A Case of Nonpenetrating Traumatic Aortic Regurgitation Detected by Transesophageal Echocardiography

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ODA, H., TANAKA, T., YAMAZAKI, Y., ITO, E., MIIDA, T., HIGUMA, N. *A Case of Nonpenetrating Traumatic Aortic Regurgitation Detected by Transesophageal Echocardiography.* Tohoku J. Exp. Med., 1997, **182** (1), 93-101 — A 67-year-old man, who had fell 5 meters, landing on his back, one month before, was referred because of heart failure due to aortic regurgitation (AR). Transesophageal echocardiogram (TEE) confirmed injuries in the aortic valve and the Valsalva sinus of the aorta before the surgery: the intimal flap in the Valsalva sinus of right coronary cusp (RCC), the prolapse of the RCC, and the dissection by longitudinal length of 3 cm in the Valsalva sinus of noncoronary cusp (NCC), ending as a blind pouch. Postoperative TEE confirmed the dissection was not repaired in the Valsalva sinus of the NCC. In this instance, TEE was extremely useful, compared with transthoracic echocardiography, computed tomography and magnetic resonance imaging, to assess the mechanism of AR following a nonpenetrating trauma, and to know to what degree the aortic valve and the Valsalva sinus of the aorta were destroyed. ————— traumatic aortic regurgitation; nonpenetrating trauma; aortic dissection; perforation of aortic valve; aortic valve replacement
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Trauma is one kind of etiology of aortic regurgitation (AR). Damage to the aortic valve is the most common valvular injury following a nonpenetrating chest trauma (Cohn and Braunwald 1992). However, it is difficult to know to what degree the aortic valve has been destroyed before operation. We reported a case in which the transesophageal echocardiogram (TEE) was extremely useful to assess the mechanism of AR following a nonpenetrating trauma, and emphasized the necessity of an intraoperative TEE assessment for determining the result of the operation.

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CASE REPORT

A 67-year-old man fell 5 meters, landing on his back. Seven days later, he went to a hospital with complaints of dyspnea. He was diagnosed with post traumatic adult respiratory distress syndrome. Steroid, diuretics and catecholamine were administered to him along with oxygen inhalation. One month later, he was discharged free from symptoms. However, 4 days after being discharged, he complained of dyspnea. He was transferred to our hospital because of heart failure. He had no histories of valvular disease or disease related to atherosclerosis. He had no chest pain which suggested ischemic heart

Fig. 1. Transesophageal echocardiogram before operation. These cross-sectional images in systole disclose that there were a thick tear in the Valsalva sinus of the NCC and a thin tear in the Valsalva sinus of the RCC, and that the Valsalva sinus of the RCC was slightly located to the side of the right ventricle. The single arrow indicates a tear in the Valsalva sinus of the NCC and the double arrows indicate a tear in the Valsalva sinus of the RCC. LA, left atrium; RA, right atrium; N, Valsalva sinus of noncoronary cusp; L, Valsalva sinus of left coronary cusp; R, Valsalva sinus of right coronary cusp; PA, pulmonary artery; RV, right ventricle; LV, left ventricle; AV, aortic valve; MV, mitral valve; IVS, interventricular septum

disease. On admission, a murmur suggesting aortic regurgitation (AR) was heard. The transthoracic echocardiography (TTE) with use of color Doppler revealed 3+ AR and 2+ mitral regurgitation (MR), and a vegetation was suspected on the right coronary cusp (RCC) of the aortic valve. There was no abnormality in the structure of the mitral valve and the ascending aorta. The wall motion of the left ventricle was normal.

Fig. 2. The color flow mapping in the Valsalva of the NCC showed similar blood flow velocities on both sides of the flap in diastole (upper), and turbulent flow entering in the pseudolumen in systole (lower).

The TEE with a 5.0 MHz transducer (SSD 870, Aloka Co., Tokyo) (Fig. 1) revealed a thick tear in the Valsalva sinus of the noncoronary cusp (NCC) which slightly moved inside in diastole and a thin tear in the Valsalva sinus of RCC which grossly moved inside in diastole. The shape of the Valsalva of the NCC was symmetrical. The flap of the Valsalva of NCC started at the beginning of the ascending aorta, and continued longitudinally toward the proximal site for 3 cm and transversely for 1.5 cm, ending as a blind pouch. The thickness of the flap

Fig. 3. The color Doppler of the TEE revealed severe AR (upper) and mild MR (lower).

in the Valsalva of the NCC was between 2.6 to 3.3 mm. The Valsalva sinus of the RCC was located slightly to the side of the right ventricle. The right coronary leaflet was dislodged into the side of the left ventricular cavity in diastole and there was no finding of a vegetation on it. The color flow mapping in the Valsalva of the NCC showed similar blood flow velocities on both sides of the flap in diastole, and turbulent flow entering in the pseudolumen in systole (Fig. 2). The color Doppler revealed severe AR and mild MR (Fig. 3). The regurgitant flow of AR went to the mitral orifice, toward the mitral anterior leaflet. There was no abnormality in the mitral valve's structure and the ascending aorta.

A meticulous examination of computed tomography (CT) and magnetic resonance imaging (MRI) showed no abnormality in the Valsalva's structure and the aorta. The aortic angiography (AOG) showed severe AR, mild MR, and double contour in the Valsalva sinus of the NCC (Fig. 4). The AOG from the left anterior oblique view did not reveal a prolapse of the RCC, and it revealed that the right and left coronary arteries were patent.

Two months after the nonpenetrating trauma, surgery was performed. An aortotomy exposed the fact that an intimal laceration extended transversely at the level of the right coronary ostium from the commissure of the RCC and the NCC to the commissure of the RCC and the left coronary cusp (LCC). The intima was completely defected around the right coronary ostium. However, the ostium of the right coronary was patent. A perforation with a diameter of 3 mm was also observed on the RCC, but vegetation was not seen. The base of the RCC was torn

Fig. 4. Aortogram from right anterior oblique view before operation showed double contour in the Valsalva of the NCC.

4 mm and the RCC was swaying (Fig. 5). No abnormality could be found in the NCC or the LCC. Therefore, an aortic valve replacement with aortoplasty was planned. After the coronary cusps were excised, interrupted mattress sutures buttressed with pledgets were passed through the aortic wall from the outside to the inside at the distal level of the intimal laceration, and in addition the sutures were stitched the aortic annulus. A 23 mm prosthetic valve was seated at the aortic ring. As the right coronary ostium was obstructed, an aortocoronary bypass was constructed to the right coronary artery using the great saphenous vein. The assessment with TEE was not performed in the surgery.

After the operation, a TEE examination was performed (Fig. 6). The thick tear which moved slightly to inside in diastole was found exactly in the Valsalva sinus of the NCC. The Valsalva sinus of the RCC was small and there was no tear in it. The function of the artificial valve was normal. The color Doppler showed a trace of AR through the artificial valve and no MR. The clinical course of the patient was good, and he was discharged with NYHA class I.

The pathological microscopic examinations of the cut aortic wall and three cusps revealed atheromatous change in the intima and dilated vessels with leucocytes and foam cells in a part of the media in the aortic wall, and that three cusps, particularly NCC, had myxoid changes.

Fig. 5. Operative view. An intimal laceration extended transversely at the level of the right coronary ostium from the commissure of the RCC and the NCC to the commissure of the RCC and the LCC. The intima was completely defected around the right coronary ostium. A perforation with a diameter of 3 mm was also observed on the RCC. The base of the RCC was torn 4 mm long.

Fig. 6. Transesophageal echocardiogram after operation. These cross-sectional images in diastole disclose that there were a thick tear in the Valsalva sinus of the NCC and an artificial valve with normal function. The single arrow indicates a dissection in the Valsalva sinus of the NCC. The abbreviations are the same as those in Fig. 1; PV, prosthetic valve.

DISCUSSION

It was reported that TTE and color Doppler was useful in diagnosing traumatic AR. However, it showed only the presence of AR, but did not show any abnormality of valve structure excluding valvular prolapse (Kasuya et al. 1993). The cause of traumatic AR is an intimal tear or laceration by which the cuspal attachment to the aortic wall is torn away. It is generally confirmed by operative finding, and not by TTE before operation. This is because the TTE acoustic window is occasionally too poor to allow precise determination due to the interference of bone or lung tissue or obesity in adults. TEE without these interferences provide images superior to TTE. It was reported that by using TEE it was possible to make the differential diagnosis between traumatic rupture and endocarditis as the cause of AR because endocarditis was negated by the absence of valvular vegetation (Habre et al. 1994). In our case, vegetation seemed to be on

the RCC by the TTE finding, however, the presence of vegetation was negated by TEE and the intraoperative finding. By using TTE, it was thought that the prolapse of the RCC was a vegetation. It was reported that TEE was successful in diagnosing a transectional intimal tear on the posterior wall of the ascending aorta, and obtaining TEE in all patients suffering from severe blunt chest trauma was necessary in detecting cardiovascular lesions (Catoire et al. 1994).

There are two mechanisms in the injuries of the aorta and the aortic valve following a nonpenetrating trauma (Eagle and De Sanctis 1992). One is that when the body crashes to a sudden stop, enormous shearing forces work at the point where a highly mobile portion of the aorta joins a fixed segment. The aortic isthmus is the most vulnerable portion, and the supra valvular portion of the ascending aorta is another. The other is that an acute increase in intraaortic pressure generated by the compression of the blood causes injuries. This case had three injuries: a perforation in the RCC, a laceration from the commissure of the RCC and the NCC to the commissure of the RCC and the LCC, and a dissection of the Valsalva sinus of the NCC. In addition, the dissection went toward the proximal side, ending as a blind pouch. Therefore, we postulate that the sudden deceleration led to a rapid rise in aortic pressure and that cardiac diastole, in which the aortic valve was closed and fixed, occurred the instant he hit the ground. The sudden increased force toward the proximal site of the aorta was focused on the Valsalva sinus and the cusps. The deference of TEE findings on the aortic isthmus between the traumatic aortic disruption and the acute aortic dissection was reported. The thickness of the flap associated with the traumatic aortic disruption was significantly greater than that associated with the acute aortic dissection (4.2 ± 0.8 mm vs 2.2 ± 0.7 mm) (Vignon et al. 1995). The thickness of the flap in the Valsalva of the NCC was between 2.6 and 3.3 mm, and the flap in the Valsalva of the RCC was thinner. We thought that the flap in the Valsalva of the RCC was a intimal tear because it was not so thick. On TEE findings, including color Doppler finding, the flap in the Valsalva of the NCC had characteristics of both reported by Vignon et al. (1995). This was because the lesion of our case was in the aortic valve and the Valsalva: the lesion which Vignon and the colleagues reported was in the aortic isthmus. We thought that the TEE finding of this case did not coincide to that of the traumatic aortic disruption due to difference of mechanism in injuries, as mentioned above.

The intraoperative TEE was reported to be useful to assess the cardiac and valvular function (Prêtre and Faidutti 1993). In our case, in spite of the surgeons' meticulous observation, the tear in the Valsalva sinus of the NCC could not be found. This was because when the aortic valve was explored at cardiac arrest under cardiopulmonary bypass, the false lumen in the Valsalva sinus of the NCC had collapsed. If the tear could have been found before the termination of the cardiopulmonary bypass; if intraoperative TEE could have been done, there may have been the possibility of repeatedly looking for and repairing the tear in the

Valsalva sinus of the NCC.

We emphasize that TEE is superior to TTE, CT and MRI in this instance as a noninvasive method of knowing to what degree the aortic valve and the aorta are destroyed in traumatic AR, and that intraoperative TEE or echocardiogram is surely necessary during operation for traumatic cardiac or aortic injuries.

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