57 ± 5.43	87.07 ± 6.4	<0.001*
40 min.	100.07 ± 5.5	79.9 ± 7.94	<0.001*
60 min.	99.43 ± 4.6	81.6 ± 7.14	<0.001*
90 min.	98.4 ± 5.2	81.3 ± 6.75	<0.001*
120 min.	98.07 ± 5.68	81.53 ± 7.7	<0.001*
6 h.	98.24 ± 5.58	82.5 ± 9.09	<0.001*
12 h.	100.3 ± 6.28	83.34 ± 10.51	<0.001*
18 h.	99.8 ± 7.03	85.42 ± 11.35	<0.001*
24 h.	94.83 ± 10	87.03 ± 11.25	<0.001*
36 h.	99.36 ± 6.87	86.5 ± 11.56	<0.001*
48 h.	98.37 ± 7.5	85.82 ± 10.55	<0.001*

Data are presented as mean ± SD. *: p value < 0.05 is considered statistically significant. Group E: Epidural group. Group D: Dexmedetomidine group. Min.: minute. h.: hour.

Table 4. Heart rate in epidural and dexmedetomidine groups.

	Group E (n = 29)	Group D (n = 29)	p-value
Baseline	101.4 ± 7.9	103.8 ± 5.8	0.234
10 min.	98.7 ± 9.7	96.27 ± 7.56	0.266
20 min.	96.07 ± 9.3	89.97 ± 6.22	0.004*
30 min.	91.23 ± 7.79	81.77 ± 5.96	<0.001*
40 min.	87.07 ± 7.89	74.8 ± 7.82	<0.001*
60 min.	86.1 ± 7.98	74.97 ± 7.1	<0.001*
90 min.	85.87 ± 8.49	74.37 ± 7.08	<0.001*
120 min.	84.7 ± 9.55	73.3 ± 6.9	<0.001*
6 h.	84.37 ± 10.4	72.79 ± 6.97	<0.001*
12 h.	82.37 ± 7.48	72.2 ± 7.85	<0.001*
18 h.	82.5 ± 7.69	74.24 ± 7.06	<0.001*
24 h.	82 ± 7.94	73.8 ± 6.78	<0.001*
36 h.	78.7 ± 7.1	74.5 ± 7.06	<0.001*
48 h.	81.54 ± 8.08	74.9 ± 7.56	<0.001*

Data are presented as mean ± SD. *: p value < 0.05 is considered statistically significant. Heart rate is measured in beat per minute. Group E: Epidural group. Group D: Dexmedetomidine group. Min.: minute. h.: hour.

RAMSAY score was significantly higher (**Figure 2**) in dexmedetomidine group. There was no statistically difference between both groups in terms of SpO_2, age and sex.

As regards complications; two cases in the epidural group experienced urine retention that required urinary catheterization. Three cases in the epidural group experienced nausea that was treated successfully by ondansetron. Only one case was reported to have a dural puncture during insertion and by follow up no post-dural puncture headache was noted. Otherwise no other complications were reported in either group.

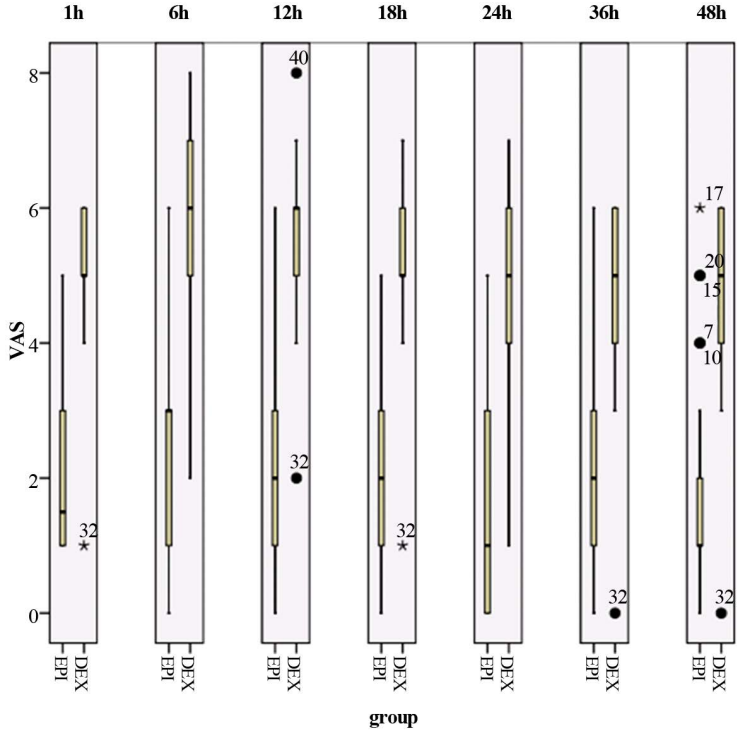

Figure 1. VAS (Visual Analogue Scale) in Epidural and Dexmedetomidine groups. The middle black solid line represents the median, the upper and lower margins of each box are IQR (inter-quartile range) and the whiskers are maximum and minimum.

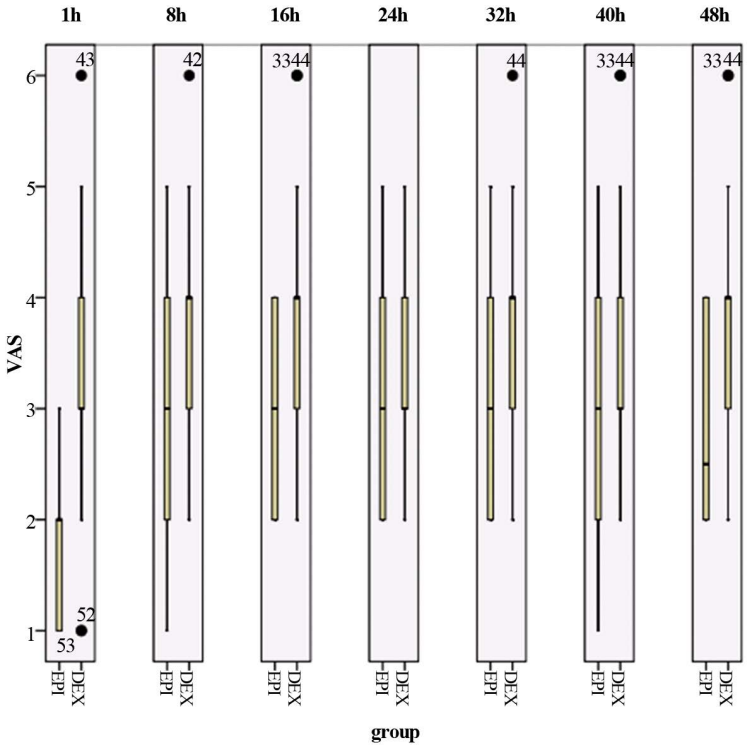

Figure 2. RAMSAY score in Epidural and Dexmedetomidine groups. The middle black solid line represents the median, the upper and lower margins of each box are IQR (inter quartile range) and the whiskers are maximum and minimum.

4. Discussion

Traumatic flail chest is a serious injury that can impair ventilation due to pain, restricted chest movement and abnormal paradoxical chest movement and mechanics. The ventilatory complications of flail chest can affect patient outcome, we compared between the epidural analgesia and parenteral dexmedetomidine as regards the effectiveness in preserving ventilation with providing analgesia and hemodynamic stability.

The results of the present study showed that PaO_2/FIO_2 ratio was significantly higher in epidural group than dexmedetomidine group and $PaCO_2$ was significantly lower in epidural group than dexmedetomidine group, these Parameters suggestive that the oxygenation and ventilation was better in epidural group than dexmedetomidine group. The incidence of mechanical ventilation was lower in epidural group (p value < 0.04). VAS was also significantly lower in epidural group after 1 hour [1.5 (1 - 3)] versus 5 (4.75 - 6) in dexmedetomidine group (p value < 0.001).

These results supports the superiority of neuro-axial blockade over the parenteral alpha 2 agonist dexmedetomidine however the limiting factor for the use of thoracic epidural was the failure rate (15%) and the unavailability in cases with contraindications to epidural insertion. These findings are in agreement with Grau T. *et al.*, who reported that, the epidural anesthesia and analgesia are particularly powerful instruments in acute pain management with failure to access the epidural space due to the challenging anatomy or the experience and skill of the anesthesiologist the failure rate around 30% in inexperienced thoracic anesthetist [21].

Our study outcome correlates well with results of National Trauma Data Bank (NTDB), which compared pain management for patients with flail chest, via the use of epidural catheters, intercostal nerve blocks, or patient-centered analgesia and concluded that, the use of epidural catheters seems to be the most preferred method, as epidural catheters allow for improved subjective pain perception, pulmonary functions tests, a lower rate of pneumonia, as well as decreased length of time on a mechanical ventilator or ICU stay [7] [8].

Luchette FA *et al.* who compared epidural catheters with intra-pleural catheters in a previous randomized controlled trial and have concluded that epidural decreased pain and improved tidal volume and negative inspiratory pressures [22].

Topçu *et al.* who Compared effectiveness of thoracic epidural and intravenous patient-controlled analgesia for the treatment of rib fractures pain in intensive care unit and concluded that the use of thoracic epidural analgesia with infusion of local anesthetics and opioids provides more effective analgesia and shortens length of intensive care unit stay in chest trauma patients with more than three rib fractures [23].

The epidural group in our study had lower rate of mechanical ventilation and lower sedation score. RAMSAY score was evidently higher in the dexmedetomidine group in our study. Despite profound sedative properties, dexmedetomidine is associated with only limited respiratory effects. This goes with, the findings of Venn RM [24] who concluded that dexmedetomidine has wide safety margin even at plasma levels up to 15 times of those typically achieved during therapy.

The study question that compared dexmedetomidine to epidural was based on the analgesic properties of dexmedetomidine, as it has supra spinal and spinal mechanism of action. Apparently, the dorsal horn of the spinal cord is believed to be the site of the analgesic effect of dexmedetomidine. The locus ceruleus is the site of origin for the descending inhibitory system which exerts its action at the synapses of the dorsal horn. When these sites are stimulated, they exert inhibitory effect on the firing nociceptor neurons stimulated by peripheral A and C [25].

A significant decrease in the heart rate and mean arterial blood pressure was observed in the dexmedetomidine group in comparison to the epidural group. The combination of the decrease in sympathetic tone and partly baroreceptor reflex and enhanced vagal activity are responsible for this effect of dexmedetomidine [26].

Bekker *et al.* [27] observed minimal hemodynamic changes with dexmedetomidine in a case report of a wake craniotomy. They reported that their findings are consistent with the reported effect of small-dose dexmedetomidine infusion on cardiovascular function in healthy adults [28]. However, they concluded that Dexmedetomidine produces dose-dependent decreases in blood pressure and heart rate as a result of its agonistic effect at the a 2-adrenoreceptors. They observed a decrease in heart rate and blood pressure after the initial dose of dexmedetomidine. Their infusion rate was comparable to ours. They used the same loading dose. The analgesic action of dexmedetomidine was not dose-dependent; they observed an apparent ceiling effect at 0.5 μg/kg [29].

Jaakola *et al.* [30] tested the analgesic effect of different doses of dexmedetomidine. They proved that the maximum analgesic effect was obtained at a rate of 0.5 μg/kg. Accordingly, we used this rate of infusion.

Arain *et al.* [31] who compared dexmedetomidine and morphine for postoperative analgesia after major inpa-

tient surgery. Thirty minutes before the end of the surgery, one group received an initial bolus dose of dexmedetomidine one µg/kg after that continuous infusion at a rate of 0.4 µg/kg/hr. was started and discontinued at the end of surgery. Before the end of surgery, the other group received an intravenous bolus of morphine 0.08 mg/kg. The morphine group experienced 66% more morphine requirements to achieve this analgesic effect. Similarly, in our study we observed a decrease in the opioid requirement in the dexmedetomidine group in comparison to patients with failed epidural [31].

Venn *et al.* [32] evaluated systemic $\alpha 2$ agonists in spontaneously breathing patients in the ICU after surgery. Apart from decreased morphine requirements, Dexmedetomidine had no effect on respiratory rate, arterial hemoglobin saturation, pH, and $PaCO_2$. Interestingly the PaO_2/FIO_2 ratios were improved in the dexmedetomidine group compared to patients receiving morphine and midazolam boluses.

In contrast to results of our study, Animal study done by Bloor BC *et al.* [33] who examined effects of dexmedetomidine on ventilatory drive in the dog and concluded that it causes an increase in minute ventilation, a decreased hypercapnic drive without a change in arterial blood gasses.

On the other hand, in human trials, done by Belleville JP *et al.* [34] who examined the Effects of intravenous dexmedetomidine in humans, concluded that the infusion of dexmedetomidine causes a mild decrease in minute ventilation and an increase in $PaCO_2$. All these effects are much less pronounced than those of opioids and resemble in order of magnitude to those seen during profound sleep. However, we cannot correlate these data with ours as all respiratory parameters are affected by lung injury and chest pain, in addition the analgesic effect of dexmedetomidine possibly improves ventilation.

Limitations

A limitation of our study was the absence of blinding, and being a monocentric study, a future study should be a multicenter with larger sample size in order to validate data of the current study.

5. Conclusion

This study concludes that epidural analgesia is more effective than parenteral dexmedetomidine in the non-surgical conservative management of flail chest, however, parenteral dexmedetomidine can represent a good alternative in cases where epidural is not a possible option due to either the presence of a contraindication or the absence of an experienced anesthetist. Dexmedetomidine has analgesic and sedative effect with minimal respiratory depression.

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