Evaluation of Cerebral Microembolic Signals in Patients with Mechanical Aortic Valves

K Ghandehari, Z Izadimoud

Citation

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Abstract

Background and Purpose: Microembolic Signals (MES) are frequently observed in Transcranial Doppler (TCD) recordings of patients with Mechanical Heart Valve (MHV).We hypothesized that number of MES produced by MHV could be reduced with oxygen inhalation, if gaseous bubbles are the underlying cause.

Methods: All consecutive patients with St Jude aortic valves visiting the cardiology clinic were refered to the neurosonology unit, Valie Asr Hospital, Khorasan during August 2003 to August 2004. TCD monitoring of MES was performed with an ultrasound device (Vingmed 800 Oslo,Norway) and a 2 MHz probe. The MES counts were recorded during 30 minutes breathing room air and thereafter 30 minutes breathing through a facial mask with reservior bag (6 liter O_2 per minute). The criteria of MES detection were characteristic chrip sound, unidirectional signal, random appearance within cardiac cycle and intensity increase 3dB above background. The MES counts in two periods of monitoring were compared with paired T test and significance was declared at $P < 0.05$.

Results: Twelve patients (8 females and 4 males) were investigated. Oxygen ventilation caused a significant decrease of MES counts in the patients in comarison to breathing room air, $P = 0.001$. Thus MES in patients with MHV are mainly gaseous bubbles caused by blood agitation with MHV.

Conclusion: The quantity of MES in patients with MHV is not related to the risk of thromboembolic complications in these patients.

INTRODUCTION

Thromboembolism is a major cause of morbidity in patients with Mechanical Heart Valve (MHV) and Microembolic Signals (MES) are commonly observed in TCD recording of these patients[1]. Since at present the precise nature of MES in patients with MHV is unknown, different etiologies are under debate including local activation of coagulation system by the MHV, local increase of platelet aggregation and gaseous cavitation bubbles[2]. A major drawback of TCD technology is its failure to provide conclusive information concerning the underlying embolic material[1]. This issue is important in the evaluation of individual risk profiles and adequate management strategies. No correlations have been found between MES count and duration after MHV replacement, valve position, cardiac rhythm, intensity of anticoagulation and history of neurological deficit[3,4]. Assumption of gaseous bubbles as embolic material could explain this dicripancy, since microbubbles remain asymptomatic by imploding or crossing over to venous circulation through the capillary bed[4]. Patients with MHV had higher MES counts in common carotid artery than middle and anterior cerebral arteries because gaseous bubble emboli are bound to implode with time[5]. This study was carried out to evaluate the influence of oxygen ventilation on MES counts in patients with mechanical aortic valves.

PATIENTS AND METHODS

All consecutive patients with St Jude mechanical valves in aortic position were referred from the cardiology clinic to the neurosonology unit, Valie Asr Hospital, Khorasan during August 2003 to August 2004. Presence of more than 70% extracranial internal carotid artery stenosis, poor transtemporal window and intolerance to oxygen ventilation for 30 minutes served as exclusion criteria. The protocol entailed 30 minutes of TCD monitoring while the patient

was breathing room air and 30 minutes while breathing 6 liters per minute oxygen through a facial mask with a reservior bag (fractional inspiratory O_2 = 60%)[6]. This facial mask was placed over mouth and nose and held in place by an examiner, providing downward pressure with thumb to ensure a tight seal[6]. Patients were instructed to breath normally, avoid hypo or hyperventilation and immediately give notice if breathing become uncomfortable or other inspiratory or cardiac complaints occurred. MES monitoring was performed with an ultrasound device (Vingmed 800, Oslo, Norway) and a 2MHz probe in 50-58 mm depth of right middle cerebral artery through transtemporal window. MES detection criteria included characteristic chrip sound, unidirectional signal, random appearance in cardiac cycle and intensity increase 3dB above background[1]. The paried T test was applied for comparison of MES counts during oxygen ventilation and resting periods. Significance was declared at $P<0.05$.

RESULTS

A total of 14 patients were enrolled in the study. One of these patients did not tolerate oxygen inhalation long enough and developed chest tightness and dizziness that led to immediate termination of oxygen inhalation. The mask couldnot be tightly applied in an additional patient because of facial hair. 12 patients (8 females and 4 males) with mean age 38.61; SD 14.41 were investigated. The influence of oxygen ventilation on MES counts in each of the patients is displayed in table1. We found a significant decrease of MES counts during oxygen ventilation in comparison to resting period, P=0.001. Thus MES in patients with MHV are mainly composed of gaseous bubbles.

Figure 1

Table 1: MES counts of the patients during 30 minutes respiration in room air and 30 minutes oxygen ventilation

Patient Number	Room Air Ventilation	Oxygen Ventilation	Patient Number	Room Air Ventilation	Oxygen Ventilation
1	54	30	7	15	2
2	18	1	8	42	12
3	21	6	9	102	4
4	198	30	10	36	21
5	258	48	11	6	0
6	95	30	12	15	0

DISCUSSION

Some basic physiological considerations must be taken into acount before our results are analysed. Oxygen inhalation leads to alveolar denitrogenation at the same time nitrogen washout from the blood. Thus assuming nitrogen bubbles are underlying embolic material in patients with MHV, one would expect an exponential reduction in MES counts under oxygen inhalation[7]. Although all our patients showed significant reduction in MES count during oxygen ventilation, this decreased fraction was not equal between them. Individual differences of denitrogenation procedures depending on lung function could cause this finding. The fraction of inspired oxygen that actively reaches the lung under this procedure is always less than the concentration delivered because of mixing of incoming oxygen with ambient room air entertained by the mask and is strongly dependent on breathing pattern. A portion of the detected MES could still arise from coexisting fibrin thrombi on the mechanical aortic valve or native cardiac embolic sources. The quantity of these MES would obviously not be affected by oxygen inhalation[7]. Complete elimination of MES during oxygen inhalation was described by Kaps et al[2]. It must be stressed that our results only apply to patients with MHV and can not be extrapolated to other patients with potential native cardiac or arterial embolic sources. If the underlying embolic material in patients with MHV consisted of cavitation bubbles, no difference in MES counts between neurologically symptomatic and asymptomatic patients would be expected because cavitation bubbles would easily cross over to venous side without causing major vessel obstraction[8]. Other studies has shown that MES counts are highest in patients with Bjork-Shily monostrut valves, significantly lower in St Jude valve carriers and lowest in those with Medtronic-Hall valves[9].

Significant reduction of MES counts in our patients with MHV during oxygen ventilation shows that cavitation bubbles are responsible for a large proportion of the MES in these patients. This cavitation bubbles are probably released during blood agitation through fluid acceleration and deceleration caused by MHV closure.

Thus the quantity of MES in patients with MHV is not related to the risk of thromboembolic complications in these patients.

CORRESPONDENCE TO

Dr Kavian Ghandehari, MD,FLSPAssociate Professor of Neurology, Valie Asr Hospital, Southern Khorasan University of Medical Sciences, Ghaffari Street, Birjand, Khorasan, Iran Telfax+98 561 4430076 Correspondence: kavianghandehari@yahoo.com

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Author Information

Kavian Ghandehari, M.D., FLSP
Associate Professor, Department of Neurology, Southern Khorasan University of Medical Sciences

Zahra Izadimoud, M.D.
Physician, Department of Cardiology, Southern Khorasan University of Medical Sciences