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The effects of dexmedetomidine based sedation for noninvasive mechanical ventilation

Noninvaziv mekanik ventilasyonda deksmedetomidin ile sedasyonun etkileri

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ÖZET

Amaç : Noninvaziv mekanik ventilasyon (NIMV) bazen hasta uyumsuzluğu ve başarısızlıkla sonuçlanabilmektedir. Bu çalışmada, NIMV desteği altındaki hastalarda; deksmedetomidinin sedatif etkisini ve noninvaziv mekanik ventilasyona uyum üzerindeki etkilerini araştırmak amaçlanmıştır.

Materyal ve Metod: Hastalar rastgele iki gruba ayrılarak, deksmedetomidin uygulanacak grup ve kontrol grubu oluşturuldu. Deksmedetomidin grubunda Ramsey sedasyon skoru 3-4 olacak şekilde 0,2–0,7 μgr/kg/saat idame dozu ayarlandı. Deksmedetomidin başlandıktan sonra 1.dk, 10.dk, 30.dk, 1.saat, 4. saat, 24. saat ve herhangi bir nedenle infüzyon sonlandırıldığında veya noninvaziv mekanik ventilasyon sonlandırıldığında ortalama arter basıncı, kalp atım hızı, solunum frekansı, arter kan gazı, PaO2/FIO2 ve Ramsey sedasyon skoru değerleri kaydedildi.

Bulgular: Noninvaziv mekanik ventilasyon desteği altındaki 30 ajite ve uyumsuz hasta değerlendirilmeye alındı. Deksmedetomidin (D) grubunda ortalama arter basıncı, kalp atım hızı ve solunum frekansı ölçümlerinde anlamlı düşüş tespit edildi. Deksmedetomidin grubunda Ramsey sedasyon skorlarında 1.dk göre 10.dk dan stoplama süresine kadar geçen zamanlarda istatistiksel olarak anlamlı yükseliş (1'den 3'e) görüldü. Deksmedetomidin grubunda PaO2/FIO2 değerlerinde görülen yükseliş anlamlı bulundu.

Sonuç: Noninvaziv mekanik ventilasyon gereksinimi olan hastalarda, Deksmedetomidin güvenli ve etkin bir sedatif ajandır.

Anahtar kelimeler : Dexmedetomidin, sedasyon, NIMV.

SUMMARY

Aim:Noninvasive mechanical ventilation (NIMV) is associated with a large number of failures, and patient refusal. The purpose of the study was to assess the feasibilty and safety of dexmedetomidine during NIMV.

Methods: The patients allocated randomly in to two groups; Dexmedetomidine (D) group and control (C) group. In D group, Dexmedetomidine infusion rate was set between 0,2-0,7 μgr/kg/hour to reach a Ramsey sedation score (RSS) between 3-4. Mean arterial pressure, heart rate, respiratory rate, blood gas analysis, PaO2/FIO2 and RSS documented at 1 min, 10 min, 30 min,1 hour, 4 hour, 24 hours after Dexmedetomidine infusion started and after the end of infusion and at non invasive mechanical ventilation support cessation in any reason.

Result: Thirty patients under NIMV support with agitation and ventilatory dyssynchrony were included in this study. In D group mean arterial pressure, heart rate, respiratory rate measurements were significantly lower than C group. RSS shown significant rises (1 to 3) at the period between 1th min to 10th min to the cessation time in D group ($p<0.01$) and PaO2/FIO2 values were significantly higher than control group. Conclusion: Dexmedetomidine is safe and effective agent for the sedation of the patients under NIMV support. **Sonuç**: Lale uçlu fiber ile endovenöz lazer ablasyon kronik venöz yetmezlik tedavisinde güvenli ve etkin bir yöntemdir.

Keywords: Dexmedetomidine, sedation, NIMV.

Introduction

The aims of pharmacological sedation in the intensive care unit are to protect patients from stressful and harmful stimuli, anxiolysis and to provide night's sleep and amnesia. Selecting the appropriate sedation agent is fundamental for safety and control of the patients in intensive care units (1-5).

The success of mechanical ventilation depends on the patient's compliance with the mechanical ventilation instrument and tolerance. Dexmedetomidine is one of a wide variety of pharmacological agents used for this purpose. Dexmedetomidine is a selective alpha-2 receptor agonist with sedative, antihypertensive, amnestic, and analgesic effects (6-11). It provides sedation and analgesia that patients can be kept to be awakened and in a cooperative state, without causing respiratory depression. The distribution half-life is about 6 minutes with a rapid distribution phase, and elimination half-life is approximately two hours. The most common side effects include hypotension, hypertension, bradycardia, nausea, vomiting, dry mouth, and hypoxia (7,9,10,11).

Ensuring the ideal level of sedation is extremely important. Several scoring systems have been used to determine the level of sedation (2, 4, 6, 7, 8, 12). Ramsey Sedation Scale (RSS) is one of them, and is a scale used in assessment of increased engine response depending on the depth of sedation. The aim of the conducted studies was to maintain sedation levels between 2 and 4 according to the RSS (2, 4, 7, 9, 10).

The aims of this study were to examine the sedative effects of Dexmedetomidine in agitated patients under Noninvasive mechanical ventilation (NIMV) support and to evaluate the compliance of patients with NIMV.

Materials and Methods

This study was conducted in the Medical Faculty of Istanbul University after receiving approval of the faculty ethics committee during a one-year period. The patients who are agitated and incompatible under non invasive mechanical ventilation support in the Intensive Care Unit were included in the study.

Exclusion criteria included the patients with heart block and whom non invasive mechanical ventilation process contraindicated, cardiopulmonary resuscitation, respiratory arrest, conditions of severe hemodynamic instability (cardiac arrest, ventricular arrhythmias, unstable cardiac rhythm, cardiogenic shock, hypotension unresponsive to fluid resuscitation), unconsciousness, status asthmaticus, status epilepticus, causes of central hypoventilation, two or more organ failure, tracheostomy and facial deformity, recent oronasal and upper gastrointestinal surgery, upper gastrointestinal bleeding, and shock. Patients were randomly divided into two groups: the Dexmedetomidine(D) group and the control(C) group. A loading dose of 1 mg/kg dexmedetomidine was initiated, then 10 minutes later the dose reduced to 0,2 to 0,7 μgr/kg/h. The maintenance dose was set to maintain RSS between 3 and 4. Dexmedetomidine level was reduced in cases increase or reduction >20% in arterial blood pressure and heart rate. Dexmedetomidine level was maintained under 0,7 μgr/kg/hour, during 24 hours infusion. The mean arterial pressure (MAP), heart rate (HR), respiratory frequency (FR), arterial blood gases, PaO2/FiO2 and RSSs were recorded after the 1st, 10th, 30th minutes, 1st, 4th and 24th hours after the infusion of dexmedetomidine and when the infusion is terminated for any reason or non invasive mechanical ventilation terminated. When additional sedation is required 0,02 to 0,03 mg/kg midazolam was used.

Side effects such as heart block, hypertension, hypotension, bradycardia, arrhythmia, nausea, vomiting and hyperglycemia were recorded.

If the desired level of compliance of the patient with NIMV mask was achieved, spontaneous breathing was followed with venturi mask intermittently. Durations of spontaneous breathing with mask and NIMV were recorded.

Duration of dexmedetomidine infusion, the used doses, the reasons for terminating infusion of dexmedetomidine, additional analgesics during the infusion, requirement and quantity of sedation, cause and duration of the transition to invasive mechanical ventilation were recorded during 24 hours of Dexmedetomidine infusion therapy.

Statistical Analysis

SPSS (Statistical Package for Social Sciences) for Windows 10.0 software was used in the statistical analysis of the results. Descriptive statistics were expressed as means and standard deviation. Unpaired Student's t test was used in the comparison of variables with the normal distribution, and paired Student's t test was used in group comparisons. Mann Whitney U test was used in the inter-group comparisons of the variables don't match the normal distribution and the Wilcoxon test was used in intra-group comparisons. Chi-square test was used to compare qualitative data. The results were accepted in 95% confidence interval, and a value of $p<0.05$ accepted statistically significant.

Results

The study included a total of 30 patients, between ages of 33 and 83, who were hospitalized in the in intensive care units of Istanbul University Faculty of Medicine Department of Anesthesiology and Reanimation, during the study period. The mean age of the patients was 61,73±12,76. 16 (53.3%) patients were female and 14 (46.7%) were male. D group included 15 patients and the C group included 15 patients. In the D group the duration of infusion was 14.56±9.41 hours, and the average maximum infusion dose was 0,62 ± 0,14 μgr/kg/h respectively. The demographic characteristics of the patients are shown in Table 1.

Table 1: Patient characteristics (mean standard deviation or number).

		Dexmedetomidine Group Mean±SD	Control Group Mean±SD	*Test values*
Age (year)		63,60±12,22	59,87±13,44	*t: 0,796; p:0,433*
Height (cm)		165,00±10,85	164,00±9,30	*t:0,271; p:0,788*
Body weight (kg)		73,33±11,90	74,80±18,69	*t:0,256; p:0,800*
APACHE II score		19,87±4,78	24,07±5,46	*t:2,242; p:0,033**
Hospitalization (day)		81,00±66,52	23,40±16,19	*t:3,258; p:0,005***
		n (%)		**n (%)**
Gender	**Female**	8 (%53,3)	8 (%53,3)	*χ^2:0,000; p:1,000*
	Male	7 (%46,7)	7 (%46,7)	
Mortality		8 (%53,3)	10 (%66.7)	*χ^2:0,566; p:0.456*

t: student t test X²: chi-square test * p<0,05 **p<0,01

APACHE II scores were found to be significantly lower in the D group (p<0.05). Duration of hospitalization was significantly higher in the D group compared to the C group (p<0.01).

The 30th minute MAP values showed slightly decrease in the D group and this decrease was statistically significant (p<0.01). D group were found to be significantly lower at the end of infusion when compared with the C group (p<0.01). In the D group, the decreases in the levels of MAP at the 1st, 10th and 30th minutes, and 1st hour were significant at the level of p<0.01, the decrease the 4th and 24th hours were significant at the level of p<0.05, and the decreases measured when the infusion was terminated were significant at the level of p<0.01(Table 2, Figure 1).

Table 2: Mean arterial blood pressure (MAP) groups (mmHg) the distribution of the measurement (mean standard deviation or number).

MAP	Dexmedetomidine Group Mean±SD	Control Group Mean±SD	*Test values*
1.min	86,60±12,56	97,60±27,84	*t: 1,395; p:0,179*
10. min	73,67±14,64††	91,40±23,29	*t: 2,496; p:0,019**
30. min	73,46±13,88††	91,73±18,22	*t: 3,088; p:0,005***
1. hour	77,75±11,09††	91,13±17,75	*t: 2,445; p:0,022**
4. hour	79,09±7,40†	85,92±14,62	*t: 1,519; p:0,144*
24. hour	69,00±11,57†	89,45±17,25	*t: 2,584; p:0,021**
Infusion Termination	69,40±16,89††	89,00±18,22	*t: 3,211; p:0,003***

t: student t test , Evaluation of intra-group Paired Samples test * p<0,05, **p<0,01 † Intra-group assessment by 1.min p<0,05 †† Intra-group assessment by 1.min p<0,01

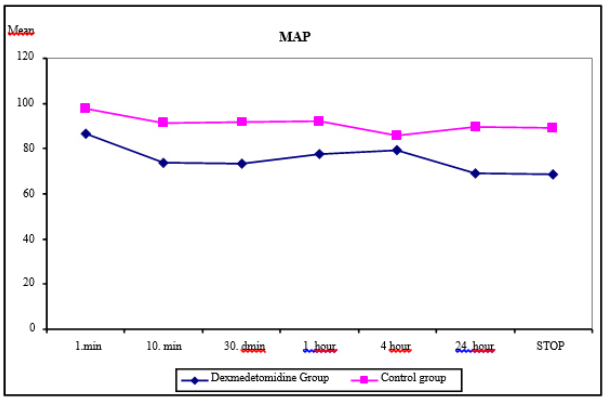

Figure 1: The distribution of groups of MAP measurement.

No statistically significant differences were observed between the D group and the C group in terms of HR levels at the 1st, 10th, and 30th minutes and 1st, 4th, and 24th hours, and during the end of the infusion (p>0.05) (Table 3).

Table 3: Heart rate of the group (HR) (beats/min) distribution of the measurements (mean and standard deviation or number).

HR	Dexmedetomidine Group Mean±SD	Control Group Mean±SD	*Test values*
1.min	108,53±23,45	114,07±20,38	*t: 0,690; p:0,496*
10.min	105,60±26,80	110,80±15,43	*t: 0,651; p: 0,522*
30. min	98,93±18,69††	108,40±12,39	*t: 1,635; p: 0,113*
1 hour	98,91±18,43†	104,06±15,43†	*t: 0,637; p: 0,530*
4 hour	94,90±22,61††	108,71±15,98	*t: 1,789; p:0,087*
24 hour	98,83±25,49	109,81±20,05	*t: 0,983; p:0,341*
Infusion Termination	94,00±20,45††	108,46±21,23	*t: 1,900; p:0,068*

t: student t test , Evaluation of intra-group Paired Samples test * p<0,05, **p<0,01 † Intra-group assessment by 1.min p<0,05 †† Intra-group assessment by 1.min p<0,01

In the D group, no significant differences were observed between the HR measurements taken at 1st and 10th minutes (p>0.05); whereas the decrease at the 30th minute was statistically significant (p<0.01). The significance level of the decrease was p<0.05 at the 1st hour, and p<0.01at the 4th hour. There were no significant differences between the HR measurements at the 1st minute and 24th hour (p>0.05), the difference at the end of the infusion was significant at the level of p<0.01 (Figure 2).

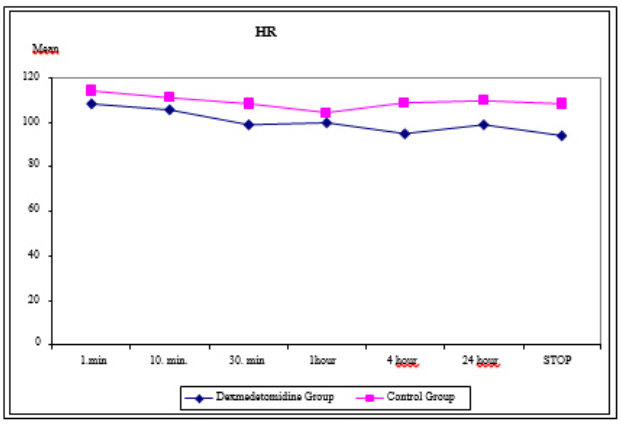

Figure 2: The distribution of groups of HR measurement.

Respiratory frequencies did not differ significantly between the groups at 1st, 10th, and 30th minutes and 1st, 4th, and 24th hour, and at the end of the infusion (p>0.05) (Figure 3).

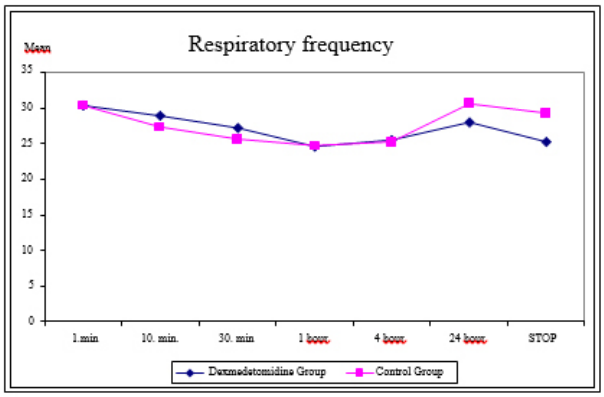

Figure 3: Distribution of the group of respiratory frequency measurement.

RSS groups did not differ significantly between the groups at the 1st minute (p>0.05). RSS were highly significantly higher in the D group (p<0.01) and significantly higher at the 30th minute, 1st and 4th hours (p<0.05). There were no significant differences between the RSS of both groups at the 24th hour and at the end of infusion (p>0.05).

In the D group RSS increased significantly from the 10th minute until the end the infusion when compared to the 1st minute (p<0.01). In the C group the RSS did not increased significantly at the 10th minute when compared to the 1st minute (p>0.05), while the increases were highly significant at the 30th minute and 1st hour (p<0, 01). The increases at the 4th hour were statistically significant, while the increases at the 24th hour, and at the end of infusion were highly significant (p> 0.01) (Figure 4).

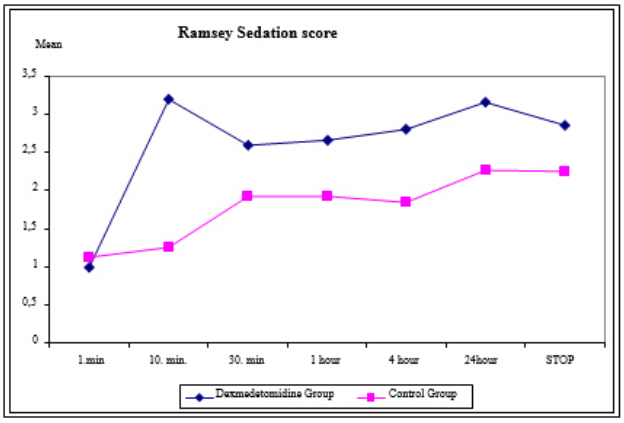

Figure 4: The distribution of grupsa of Ramsey sedation scores.

PaO2/FIO2 levels did not differ significantly between the groups at 1st, 10th, and 30th minutes and 1st, 4th, and 24th hour, and at the end of the infusion (p>0.05).

In the D group no significant differences were found between the PaO2/FIO2 levels at the 10th minute when compared with 1st minute and (p>0.05), while the increases at the 30th minute were statistically significant (p<0.05). The PaO2/FIO2 levels further increased at the 1st hour and these increases were highly significant (p<0.01), whereas the differences at the 24th hour and at the end of the infusion were not significant when compared to 1st minute (p>0.05). In the C group PaO2/FIO2 levels increased significantly at the 10th minute when compared with the 1st minute (p<0.05). The PaO2/FIO2 levels further increased at the 30th minute and the difference was highly significant (p<0.01). No statistically significant changes of PaO2/FIO2 levels were observed at the1st and 4th hours when compared with the levels at the 1st minute (p>0.05). The increase of PaO2/FIO2 levels were significant at the 24th hour (p<0.05), and there were no significant differences at the end of the infusion when compared with the 1st minute (p>0.05).

PaO2 levels did not differ significantly between the groups at the 1st, 10th, and 30 th minutes, at the 24 th hour and at the end of infusion (p>0.05). PaCO2 levels did not differ significantly between the groups at 1st, 10th, and 30th minutes and 1st, 4th, and 24th hour, and at the end of the infusion (p>0.05). SaO2 levels were found to be significantly higher in the D group at the 1st minute (p<0.05). There were no statistically significant differences between the SaO2 levels of D and C groups from the 10th minute until the end of the infusion when compared with the 1st minute (p>0.05). Blood glucose values did not differ significantly between the groups at any measurement points (p>0.05).

Although the median duration of ventilation with mask was was found to be lower in the D group, the difference was not statistically significant (p>0.05). Again there were no differences between the groups in terms of the duration of non-invasive mechani-

cal ventilation (p>0.05). In the C group the ratio of additive drugs that were used to support sedation (Midazolam 0,02 to 0,03 mg/kg) was significantly higher (p<0.01). No side effects were observed in the C group, whereas 53.3% of the patients had side effects in the D group; the difference was significant (p<0.01). There were no significant differences between the groups in terms of the need for intubation, maintenance of non invasive mechanical ventilation and spontaneous respiration rate (p>0.05) (Table 4).

Table 4: Duration of NIV, sedative use, results (mean standard deviation or number).

	Dexmedetomidine Group Mean±SD	Control Group Mean±SD	*Test values*
Spontaneous breathing time with Mask (hour)	4,33±3,77 (3,5)	9,72±7,76 (4,0)	***Z: 1,373; p:0,180***
Duration of NIV (hour)	12,83±8,87	12,36±8,01	***t: 0,152; p:0,880***
	n (%)	**n (%)**	
Use of additional sedative agent	2 (%13,3)	9 (%60,0)	***χ^2: 7,033; p:0,008*****
Advers effects	8 (%53,3)	0	***χ^2: 10,909; p:0,002*****
Results			
Intubation	6 (%40,0)	6 (%40,0)	***χ^2: 1,000; p:0,607***
Continue NIV	7 (%46,7)	5 (%33,3)	
Spontaneous respiration	2 (%13,3)	4 (%26,7)	

Z: Mann Whitney U test t: student t test
2: Ki kare test **p<0,01

In the C group the infusions were terminated in cases of hypercarbia, hypoxia, transition to spontaneous respiration, loss of consciousness, and in the D group intubations were terminated in cases of hypotension, intubation requirement, hypercarbia, hypoxia and transition to spontaneous respiration. In the C group 46.7% and in the D group 33.3% of the patients completed the 24-hour infusion time (Table 5).

Table 5: Causes of infusion cessation.

	Control group		Dexmedetomidine group	
	n	**%**	**N**	**%**
Hypotension	0	0	2	13,3
Intubation	0	0	2	13,3
Hypercarbia	1	6,7	2	13,3
Hypoxia	3	20,0	2	13,3
Spontaneous respiration	2	13,3	2	13,3
Unconciousness	2	13,3	0	0
24 hour infusion	7	46,7	5	33,3

In the D group 53.4% of the patients had hypotension and 6.7% of the patients had hyperglisemia, whereas no side effects were observed in the C group (Table 6).

Table 6: Advers effects

	Control group		Dexmedetomidine group	
	n	**%**	**n**	**%**
Hypotension	-	-	8	53,4
Hyperglycemia	-	-	1	6,7
No side effects	15	100,0	7	46,7

Discussion

The success of mechanical ventilation depends on the patient's compliance with the mechanical ventilation instrument. Therefore sedation and analgesia are often required in mechanically ventilated patients in the intensive care unit (6). In a similar study, Galves-Band C at al investigated the availability of Dexmedetomidine to reduce anxiety in patients undergoing non invasive mechanical ventilation support. They evaluated Riker Sedation Agitation Scale (SAS), motor activity assessment scale (MAAS), heart rate, respiratory rate, mean arterial pressure and arterial blood gas at the 30th minute, 1st, 4th and 24th hours of Dexmedetomidine infusion and at the end of mechanical ventilation. In this study they used Dexmedetomidine at the dose of 0,1-0,3 μgr/kg/h, and at the end of the study they found that agitation decreased significantly with the decrease in sedation scores (SAS, MAAS). Hypoxemia developed in all patients (PaO2/FIO2=122) and respiratory acidosis was observed in 47% of the patients. Oxygen saturation improved 30 minutes after the infusion of

Dexmedetomidine, and remained stable during the study. They did not find any significant difference in mean arterial pressure, heart rate, and respiratory rate. They concluded that Dexmedetomidine is effective in ensuring compliance with non invasive mechanical ventilation in intensive care patients, by reducing anxiety (7).

In our study, in the D group RSS increased statistically significantly (from 1 to 3) from the 10th minute until the end of the infusion ($p<0.01$). Similar to that study, in our study, in D group PaO2/FIO2 levels increased significantly at the 30th minute ($p<0.05$), further increased at the 1st hour ($p<0.01$) and remained high at the 4th hour ($p<0.05$). Unlike that study, in our study in the D group mean MAP levels decreased significantly at the 10th and 30th minutes, 1st hour ($p<0.01$), 4th and 24th hours ($p<0.05$) and at the end of the infusion($p<0$ 01), when compared to 1st minute. We also observed that MAP levels decreased at 10th ($p<0.05$), and 30th ($p<0.01$) minutes, 1st hour ($p<0.05$), 24th hour ($p<0.05$), and at the end of the infusion ($p<0.01$), when compared with the C group. In addition, in the D group HR decreased significantly at 30th minute, 4th hour and at the end of the infusion ($p<0.01$). We also found that respiratory rates decreased significantly at 30th minute, 1st hour and at the end of the infusion ($p<0.05$). We attributed the decreases in MAP and HR levels to that as a highly selective □-2 agonist, the main hemodynamic effects of Dexmedetomidine are hypotension and bradycardia. In addition, in our study the mean maximum infusion dose was $0,62\pm0,14$ μgr/kg/h which is higher than that in the study of Galves-Band et al (7).

Patil et al. found that in the intensive care unit acute delirium table improved within 48 hours due to sedative effect of Dexmedetomidine (in the Dexmedetomidine group 93.3%,in the control group 5.6% ($p<0.0001$)). In the same study they found hospital mortality rate was 6.6% in the Dexmedetomidine group and 22.2% in the control group ($p<0.4$) (8). In our study, APACHE II scores were significantly lower ($p<0.05$) and duration of hospitalization was longer ($p<0.01$) in the Dexmedetomidine group) when compared with the control group, whereas we did not find any differences between groups in terms of mortality ($p<0.05$).

Hall et al. found that dexmedetomidine provided analgesia, decline in instant memory and cognitive function, and sedation that can be easily awakened with verbal and physiological stimuli, with low doses. There were no significant differences in the cardiovascular system. They concluded that dexmedetomidine is useful in the postoperative period in the intensive care units (9).

Venn et al. conducted a phase 2 study to investigate sedative effect of dexmedetomidine in the intensive care unit. They administered 0,2 to 0,7 μgr/kg/h dose of dexmedetomidine for 7 days in patients under mechanical ventilatory support, they added propofol and morphine when sedation and analgesia required and they evaluated the level of sedation by using RSS. They increased infusion of dexmedetomidine to dose of 2,0 μgr/kg/h due to requirement of additional sedation, and reduced the infusion rate due to development of hypotension. In conclusion they suggested that dexmedetomidine is effective in critical partients who require mechanical ventilation in the intensive care unit, but the dose of infusion should be should be kept low (10).

Shehabi et al. evaluated sedative and cardiovascular effects of infusions of Dexmedetomidine that exceeded 24 hours. They administered Dexmedetomidine at a dose of 0,2 to 0,7 μgr/kg/h to achieve a RSS between 2 and 4. The mean infusion time was 71.5 (35-168) minutes and they found RSS between 2 and 5 in 83% of the patients. The reduction of incidence of excessive sedation from 13% to 3% over the first 24 hours was attributed to effects of prolonged anesthesia in postoperative patients or to possible accumulating effects of the sedatives used prior to the study. They found that systolic blood pressure decreased 16% and heart rate decrease 21% over the first 4 hours of Dexmedetomidine infusion. During Dexmedetomidine infusion minimal or additional sedation was required in 80% of the patients. They concluded that Dexmedetomidine may be used safely as an effective sedative and analgesic agent up to 7 days, on condition that taking the predictable hemodynamic effects into account (11).

Similarly, we found that MAP levels decreased significantly at the 10th and 30th minutes, and 1st hour ($p<0.01$), 4th and 24th hours ($p<0.05$), and at the end of infusion ($p<0.01$) in the Dexmedetomidine group. In addition HR were significantly low at the 30th minute ($p<0.01$), 1st hour ($p<0.05$), 4th hour, and at the end of infusion ($p<0.01$). Again similar to the results of Shehabi et al (10), we observed statistically significant increases in the RSS during dexmedetomidine infusion ($p<0.01$). Unlike that study, we observed that the requirement of additional sedation (midazolam 0,02 to 0,03 mg/kg) is significantly higher in the control group ($p<0.01$).

In this study we compared the sedative effect of Dexmedetomidine in agitated patients under non invasive ventilatory support in the intensive care unit, with the control group. We found that RSS increased significantly in the Dexmedetomidine group when compared with the control group, while requirement for additional sedation was found to increase in the control group. Mean arterial pressure decreased significantly in the Dexmedetomidine group when compared with the control group. In addition, increases of PaO2/FIO2 levels and decreases of heart rate and respiratory rate were statistically significant, in the dexmedetomine group. Heart rate, respiratory rate, mortality, the duration of non invasive mechanical ventilation mask and total mask did not differ significantly between the groups.

In conclusion, we suggest that adequate sedation can be provided by careful and controlled management of side effects of dexmedetomine in patients under non invasive mechanical ventilation in the intensive care units.

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