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CLINICAL REPORT

Administration of dexmedetomidine alone during diagnostic cardiac catheterization in adults with congenital heart disease: two case reports

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Abstract We report the clinical management of 2 adults with mental retardation because of trisomy 21 who were sedated with high-dose dexmedetomidine (DEX) alone during diagnostic cardiac catheterization (DCC). The first patient was a 25-year-old man with aortic regurgitation and ventricular septal defect. DEX increased his Ramsay sedation score; however, a high dose and bolus injection of DEX were required to perform an invasive procedure. Cardiovascular drugs were not administered and heart rate was maintained in the low 40s. The maximum predicted plasma concentration (pCp) of DEX was 2.3 ng/mL. The second patient was a 26-year-old woman who had developed hypoxia 20 years after palliative surgery for tetralogy of Fallot. High-dose DEX was administered to keep the bispectral index value below 70 and maintain an immobile state; her maximum pCp of DEX was 4.3 ng/mL. Percutaneous oxygen saturation was kept above 83%, because of the suspicion that DEX may increase the ratio of pulmonary artery flow to systemic artery flow. In both cases, no respiratory system complications occurred despite inspiration of room air, indicating the usefulness of DEX for DCC. However, because of DEX may affect DCC data, it is necessary to pay careful attention to the use of DEX during DCC.

Keywords Dexmedetomidine · Diagnostic cardiac catheterization · Congenital heart disease

Introduction

The usefulness of dexmedetomidine (DEX) in diagnostic cardiac catheterization (DCC) in children has been reported; however, the ratio of patients managed by DEX alone is still low and other drugs or rescue drugs are needed [1–4]. We encountered two cases of sedative management in which DEX was used as the sole sedative for DCC, and a higher dose of DEX than the standard dose based on the package insert (initial dose, 1 μg/kg over 10 min; maintenance dose, 0.2–0.7 μg/kg/h) was needed.

Case reports

The use of DEX for sedation during DCC was approved and monitored by the Research Ethics Committee of Asahikawa Medical College, and informed consent was obtained from the patients' parents.

Case 1

The patient was a 25-year-old man (height 153 cm; weight 48 kg) who had mental retardation because of trisomy 21. He was diagnosed with ventricular septal defect (VSD) and valvular disease in childhood. Because the time he had been confined to bed had increased during the previous few months, his parents admitted him to hospital. Because

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transthoracic echocardiography revealed severe aortic regurgitation by bicuspid aortic valve and VSD, DCC was scheduled. The patient did not seem to be tolerant of an invasive procedure, because of his mental retardation; a cardiologist therefore recommended that the patient be kept under monitored anesthesia during DCC. Famotidine (20 mg) was intravenously injected 30 min before the patient was brought into the catheterization room, and standard monitoring was started on his arrival there. A 2.0 μg/kg/h dose of DEX was administered to increase the Ramsay sedation score (RSS) from 1 to 2; subsequently, the anxious expression and grimace on his face disappeared. Because noxious stimuli, for example local anesthesia at the cannulation site, reduced the RSS to 1 again, it was necessary to administer a bolus injection of DEX at high dose (Fig. 1). Because glossoptosis can cause temporal airway obstruction and desaturation and result in body movement to regain deep breathing, we performed a chin lift. However, no oxygen supply was needed. Systolic blood pressure (SBP) was maintained above 96 mmHg, but heart rate (HR) decreased and was maintained at low levels. Cardiovascular drugs were not administered, because the HR could not be restricted to less than 40 bpm; hence, the cardiologist suggested we avoid the use of cardiovascular drugs because these drugs can affect cardiac data. The procedure was smoothly performed; DCC revealed the patient had severe aortic valve regurgitation with normal pulmonary artery pressure and a small VSD without oxygen step-up in the right ventricle. There was no complication during and after the procedure. The arterial blood gas analysis (BGA) data obtained 50 min after starting DEX administration were normal: pH was 7.35, partial pressure of carbon dioxide ($PaCO_2$) was 41.9 mmHg, and partial pressure of oxygen (PaO_2) was 92.0 mmHg. The maximum and mean predicted plasma concentrations of DEX, which were calculated by use of TIVAtrainer™ (available at: http://www.eurosiva.org/; accessed on May 1, 2010) after the procedure, were approximately 2.3 and 1.5 ng/mL, respectively.

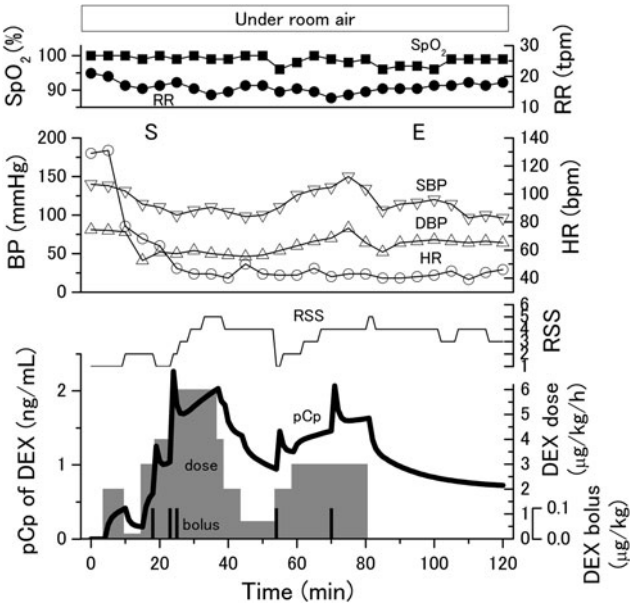

Fig. 1 Vital signs, dexmedetomidine (DEX) dose, and predicted plasma concentration of DEX in case 1. Dexmedetomidine (*DEX*) was continuously administered and a bolus injection was given to maintain RSS at 3 or more. Ramsay sedation score (*RSS*) changed according to the dose of DEX. Although blood pressure (*BP*) was stable, heart rate (*HR*) decreased and was maintained at a lower value after DEX administration. Percutaneous oxygen saturation (SpO_2) was at 96% or more and respiratory rate (*RR*) was within 13–21 times per min (*tpm*) throughout the surgery. Maximum and mean predicted plasma concentration of DEX were calculated as 2.3 and 1.5 ng/mL, respectively. SpO_2 percutaneous oxygen saturation, *BP* blood pressure, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *bpm* beats per minute, *DEX* dexmedetomidine, *DEX dose* dose of dexmedetomidine, *pCp* predicted plasma concentration, *RSS* Ramsay sedation score, *RR* respiratory rate, *tpm* times per minute, *S* start of the procedure, *E* end of the procedure

Case 2

The patient was a 26-year-old woman (height 140 cm, weight 69 kg) with mental retardation because of trisomy 21 who had been diagnosed with tetralogy of Fallot and atrioventricular septal defect at the time of birth and had undergone a modified Blalock–Taussig shunt procedure at the age of 6 years. Intracardiac repair surgery was not indicated, because of her limited left ventricular capacity; however, DCC under sedation was scheduled to evaluate the indication for additional left-to-right shunt surgery because her cyanosis was getting worse (SpO_2 of low seventies% when she inspired room air). She had no premedication, and was transferred to the catheterization room. Instead of RSS, bispectral index (BIS) value was monitored using BIS® monitor (Aspect, BIS Monitor A-2000; Nihon Kohden, Tokyo, Japan) to evaluate the depth of sedation, because the monitor became available for use in the catheterization room in our institution after case 1, in addition to standard monitoring. The initial dose of DEX was 10 μg/kg/h, which was subsequently adjusted to keep the BIS value within 40–70 and to make the patient immobile (Fig. 2). SpO_2 concentration (83–88%) and respiratory rate (RR; 16–20 times per min (tpm)) were stable, even though the patient was breathing room air. Because her hemodynamics did not change remarkably, no cardiovascular drugs were administered throughout the procedure. The arterial BGA data obtained 55 min after starting DEX administration were thought to be no problem: pH was 7.35, $PaCO_2$ was 37.4 mmHg, and PaO_2 was 57.7 mmHg. The maximum and mean predicted plasma concentration of DEX were calculated as 4.3 and 1.1 ng/mL, respectively.

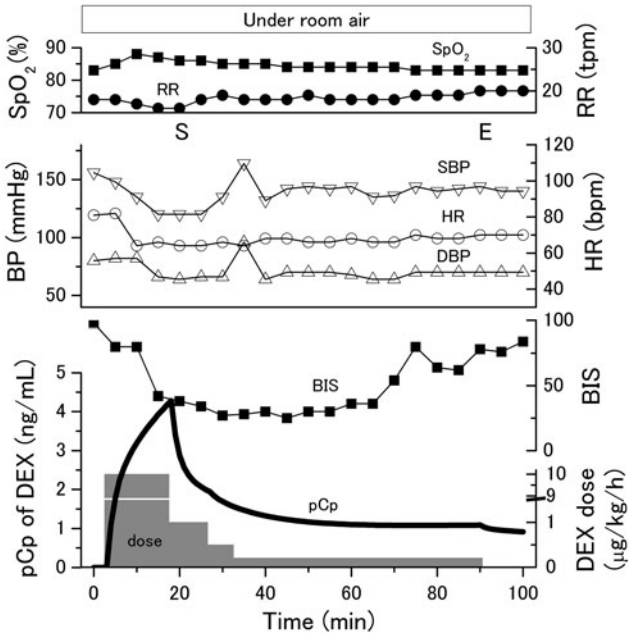

Fig. 2 Vital signs, dexmedetomidine dose, and predicted plasma concentration of dexmedetomidine in case 2. Dexmedetomidine (*DEX*) was administered to maintain the bispectral (*BIS*) value below 70 and to keep the patient immobile. BIS value changed according to the dose of DEX. Because the patients' hemodynamics were stable, no cardiovascular drugs were administered. Percutaneous oxygen saturation (SpO_2) increased to 89% after starting DEX administration and was maintained at 83% or more, and respiratory rate (*RR*) was within 16–20 times per min (*tpm*) throughout the surgery. Predicted plasma concentration of DEX temporarily increased to 4.3 ng/mL after starting DEX administration and was stabilized at approximately 1.1 ng/mL. SpO_2 percutaneous oxygen saturation, *BP* blood pressure, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *bpm* beats per minute, *DEX* dexmedetomidine, *DEX dose* dose of dexmedetomidine, *pCp* predicted plasma concentration, *BIS* bispectral index, *RR* respiratory rate, *tpm* times per minute, *S* start of the procedure, *E* end of the procedure

Discussion

Munro et al. [3] reported that DEX alone was sufficient for only 40% of the patients during DCC, whereas all other patients required treatment with an additional drug. A moderate dose of DEX (0.6–2.0 μg/kg/h) was used, however. Another recommended procedure is use of ketamine from the beginning of DCC [1, 2]. Moreover, many reports have indicated the need for and usefulness of high-dose DEX during invasive procedures in adults [5, 6]. Because our patients were adults, we increased the dose of DEX until sufficient sedation was achieved; these patients therefore received a high dose of DEX. The reasons for increasing DEX dose were as follows. First, the concentration of DEX required for sedation in DCC was similar to that required for other invasive procedures but was higher than that generally used for sedation in intensive care units [6, 7]. For our first patient, a high dose of DEX was required to restore the RSS, which was reduced to 1 because of local infiltration. For the second patient, a high dose of DEX was required to maintain the BIS value below 70 and to keep her immobile. Second, the pharmacokinetics for patients with congenital heart disease are not thought to be the same as those for healthy adults or for patients with other diseases [8, 9]. However, we have not addressed this issue in this report because we did not measure the actual plasma concentration of DEX.

In most patients with congenital heart disease, it is very important to prevent an anesthetic-induced change in Q_p/Q_s and to maintain a constant ratio of pulmonary to systemic blood flow (Q_p/Q_s) while performing DCC. Administration of high-dose DEX alone indicated the usefulness of DCC in terms of 2 points related to Q_p/Q_s. First, neither patient required oxygen supply throughout the procedure. Because oxygen supply reduces pulmonary vascular resistance and increases Q_p/Q_s, the aforementioned concerns were not relevant in these cases. Second, it has been reported that combined treatment with DEX and ketamine increased the $PaCO_2$ to 48 mmHg [1], which can also change the Q_p/Q_s. However, because only DEX was administered to these present patients, $PaCO_2$ was maintained within the normal range; it was also thought to have no effect on Q_p/Q_s. Therefore, the advantage of this technique is that the respiratory condition of the patient does not affect Q_p/Q_s during DCC.

However, there are some concerns about the effect of DEX on Q_p/Q_s. DEX can constrict both systemic vascular vessels and pulmonary vascular vessels and increase the SVR and PVR [10], indicating that DEX can change the Q_p/Q_s. We did not consider this possibility because there were no hemodynamic data before starting DEX administration or when DEX concentration was changed regularly. Nevertheless, because SpO_2 increased after starting DEX administration and was maintained above 83% in the second patient, which usually occurs because of a decrease in oxygen demand, we suspected that DEX increases pulmonary flow by changing Q_p/Q_s. We need to take into consideration the possibility that the data recorded during DCC might be affected by DEX.

Bradycardia, which is one concern when using DEX, occurred in case 1. HR was higher than the usual value (60–80 bpm in the ward) before induction of sedation, because the patient was excited by stress and anxiety. Even taking that into consideration, we should have intervened to maintain HR in the low 40s and temporarily 39 bpm, although a cardiologist asked us not to administer cardiovascular drugs. Moreover, one of the reasons for the bradycardia might have been an inadequate level of sedation (RSS 2) at the start of the procedure that was not able to prevent the patient from being agitated by invasion of local infiltration, because bolus injections and increase in the

dose of DEX were needed to treat the agitation; those treatments may have caused the adverse bradycardia. It was thought that management needed to be improved, because the quality of induction of sedation and vital signs in case 2 managed by the dose of DEX decided on the basis of the experience in case 1 were better. Moreover, critical complications did not occur as many reports have indicated a high dose of DEX might be safely used [5, 6]; however, strict monitoring is still required because DEX can affect hemodynamics. Furthermore, bradycardia in case 1 led to a concern similar to that mentioned above in terms of interpreting the DCC data. Reduced HR indicated that DEX reduced the cardiac output (CO), which is consistent with the results of a previous study [10]. Although underestimation of CO levels in the first patient did not affect his diagnosis, DEX should be used cautiously in other cases in which measurement of CO is important. When selecting a sedative or interpreting DCC data, the effect of DEX on DCC data should be taken into consideration.

Another drawback is vigorous body movements, which may be caused by glossoptosis. Glossoptosis occurred in one of these patients, which is consistent with findings of previous studies [1, 5]. Because glossoptosis can be treated by chin lift or by repositioning the head, it is not a key concern. However, it is necessary to pay attention to the occurrence of glossoptosis because if left undetected it can reduce SpO_2, which may cause deep breathing accompanied by body movements that may hamper the procedure, because DEX preserves the ventilatory response to hypoxia or hypercapnia.

We sedated the adult patients during DCC with a high dose of DEX. DEX is useful for diagnosing patients with congenital heart disease because normoxia and normocapnia are preserved, even when a high dose of DEX is administered. However, because the direct vasoconstrictive effect of DEX and the decrease of CO may affect the DCC data, although the extent of this effect is not yet known, it may not be appropriate to use DEX during DCC in some cases. Further studies should be conducted to prove the usefulness of DEX in DCC.

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Conflict of interest None.

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