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Local Anesthesia for Percutaneous Thoracic Endovascular Aortic Repair

Martijn van Dorp, MD, Martijn Gilbers, MD, Patrick Lauwers, MD, Paul E. Van Schil, MD, PhD, Jeroen M.H. Hendriks, MD, PhD*
Department of Thoracic and Vascular Surgery, Antwerp University Hospital, Edegem (Antwerp), Belgium

Abstract

Background: Thoracic endovascular aortic repair (TEVAR) requires large-bore vascular access due to the considerable diameters of the endoprosthesis and delivery device. The preclose technique preceding endograft delivery has opened the door for an evolved access strategy. In addition, treatment under local anesthesia offers the advantage of optimal neuromonitoring. The goal of this study was to analyze the efficacy and safety of percutaneous TEVAR under local anesthesia.

Methods: All patients undergoing TEVAR in an elective setting at the Antwerp University Hospital between June 2012 and June 2015 were prospectively entered into an endovascular database. This database was queried for demographics, procedural details, and access-related complications. All patients underwent a percutaneous approach with the Perclose Proglide under local anesthesia.

Results: This review identified 34 patients in whom 37 percutaneous TEVAR procedures were completed under local anesthesia. All patients experienced adequate analgesia, and no conversions to general anesthesia were implemented. The mean size of the arteriotomy was 23.8 ± 1.3 French (F). The number of Proglide deployments was 80, with an 8% rate of failure on deployment. There were no conversions to surgical cutdown, and adequate hemostasis was obtained in all procedures. The incidence of postprocedural access-related complications was 3%.

Conclusion: Local anesthesia for percutaneous TEVAR can be performed safely and effectively. The percutaneous approach facilitates local anesthesia, which provides the added benefit of early recognition of neurologic complications while maintaining a low risk of access-related complications despite the need for large-bore vascular access.

Key Words
Thoracic endovascular aortic repair • Percutaneous • Local anesthesia

Introduction

Abbott’s suture-mediated closure devices (SMCDs) have revolutionized the field of thoracic endovascular aortic repair (TEVAR), making preclosing with the Perclose Proglide or the Prostar XL (Abbott Vascular, Redwood City, CA, USA) a crucial step in limiting procedure invasiveness. Several recent articles have described the noninferiority of this percutaneous approach over the classic femoral cutdown for endovascular aortic repair [1-4]. In addition, patients who underwent percutaneous access had shorter hospital stay, reduced procedure-related complications and overall improved patient satisfaction compared with open femoral access.

Several reports have appeared regarding the analysis of risk factors as a determinant of success for percutaneous access [5-8]. However, the size of the arteriotomy can be seen as the major limiting factor for the percutaneous approach [4, 9]. The size of the arteriotomy is determined by the outer diameter (OD) of the vascular sheath or the sheathless delivery device. The use of ultrasound-guided access also significantly decreases the rate of access-related complications [9, 10].
The use of local anesthesia can facilitate the percutaneous approach in TEVAR by further minimalizing procedure invasiveness and allowing early recognition and treatment of neurologic impairment. This neuromonitoring for cerebrovascular accidents and spinal cord ischemia delays the time to treatment for these devastating complications and avoids the need for routine spinal drainage. The goal of this study was to analyze the efficacy and safety of percutaneous TEVAR under local anesthesia.

Materials and Methods

Patient Selection

We performed a prospective analysis of three-year period between June 2012 and June 2015 on all patients who underwent an elective endovascular repair for thoracic aortic disease. Patients were presented the option of treatment under local or general anesthesia. Patients preferring treatment under general anesthesia were excluded. Patient demographics, procedural details, and access-related complications were recorded.

Endovascular exclusion of the thoracic aneurysm, dissection, or endoleak was performed with either the Valiant Captivia thoracic stent graft (Medtronic, Santa Rosa, CA, USA) or the Zenith TX2 endoprosthesis (Cook, Bjaeverskov, Denmark). The delivery device of the Valiant stent graft has an OD range of 22 to 25 French (F, 7.3–8.3 mm), whereas the delivery device for the TX2 endoprosthesis has an OD range of 23 to 26 F (7.6–8.5 mm). Patients were excluded if the OD of the delivery device exceeded the inner diameter of the access site at the common femoral artery.

Vascular access was always obtained under ultrasound guidance. Ultrasound offers the advantage of meticulous localization of the entry site while avoiding calcifications and allows for precise infiltration of the local anesthetic, thereby increasing patient comfort. This study analyzed the data of one operator at the Antwerp University Hospital.

Data Collection

Each puncture site was assessed by clinical examination in the immediate postoperative period. One month postoperatively, patients were seen on an ambulatory basis for clinical assessment and duplex ultrasonography of the groin. Complication documentation and grading were in accordance with literature [11], and a period of one postoperative month was used for documenting all access-related complications.

Preclose Technique under Local Anesthesia

Under ultrasound guidance the ideal entry site was localized. Calcifications are avoided with this method, and the top of the artery is punctured in a monowall fashion, while the common femoral artery (CFA) is punctured well above its bifurcation. Local anesthesia was achieved using infiltration of lidocaine 1% with epinephrine. If necessary, intravenous (IV) sedation with midazolam IV or propofol IV was used to maximize comfort. However, the goal was to maintain the patient fully awake and cooperative. Pain was treated with fentanyl IV bolus or occasionally remifentanil continuous infusion.

We performed a preclose technique of the large-bore vascular access site after ultrasound-guided retrograde puncture of the CFA. Two 6 F Perclose Proglide devices were inserted and deployed in a standard manner after 30° rotation. Upon completion of the procedure, the preformed knots were lubricated with saline and gradually tightened. In the first step, the knots were tied with the guidewire in place to assess accurate hemostasis while maintaining access. In case of inadequate hemostasis, an additional 8 F Angioseal was used. In this case, the Angioseal was only placed after the Perclose Proglide wires were tightened. With this maneuver, the puncture hole was closed as maximally as possible, after which the Angioseal (anchor inside and sponge outside) could adequately cover the residual hole. If adequate hemostasis was achieved immediately, further tightening of the knots was performed upon guidewire removal. A detailed description of the preclose technique has been previously published [12].

Results

From June 2012 to June 2015, 37 TEVAR procedures were performed in 34 patients via a percutaneous approach. The mean (± SD) age was 68.9 ± 11.5, and 29 patients (78%) were male. Twenty-two patients were treated for a thoracic aortic aneurysm, 11 for an aortic dissection, and one for correction of an endoleak. Three patients required a second TEVAR procedure during the study period for the correction of an endoleak, for a total of 37 TEVAR procedures. Patients were followed for a period of one month to document all access-related complications. All patients completed the follow-up period.

All patients were treated in an elective setting under ultrasound-guided local anesthesia. All patients experienced adequate analgesia, and no conversions to general anesthesia were implemented. Local anesthesia has the added benefit of allowing neurologic monitoring for intraoperative cerebrovascular accidents and spinal cord ischemia. This was illustrated in one patient who developed a left-sided hemiparesis during the procedure. Due to early recognition, an immediate stroke protocol was implemented. A cerebral angiogram excluded large emboli.
or a cerebral hemorrhage. The patient was placed on antiplatelet therapy, and an extra dose of heparin was administered. Moreover, controlled hypertension was implemented, after which complete recuperation of the impairment was noted.

The procedural characteristics are summarized in Table 1. Independent delivery and deployment of the Valiant and TX2 endoprostheses are possible without a vascular sheath. Arteriotomy sizes determined by delivery device ODs were 22 F (27%), 24 F (50%), 25 F (16.0%), and 26 F (8%). The Valiant Captivia thoracic stent graft was used in 86% of cases, and the Zenith TX2 endoprosthesis in 14%. The average number of endoprostheses per procedure was 1.6.

A total of 80 Proglide devices were used to pre-close 37 access sites. Adequate placement of tandem Proglide devices is mandatory before delivery device insertion. Six (8%) Proglide devices failed on deployment, resulting in use of an extra Proglide before starting the procedure. Successful hemostasis was obtained in all procedures. There were no conversions to a surgical cutdown. One procedure, a 22 F arteriotomy, required an extra 8 F Angioseal after knotting the Proglide wires to obtain complete hemostasis. Immediate postoperative ultrasound showed excellent results without increased peak-systolic velocity.

In this study, we have also focused on the access-related outcome (Table 2) and documented complications by means of the reporting standards for endovascular aortic aneurysm repair [11]. Only one patient developed an access-related complication by means of a subocclusive stenosis of the CFA requiring a surgical endarterectomy. This was not the patient in whom an additional Angioseal was placed. Clinical evaluation and duplex ultrasonography revealed no incidence of pseudoaneurysm formation at the access site. There were no incidences of significant hematoma or seroma formation, delayed wound healing, wound infection, prolonged pain, or femoral neuropathy during the follow-up period.

### Discussion

Abbott guidelines state that the Perclose Proglide can be applied for an arteriotomy of 5 to 21 F, and tandem Proglide deployment is required in the 8.5 to 21 F range. In TEVAR, large-bore vascular access is required for endoprosthesis delivery and deployment, and all of our patients received an arteriotomy of more than 21 F. However, there were no conversions to a surgical cutdown, and adequate hemostasis was obtained in all procedures. In our previous study, we were successful in preclosing 14 and 16 F arteriotomies with only one Proglide device [13]. While expanding the indications and expectations of the Perclose Proglide, we are able to obtain a relatively low risk of 3% for access-related complications. It seems that one Proglide device can be

<table>
<thead>
<tr>
<th>Table 1. Procedural Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indication</strong></td>
</tr>
<tr>
<td>Aneurysm</td>
</tr>
<tr>
<td>Dissection</td>
</tr>
<tr>
<td>Endoleak</td>
</tr>
<tr>
<td><strong>Endograft type</strong></td>
</tr>
<tr>
<td>Valiant Captivia</td>
</tr>
<tr>
<td>Zenith TX2</td>
</tr>
<tr>
<td><strong>Delivery device outer diameter</strong></td>
</tr>
<tr>
<td>22 Fr</td>
</tr>
<tr>
<td>24 Fr</td>
</tr>
<tr>
<td>25 Fr</td>
</tr>
<tr>
<td>26 Fr</td>
</tr>
<tr>
<td><strong>FR</strong> = French.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2. Access-Related Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outcome</strong></td>
</tr>
<tr>
<td><strong>Inadequate hemostasis</strong></td>
</tr>
<tr>
<td>Femoral cutdown</td>
</tr>
<tr>
<td>Extra angioseal postclosure</td>
</tr>
<tr>
<td><strong>Complication</strong></td>
</tr>
<tr>
<td>Hematoma</td>
</tr>
<tr>
<td>Seroma</td>
</tr>
<tr>
<td>Pseudoaneurysm</td>
</tr>
<tr>
<td>Sensibility disorder</td>
</tr>
<tr>
<td>Delayed wound healing</td>
</tr>
<tr>
<td>Prolonged pain</td>
</tr>
<tr>
<td>Secondary intervention</td>
</tr>
</tbody>
</table>
applied for preclosing an arteriotomy of 5 to 16 F and two Proglide devices can be used to preclose an arteriotomy of 17 to 26 F. Further research is required to assess the efficacy of the Proglide for preclosing larger arteriotomies. However, this may become a futile discussion as endovascular devices are continuously downsized.

The focus of this study was treating patients under local anesthesia. This approach offers many benefits, mostly due to early and direct recognition of complications, especially neurologic impairment at the cerebral or spinal level. We believe that treating patients under local anesthesia is the next step toward decreasing the stroke rate and the incidence of spinal cord ischemia, which remain among the most devastating complications following TEVAR. Several other benefits of local anesthesia have been described. Verhoeven et al. [14] stated that overstretching the arterial system with the delivery device induces discomfort, which alerts the physician of the risk of impending rupture. They also demonstrated that in a subset of TEVAR procedures, the average hospital stay is significantly longer with those operated under general anesthesia compared to local anesthesia [14].

Ultrasound offers the added benefit of precise distribution of the local anesthetic while avoiding calcified lesions during needle puncture, lowering the failure rate for placement of the Proglide system. All patients were perfectly able to complete the less than one-hour procedure without conversion to general anesthesia.

Our results show that percutaneous TEVAR in an elective setting under local anesthesia is effective and safe in a consecutive patient population. The percutaneous approach facilitates local anesthesia, which provides the added benefits of early recognition of neurologic complications and a low risk of access-related complications despite the need for large-bore vascular access.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

**References**


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KIF6 719Arg Genetic Variant and Risk for Thoracic Aortic Dissection

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² Aortic Institute at Yale-New Haven Hospital, Yale University School of Medicine, New Haven, Connecticut, USA

Abstract

Background: Carriers of the 719Arg variant in KIF6, compared with noncarriers, have been reported to be at greater risk for coronary heart disease (CHD) in six prospective studies. Because CHD, thoracic aortic dissection, and nondissection thoracic aortic aneurysm share some risk factors and aspects of pathophysiology, we investigated whether carriers of the 719Arg variant also have greater odds of thoracic aortic dissection or nondissected thoracic aortic aneurysm than noncarriers.

Methods: We genotyped 140 thoracic aortic dissection cases, 497 nondissection thoracic aortic aneurysm cases, and 275 disease-free controls collected in the United States, Hungary, and Greece and investigated the association between KIF6 719Arg carrier status and thoracic aortic dissection, and between KIF6 719Arg carrier status and nondissection thoracic aortic aneurysm, using logistic regression models adjusted for age, sex, hypertension, smoking, and country.

Results: The odds of aortic dissection were two-fold greater in KIF6 719Arg carriers compared with noncarriers (odds ratio (OR) 2.14, 95% confidence interval (CI) 1.18-3.9). To account for the potential of concomitant CHD to confound the association between the KIF6 719Arg and thoracic aortic dissection, we repeated the analysis after removing subjects with concomitant CHD; the estimates for association of KIF6 719Arg carrier status remained essentially the same (OR 2.04, 95% CI 1.11-3.77). In contrast, KIF6 719Arg carrier status was not associated with risk for nondissection thoracic aortic aneurysm.

Conclusions: We observed an association of the KIF6 719Arg genetic variant with thoracic aortic dissection in this multicenter case-control study. This association may enhance our management of patients with thoracic aortic disease.
identify individuals at greater risk for thoracic aortic dissection or nondissection aneurysm before clinical manifestations of the disease would represent a substantial advance in clinical care.

Because coronary heart disease (CHD), thoracic aortic dissection, and thoracic aortic aneurysm share risk factors and some aspects of underlying pathophysiology (vessel wall inflammation, common risk factors of hypertension, familial clustering, and smoking) [7, 8], a genetic variant associated with risk for CHD could be also associated with risk for thoracic aortic dissection or aneurysm.

It has recently been reported that carriers of the 719Arg variant in the kinesin-like protein 6 (encoded by KIF6), compared with noncarriers, were at greater risk for CHD in six prospective studies [9 -15]. These studies included two large population-based studies (ARIC, Atherosclerosis Risk in Communities [9] and CHS, Cardiovascular Health Study [10]) and placebo arms of four landmark randomized clinical trials (CARE, Cholesterol and Recurrent Events [11]; WOSCOPS, West of Scotland Coronary Prevention Study [11]; PROSPER, PROspective Study of Pravastatin in the Elderly at Risk [12]; and WHS, Women’s Health Study [13]). In contrast to these earlier trials, the association of KIF6 719Arg with CHD failed to replicate in the placebo arms of the two more recent randomized clinical trials such as JUPITER (Intervention Trial Evaluating Rosuvastatin) [14] and HPS (Heart Protection Study) [15]. However, some important differences between the earlier and more recent trials may account for inconsistency in the observed association in these studies. For example, patients enrolled in the latter trials had lower low-density lipoprotein-cholesterol (LDL-C) at the time of randomization (in JUPITER, due to enrollment criteria; in HPS, due to the run-in period, which included treatment of placebo arm patients with high-dose simvastatin therapy). The lower LDL-C values in these studies could have ameliorated the risk associated with the deleterious KIF6 719Arg variant [16]. This thesis is supported by a recent meta-regression analysis of 88,535 participants by Ference, which included KIF6 negative studies such as case-control studies of angiographically defined coronary artery disease [17] in the HPS and JUPITER trials. This meta-regression analysis concluded that the KIF6 719Arg variant significantly increases vulnerability of patients to the harmful effect of high LDL-C levels on the risk of CHD.

Given the common aspects of pathogenesis between CHD and thoracic aortic dissection and nondissection thoracic aortic aneurysm, in this case-control study we investigated whether carriers of the CHD risk variant KIF6 719Arg have greater odds for thoracic aortic dissection or nondissection thoracic aortic aneurysm compared with noncarriers.

Methods

Study Subjects

Study subjects included 140 patients with thoracic aortic dissection, 497 patients with nondissecting thoracic aortic aneurysm, and 275 disease-free control subjects collected in the United States (Yale University), Hungary (Semmelweis University), and Greece (Athens Medical Center and Evangelismos Hospital). Characteristics of cases and controls are presented in Table 1. Controls included randomly selected unaffected individuals from Greek and Hungarian study centers (nonpatient visitors and staff) (n = 206) and unaffected spouses of patients with thoracic aortic aneurysm or dissection recruited at Yale University (n = 69). The patients from the United States, Hungary, and Greece (and the respective controls) were analyzed together to increase statistical power, despite the possibility for inherent differences between geographic and ethnic regions. To account for geographic differences, risk estimates were adjusted for the participating center (country). Controls generally shared the same ethnic background, geographic location, and environmental milieu as cases. The physicians caring for the patients verified the aneurysm and/or dissection phenotype from their own radiographic and surgical records, and provided the demographic and clinical data on the patients and controls.

None of the patients had Marfan’s disease or Ehlers-Danlos syndrome. In the dissection group (n = 140), 77 (55%) were ascending dissection patients, and 63 (45%) descending. Among the aneurysms (n = 497), 97 (19.5%) were familial, and 58 (11.7%) had a bicuspid aortic valve. Among the dissections, 25 (17.9%) were familial, and 3 (2.1%) had bicuspid aortic valve. Among the aneurysms, 369 (74.2%) were in the aortic root or ascending aorta, 104 (20.9%) were in the descending aorta, and 24 (4.8%) patients had an aneurysm in both locations.

All subjects provided written informed consent to participate in the study. Both the study and informed consent procedure were approved by the institutional review boards of the participating centers: the Yale University Human Investigation Committee, the Semmelweis University (Budapest, Hungary) Institutional Review Board, the Athens Hospital Center (Athens, Greece) Institutional Review Board, and the Evangelismos General Hospital (Athens, Greece) Institutional Review Board.
Dissection and Aortic Aneurysm Assessment

Patient demographics and phenotypes were ascertained from clinical records available at each enrolling site. Aneurysm and dissection were documented conclusively by echocardiography, computerized tomography (CT), magnetic resonance imaging, or operative findings.

Genotyping and Laboratory Measurements

Genotypes for individual DNA samples were determined by real-time kinetic polymerase chain reaction as described previously [11].

Statistical Analysis

Differences in traditional risk factors between thoracic aortic dissection cases and controls or between nondissection thoracic aortic aneurysm cases and controls were assessed by the Fisher exact test or Wilcoxon rank sum test for discrete and continuous characteristics, respectively. An exact test was used to assess deviation of genotype frequencies from Hardy-Weinberg expectations. The associations of KIF6 Trp719Arg genotype with dissection or nondissection aneurysm were assessed using logistic regression models without or with adjusting for traditional risk factors including age (at the index aneurysm diagnosis or dissection event for cases, at enrollment for controls), sex, current smoker (versus not) status, hypertension (defined as systolic blood pressure >140 mm Hg, diastolic blood pressure >90 mm Hg, a physician's diagnosis of hypertension, or the use of antihypertensive medications), and participating center. The difference in frequency of KIF6 719Arg carrier status among participating centers, while controlling for thoracic aortic disease, was tested for statistical significance with the Cochran-Mantel-Haenszel statistic. Information on concomitant CHD for thoracic aortic dissection cases was available for samples collected in the United States (Yale University). All other probability values are two-sided, and 95% confidence intervals (CIs) are presented. All statistical tests were two-sided and performed using SAS version 9 (SAS Institute Inc., Cary, NC, USA).

Results

Subject Characteristics and Allele Frequencies

The characteristics of cases and controls are presented in Table 1. Traditional risk factors such as older age, hypertension, and male sex were significantly more prevalent among cases. The smoking status differed between thoracic aortic dissection cases and controls but not between nondissection thoracic aortic aneurysm cases and controls.

Among controls, the genotype frequencies were 14.5% for ArgArg, 41.8% for ArgTrp, and 43.6% for TrpTrp; overall, 56.4% of controls carried at least one copy of the KIF6 719Arg variant. The carrier frequency in controls in this genetic study did not differ from that in other large Caucasian populations such as ARIC (52.6%, p = 0.24 for difference between studies). Since the majority of cases were from the United States (Yale University) and the majority of controls were from Hungary and Greece, there was a potential for confounding in this study if the Hungarian and Greek...
controls had lower KIF6 719Arg carrier frequency than the controls from the United States. Therefore, we compared KIF6 719Arg carrier frequency in Hungarian and Greek controls with that in the controls from the United States; we found that the carrier frequency in Hungarian (56.8%) and Greek (58.9%) controls was not lower than in U.S. controls (52.2%). KIF6 719Arg carrier frequency did not differ by participating center (p > 0.5) after controlling for disease status (Table 2). In addition, we adjusted association results for participating centers. The genotype distribution of the KIF6 719Arg variant among controls did not deviate from Hardy-Weinberg equilibrium expectations (p = 0.15).

**Association of KIF6 719Arg with Dissection and Nondissection Aneurysm**

Carriers of the KIF6 719Arg variant, compared with noncarriers, had greater odds of thoracic aortic dissection (odds ratio (OR) 2.14, 95% CI 1.18-3.90) after adjusting for age, sex, hypertension, smoking status, and participating center (Table 3).

Because concomitant CHD could potentially confound the observed association between the KIF6 719Arg variant and thoracic aortic dissection, we reanalyzed the data after excluding cases with known CHD. The magnitude of association between carrier status and thoracic aortic dissection remained largely unchanged after excluding those with CHD (adjusted OR 2.04, 95% CI 1.11-3.77; Table 3).

In contrast to thoracic aortic dissection, nondissection thoracic aortic aneurysm was not significantly associated with KIF6 719Arg carrier status, compared with noncarriers: adjusted OR for KIF6 719Arg carriers was 0.94, 95% CI 0.64-1.38 (Table 4). Although KIF6 719Arg carriers, compared with noncarriers, were at greater risk for descending nondissection aneurysm (unadjusted OR 2.07, 95% CI 1.26-3.40), the association was no longer significant after adjusting for potential confounders (OR 1.73, 95% CI 0.91-3.29; Table 5).

**Discussion**

We found that the KIF6 719Arg variant, which has previously been associated with CHD in six prospective studies, is also associated with thoracic aortic

<table>
<thead>
<tr>
<th>Center</th>
<th>Dissection (n, %)</th>
<th>Nondissection aneurysm (n, %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>Arg/Arg: 14(14.0)</td>
<td>Arg/Arg + Arg/Trp: 32(32.0)</td>
</tr>
<tr>
<td>Hungary</td>
<td>Arg/Trp: 54(54.0)</td>
<td>Arg/Trp: 32(32.0)</td>
</tr>
<tr>
<td>Greece</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 3. Association of KIF6 Trp719Arg with thoracic aortic dissection.

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Case</th>
<th>Control</th>
<th>Unadjusted</th>
<th>Adjusted*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95% CI</td>
<td>p Value</td>
<td>OR</td>
</tr>
<tr>
<td>Including CHD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arg/Arg + Arg/Trp</td>
<td>97</td>
<td>155</td>
<td>1.75</td>
<td>0.0112</td>
</tr>
<tr>
<td>Arg/Trp</td>
<td>77</td>
<td>115</td>
<td>1.87</td>
<td>0.0068</td>
</tr>
<tr>
<td>Arg/Arg</td>
<td>20</td>
<td>40</td>
<td>1.40</td>
<td>0.3077</td>
</tr>
<tr>
<td>Trp/Trp</td>
<td>43</td>
<td>120</td>
<td>ref</td>
<td></td>
</tr>
<tr>
<td>Without CHD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arg/Arg + Arg/Trp</td>
<td>81</td>
<td>155</td>
<td>1.70</td>
<td>0.0234</td>
</tr>
<tr>
<td>Arg/Trp</td>
<td>63</td>
<td>115</td>
<td>1.78</td>
<td>0.0189</td>
</tr>
<tr>
<td>Arg/Arg</td>
<td>18</td>
<td>40</td>
<td>1.46</td>
<td>0.2670</td>
</tr>
<tr>
<td>Trp/Trp</td>
<td>37</td>
<td>120</td>
<td>ref</td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for sex, age, smoking (current versus noncurrent), country, and hypertension.

CHD = congestive heart disease; CI = confidence interval; OR = odds ratio; ref = reference.

### Table 4. Association of KIF6 Trp719Arg with nondissection aneurysm.

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Case</th>
<th>Control</th>
<th>Unadjusted</th>
<th>Adjusted*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95% CI</td>
<td>p Value</td>
<td>OR</td>
</tr>
<tr>
<td>Arg/Arg + Arg/Trp</td>
<td>298</td>
<td>155</td>
<td>1.17</td>
<td>0.3154</td>
</tr>
<tr>
<td>Arg/Trp</td>
<td>217</td>
<td>115</td>
<td>1.14</td>
<td>0.4114</td>
</tr>
<tr>
<td>Arg/Arg</td>
<td>81</td>
<td>40</td>
<td>1.23</td>
<td>0.3632</td>
</tr>
<tr>
<td>Trp/Trp</td>
<td>198</td>
<td>120</td>
<td>ref</td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for sex, age, smoking (current versus noncurrent), hypertension, and participating center.

CI = confidence interval; OR = odds ratio; ref = reference.

### Table 5. Association of KIF6 Trp719Arg with descending and ascending nondissection aneurysm.

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Case</th>
<th>Control</th>
<th>Unadjusted</th>
<th>Adjusted*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95% CI</td>
<td>p Value</td>
<td>OR</td>
</tr>
<tr>
<td>Descending</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arg/Arg + Arg/Trp</td>
<td>75</td>
<td>155</td>
<td>2.07</td>
<td>0.0039</td>
</tr>
<tr>
<td>Arg/Trp</td>
<td>53</td>
<td>115</td>
<td>1.98</td>
<td>0.0110</td>
</tr>
<tr>
<td>Arg/Arg</td>
<td>22</td>
<td>40</td>
<td>2.36</td>
<td>0.0113</td>
</tr>
<tr>
<td>Trp/Trp</td>
<td>28</td>
<td>120</td>
<td>ref</td>
<td></td>
</tr>
<tr>
<td>Ascending</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arg/Arg + Arg/Trp</td>
<td>207</td>
<td>155</td>
<td>1.00</td>
<td>0.9920</td>
</tr>
<tr>
<td>Arg/Trp</td>
<td>153</td>
<td>115</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Arg/Arg</td>
<td>54</td>
<td>40</td>
<td>1.01</td>
<td>1.0</td>
</tr>
<tr>
<td>Trp/Trp</td>
<td>160</td>
<td>120</td>
<td>ref</td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for sex, age, smoking (current versus noncurrent), hypertension and participating center.

CI = confidence interval; OR = odds ratio; ref = reference.
dissection. The ORs approaching 2.00 are quite substantial for association of a single genetic polymorphism with a complex clinical disease. The effect size for this association remained essentially unchanged after adjusting for sex, age, smoking, participating country, and hypertension. This association of the KIF6 719Arg variant with thoracic aortic dissection was not confounded by concomitant CHD, since the effect size was not attenuated and remained statistically significant after removing cases of the thoracic aortic dissection with CHD from the analysis.

The extension of a genetic association from CHD to thoracic aortic disease perhaps is not surprising given that both diseases share risk factors and some aspects of underlying pathophysiology. Another example of such extension is the genetic variant on chromosome 9p21 that has been reported to be associated with both CHD and abdominal aortic aneurysms, as well as intracranial aneurysms [18]. In contrast to the 9p21 genetic variant, KIF6 719Arg was associated with thoracic aortic dissection but not with nondissection thoracic aortic aneurysm. Although we have observed an association with descending, but not ascending, aortic aneurysm in the unadjusted model, effect size was attenuated and became insignificant after adjustment for sex, age, smoking, participating center, and hypertension. Interestingly, in a recent study by LeMaire, investigators observed larger effect size for the association of the genetic polymorphisms in the FBN1 gene with thoracic aortic dissection than for association with nondissection thoracic aortic aneurysm [19].

The KIF6 protein is a member of the kinesin superfamily of proteins that mediate the intracellular transport of organelles, protein complexes, and messenger ribonucleic acids [20]. Typical kinesins are homodimeric molecules consisting of two N-terminal domains (“heads”) that move along microtubules and C-terminal domains (“tails”) that directly interact with the transported cargos or indirectly through adapter molecules. The KIF6 polymorphism replaces a nonpolar residue (Trp) with a basic residue (Arg) near the putative cargo-binding tail domain; thus, it has the potential to alter the affinity for the cargo molecules or possibly to modulate motor activity of the KIF6 protein. These cargos, which have yet to be identified, may play a role in modulating cardiovascular risk and the statin response. In addition, KIF9, the closest homolog of KIF6, interacts with a member of the small guanosine triphosphatase family that promotes endothelial cell sprouting and cytoskeleton reorganization. A similar role for KIF6 in endothelial cell growth and function could provide a link between KIF6 and CHD pathogenesis. KIF6 ribonucleic acid has been shown to be expressed in coronary arteries (see data deposited by King et al [21]), and KIF6 expression is reportedly higher in healthy homozygous carriers of the chromosome 9p21 CHD and aortic dissection risk allele than in noncarriers of the 9p21 risk allele. In terms of any potential mechanisms for predisposition to aortic dissection, these are currently unknown.

This study had some limitations. This genetic study had a case–control design and therefore the deaths from thoracic aortic dissection or nondissection aneurysm rupture were not included in the analysis. The impact of absent lethal cases on the analysis can be substantial [16]; for example, a presumptive preferential mortality of Arg/Arg homozygotes in addition to insufficient power might explain the smaller effect size for association of Arg/Arg genotype with nonlethal thoracic aortic dissection than the association of Arg/Trp heterozygotes. Because there were only a few non-Caucasian participants in this study, we analyzed only Caucasians; therefore, the association of the KIF6 719Arg with thoracic aortic dissection should be further investigated in large replication studies that would include additional ethnic groups. Also, due to the use of spousal controls in the U.S. cohorts, males and females predominated the patient and control groups, respectively. This was addressed by taking potential confounders into account in the adjusted analysis.

Also, there are differences in the underlying characteristics of the aortic aneurysm, aortic dissection, and control groups. While male sex, smoking, and hypertension (Table 1) can fairly be expected to predominate in cardiovascular disease, we do not have detailed information on other risk factors for our control group patients. Differences in underlying group characteristics may have introduced bias in our findings. Also, while inclusion of three geographic patient sources (United States, Hungary, Greece) enhances the generalizability of our findings, an imbalance between origin of patients and controls (most patients from United States, most controls from abroad) may introduce its own bias, although we adjusted the risk estimates for geographic location of the participating clinical center...
We feel this study raises the possibility that KIF6 variant status may serve as a predictor for likelihood of aortic dissection. However, there are several methodologic shortcomings of this initial examination of this topic. Additional work is clearly needed, with more ideal group matching, deeper background information on controls, and greater patient numbers. If our findings are borne out in subsequent prospective studies, assessing KIF6 status could reasonably be added to our armamentarium for predicting aortic dissection risk in patients with thoracic aortic aneurysm.

**Conclusion**

In this multicenter case-control study, the KIF6 719Arg genetic variant was associated with thoracic aortic dissection but not thoracic aortic aneurysm. If the association of the KIF6 719Arg variant with thoracic aortic dissection is further confirmed, this variant could be useful in assessing thoracic aortic dissection risk.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

**Comment on this Article or Ask a Question**

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Iakoubova, O.A. et al. KIF6 Variant and Risk for Aortic Dissection


Abstract

A 57-year-old male with ascending aortic aneurysm, severe aortic regurgitation, and severe mitral regurgitation (MR) underwent ascending aortic replacement and aortic valve replacement. MR in this patient with normal mitral valve morphology was considered secondary to aortic valve incompetency. Consequently, a surgical approach to restore aortic valve function was adopted with successful MR resolution. This case report demonstrates the possibility of reversing early functional mitral regurgitation without surgically approaching the mitral valve.

Key Words

Functional mitral regurgitation • Aortic regurgitation • Aortic aneurysm

Introduction

Functional mitral regurgitation (FMR) is due to incomplete closure of mitral leaflets secondary to pathological left ventricular (LV) dilatation. In this case, aortic valve replacement (AVR) proved sufficient for resolving FMR secondary to aortic insufficiency in a patient without intrinsic anatomic mitral valve abnormalities.

Case Presentation

A 57-year-old male with history of well-controlled hypertension, smoking, and ascending aortic aneurysm was transferred from a community healthcare facility to the cardiac surgery intensive care unit of a tertiary care center after developing chest tightness, worsening dyspnea, orthopnea, and significantly decreased exercise tolerance. Acute myocardial infarction and aortic dissection were ruled out based on a normal electrocardiogram (ECG), normal levels of troponins, and a computed tomography angiogram (CTA) of the chest. The ascending aortic aneurysm was noted again. Approximately 4 months prior to this episode, a chest computed tomography for pneumonia had revealed this ascending aortic aneurysm incidentally. At the time of admission, his medications included furosemide 40 mg four times daily and metoprolol 12.5 mg twice daily. Physical examination revealed an afebrile, male with a body mass index of 28.7, heart rate of 78 per minute, respiratory rate of 20 per minute, blood pressure of 142/62 mm Hg, bibasilar rales, and 3+ diastolic and systolic heart murmurs, which were loudest at the base.

A preoperative transthoracic echocardiogram (TTE) revealed severe aortic regurgitation (AR); mild mitral, tricuspid, and pulmonic regurgitation; moderate left atrial (LA) dilatation, normal LV size with mild concentric hypertrophy, and normal systolic and diastolic function. The estimated ejection fraction was 64%. Based on the TTE, the diameters of the visualized portions of the aorta were 4.0
cm at the sinus of Valsalva, 3.4 cm at the sinotubular junction, 4.7 cm at the ascending aorta, and 3.7 cm at the aortic arch. Chest CTA with contrast showed 4.7-cm dilatation of the mid-ascending aorta (Figure 1A) and atherosclerotic calcifications within the left anterior descending coronary artery (LAD). The aortic arch and descending aorta were of normal calibers. Cardiac catheterization exhibited single vessel coronary artery disease with 80% stenosis in the LAD and severe AR. The LV end diastolic pressure (LVEDP) was estimated to be 30–35 mm Hg.

Overall, this patient’s symptoms were consistent with acute exacerbation of congestive heart failure secondary to aortic root dilatation and concomitant AR. With an aortic diameter greater than 4.5 cm and persistent chest tightness, he was at risk for aortic dissection, and a prompt repair of ascending aortic aneurysm was warranted. Intraoperative transesophageal echocardiogram (TEE) revealed moderate-to-severe mitral regurgitation (MR) with normal morphology of mitral leaflets in addition to previously noted severe AR and ascending aortic aneurysm (Figure 2A). Both the anesthesia and surgical teams were very concerned about the severe MR, and we strongly considered approaching the valve surgically, in addition to the other necessary procedures. The patient underwent ascending aortic replacement, AVR, and coronary artery bypass grafting to the left anterior descending artery with left internal mammary artery. The mitral valve was not approached. Intraoperative TEE showed immediate improvement of MR following the procedure (Figure 2B).

After surgery, the patient had an uneventful recovery and was discharged home on postoperative day 5. Two months later, chest CTA revealed normal caliber and contour of ascending aorta and aortic arch without any evidence of complication (Figure 1B).

Discussion

FMR is the most common etiology of MR in the United States [1]. Contrary to MR caused by diseases of valvular tissue such as flail leaflet and infective endocarditis, FMR is due to incomplete closure of mitral leaflets secondary to pathological LV dilatation. Chronic volume overload (as from AR) eventually overwhelms LV compliance and induces adverse ventricular remodeling, which gives rise to LV enlargement and dilatation of the mitral annulus. FMR develops when the mitral leaflets are no longer able to coapt. Chronic FMR may lead to further adverse remodeling of the heart including LA dilatation. When the compliance of LA exceeds a certain limit, LA pressure starts to rise, eventually resulting in pulmonary hypertension [2-7].

In this case, we surmised that restoring aortic valve competency (AVR) in the setting of normal systolic and diastolic functions and a morphologically normal mitral valve was likely to resolve FMR by returning left ventricular end diastolic volume (LVEDV) and LVEDP to normal levels. MR was immediately and
dramatically improved in this patient following AVR. We were surprised and gratified to see immediate MR resolution upon elimination of the severe AR. In retrospect, our decision to avoid mitral valve surgery avoided unnecessary prolongation of an already major surgical procedure.

Double valve replacement has a reported mortality rate of 5.6–17.5%, which is much higher than that of AVR alone (2.0–7.8%) [6]. Thus, it is critical to evaluate the contribution of each individual lesion in order to choose the surgical treatment with the most favorable outcome. MR has long been considered a marker of impaired LV performance in aortic stenosis (AS) [8]. Mild-to-severe FMR in AS can be resolved with AVR alone, probably due to improved LV function [9, 10]. However, the efficacy and limitations of AVR for FMR secondary to AR have not been studied. Here, we report a successful reversal of functional MR after AVR. This case report highlights the relationship between valve function and ventricular geometry.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Figure 2. Panel A, B and C: Preoperative transthoracic echocardiogram showing moderate to severe mitral regurgitation. Panel D: Postoperative transthoracic echocardiogram showing mitral competency.
References


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Abstract
We report the cases of two patients who presented with screw misplacement following spinal surgery. Both benefited from unusual vascular surgical management with removal of the material and injection of biological glue facing the injury, with uneventful postoperative courses.

Key Words
Aortic injury • Spinal surgery • Biological glue

Introduction
Placing spinal instrumentation in contact with major vessels is a well-known risk for vascular damage in spinal surgery. This iatrogenic pathology has been described in the literature but is rare. Its treatment is challenging, and surgical techniques including endovascular and open surgery have been described.

We report two patients who underwent spinal instrumentation for spinal pathology. Postoperative imagery revealed screw misplacement in the column extending into the thoracic aorta. The patients benefited from unusual vascular surgical management, with removal of the orthopedic material and injection of biological glue facing the injury. The postoperative courses were uneventful in both cases. The 6-month follow-up computed tomography (CT) scans showed normal anatomy of the spine and thoracic aorta secured by the biological glue.

Unusual Management of Thoracic Aortic Injury After Spinal Instrumentation: Just Glue It!

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Case Presentation

Patient 1

The first patient was a 24-year-old female who presented with a traumatic D3-D4-D5 vertebral fracture after a traffic accident. Urgent repair through a posterior approach was performed in another hospital, with osteosynthesis of D1 to D7.

A radiologic control with CT and angio-magnetic resonance imaging (Figure 1A and B) was performed and demonstrated perforation of two screws through the posterior wall at D6-D7 in contact with the aortic arch. There was no sign of leakage, bleeding, or pseudoaneurysm. The patient was totally asymptomatic. On account of the contact of two screws with the esophagus on MRI, the patient underwent a gastroscopy that revealed two mucosal protrusions in the esophagus with the erosion of the top of one of the protrusions.

After multidisciplinary discussion, we felt it was indicated to remove the prosthetic materials, given the digestive preperforation, to avoid mediastinitis. During surgery, percutaneous access to the femoral artery was prepared using a 6-French introducer to prepare for implantation of a covered thoracic aorta endograft in the event of acute bleeding of the aorta after screw removal. No bleeding occurred when the screws were removed. We decided to secure the aortic wall by injecting BioGlue (CryoLife, Inc., Kennesaw, GA, USA)
into the spinal holes in contact with the aortic wall to prevent late bleeding. No new instrumentation was placed because of good spinal stabilization.

The patient had an uneventful postoperative course. CT performed 6 months postoperatively showed the persistence of lacunar images in the aortic arch without any complications (Figure 2).

**Patient 2**

The second patient was a 64-year-old female who underwent surgical treatment for streptococcal spondylodiscitis of D5-D6 with spinal cord compression. The postoperative course was characterized by a neurologic deterioration with right-sided motor deficit requiring a radiologic examination. MRI confirmed spinal cord compression with a slip of the posterior wall of D5. A new surgical approach with an osteosynthesis and arthrodesis in D3 to D7 was performed, and evolution was satisfactory.

The control CT in the postoperative setting indicated misplacement of one screw in the left D4. Contact with the descending thoracic aorta was confirmed by CT scan, without any sign of leakage, hemorrhage, or pseudoaneurysm (Figure 3A).

We felt that there was indication for revision and material removal to minimize the risk of erosion, pseudoaneurysm, or bleeding. This procedure was performed 10 months later because of pulmonary emboli and anticoagulation treatment. We used a similar surgical approach to that in the first case, with percutaneous access established in case endovascular control of aortic bleeding was required. The transpedicular screw at the D4 level was removed, and BioGlue was injected into the holes in contact with the aortic wall. No new instrumentation was placed because of good spinal stabilization. Despite anticoagulation, no intraoperative vascular complication occurred, and the patient had an uneventful postoperative course and follow-up.

A postoperative CT scan was performed the day after the procedure and showed “compression” of the aortic wall at the level of the removed screw. This simply reflected excess glue containing some air bubbles, and this disappeared on the 6-month follow-up CT (Figure 3B).

**Discussion**

Thoracic aortic injury is an uncommon but recognized complication after posterior spinal surgery related to the misplacement of fixation hardware in the column. Different treatment approaches have been reported in the literature. If the injury is discovered in the acute setting, the aorta is repaired by means of an endovascular stent graft or open repair where the misaligned
hardware is removed and replaced [4, 5]. For asymptomatic patients, a multidisciplinary approach with revision of spinal fixation and aorta repair has been reported, as has primary repair of the vascular injury [1].

Traumatic aortic rupture is a life-threatening injury associated with high operative mortality of 32% and a paraplegia rate of 36.4% in open surgery [2]. However, the mortality rate associated with endovascular repair is significantly lower (16.6%), with a paraplegia rate of 2.7% [3]. Since first described by Parodi [4] and Volodos [5] in 1991, endografting aortic lesions has clearly transformed the outcomes for these critical patients.

The midterm results of thoracic aorta endografting for aortic rupture published by Astarci [6] were excellent at 48 months follow-up. For this reason, we prepared our two patients to receive an endograft if aortic rupture occurred during screw retrieval. The patients remained stable, without any acute bleeding, and did not require an endograft. However, to avoid late bleeding of the aorta, we decided to use biological glue to secure the assumed aortic wall injury due to the screws. A more aggressive approach is possible by preemptively inserting the endograft prior to screw removal. This is a very secure method; however, it may be unnecessary to cover at least 10 cm of thoracic aorta, with the inherent risk of covering the spinal arterial supply and inducing paraplegia by spinal cord ischemia.

In the first published case of aortic endografting simultaneous with screw retrieval by Minor [7], the author rejected the option of initial screw removal due to the associated risk of massive bleeding. The author also rejected graft deployment followed by screw removal because of the risk of graft perforation by the tip of the screw. He finally decided to deploy the graft simultaneously with screw retrieval.

Acute bleeding at the time of screw removal is almost impossible unless there is a huge pseudoaneurysm of the thoracic aorta at the level of the screw or aortic bleeding into the pleura as reported by Kokotsakis [8]. We describe herein the feasibility of a safe gluing technique that can offer a less aggressive alternative in selected patients, without placement of a preventive thoracic endograft.

**Conclusion**

There is a rare but potentially morbid complication of spinal instrumentation surgery at the thoracic level. The endovascular approach allows minimally invasive treatment of the injured thoracic aorta avoiding thoracotomy and cross-clamping. However, preventive endograft deployment seems to be excessive, and as in our cases, screw removal can take place without acute bleeding.

Toour knowledge, the use of BioGlue through screw holes to secure the aortic wall after spinal screw removal has never been described. Based on the outcomes of these two patients, we proposed that this treatment is a good alternative to endograft deployment in selected cases.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

**References**


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Abstract
Epioaortic ultrasound is an imaging modality that is commonly used to evaluate the ascending aorta for atheroma and other mural lesions during elective cardiac surgery. Its use in contained aortic rupture has not been established. We present a case of thoracic trauma with contained pseudoaneurysm of the ascending aorta. At operation, the precise location of the aortic tear could not be identified by visual inspection, manual palpation, or transesophageal echocardiography. Epioaortic ultrasound was employed and the aortic defect was identified and successfully repaired. This intraoperative imaging modality may play an increasing role in the identification of aortic pathology when visual inspection and other intraoperative imaging is insufficient.

Case Presentation
The patient was a 33-year-old Hispanic male. He had been a restrained passenger of a motor vehicle traveling approximately 60 miles per hour when a liquid-containing metal canister was dislodged from a truck in front of his vehicle and came through the front windshield, striking the patient in the face and chest. At the time of presentation, the patient was noted to have a mandibular fracture, sternal fracture, and multiple rib fractures. Due to the mechanism of injury, the patient underwent computed tomography (CT) angiography of the chest with 3-dimensional reconstruction which was significant for a 1.4 × 0.7-cm pseudoaneurysm above the sinotubular junction, projecting medially toward the lesser curve of the aortic arch. The pseudoaneurysm demonstrated...
no evidence of rupture, and the patient remained hemodynamically stable.

The patient was taken electively to the operating room for definitive repair. Intraoperatively, TEE was used to visualize the pseudoaneurysm sac but the exact location of the tear in the aorta could not be identified (Figure 1). Due to the uncertainty of the location of the aortic defect, the necessary distal extent of our repair could not be determined. In addition, we were prevented from cannulating the aorta for cardiopulmonary bypass because it was unclear whether we would need to initiate deep hypothermic circulatory arrest and proceed with a more extensive arch repair.

Direct epiaortic ultrasonography was used to interrogate the lesion and identified a 1.6 × 1.0-cm pseudoaneurysm along the medial aspect of the ascending aorta, above the sinotubular junction, and between the aorta and main pulmonary artery (Figure 2). A full-thickness, oval-shaped defect was identified that traversed one-third of the aortic circumference. At this time, the decision was made to replace the ascending aorta with a supracoronary 24-mm Hemashield tube graft. The patient did not require vasopressor or inotropic support and was extubated in the immediate postoperative period. He was discharged on postoperative day four. Surveillance CT angiography performed at one-year follow-up demonstrated an intact graft without pseudoaneurysm.

**Discussion**

TEE is the modality of choice to detect intravascular and transmural defects of the ascending aorta, specifically ascending aortic dissection and aortic pseudoaneurysm [3]. However, views of the distal ascending aorta and proximal arch are limited due to the interposition of the trachea and right main bronchus, with the air-filled structures causing a ‘blind spot’ when using TEE [4]. When one of these air-filled structures is superimposed, identification of the precise location of vascular pathology may be difficult. When TEE fails to enable an accurate diagnosis that may affect surgical decision making, immediate availability of other imaging modalities may be useful [3-5]. In this case, the ascending aortic defect could not be identified.

![Figure 1](image1.png)  **Figure 1.** Transesophageal echocardiogram demonstrating the ascending aortic pseudoaneurysm. LVOT = left ventricular outflow tract; AA = ascending aorta; PSA = pseudoaneurysm.

![Figure 2](image2.png)  **Figure 2.** Epiaortic ultrasound demonstrating 1.6 × 1.0-cm pseudoaneurysm along the medial side of the ascending aorta. Panel A. Ultrasound image alone. Panel B. Ultrasound with color Doppler. AA = ascending aorta; PSA = pseudoaneurysm.
by inspection and digital palpation as it was directly covered by the main pulmonary artery. The mediastinum had an otherwise normal appearance, and TEE did not show the exact location of the aortic tear due to the interposed airway. Epiaortic ultrasound is a readily available and inexpensive imaging modality that does not increase morbidity for the patient. Epiaortic ultrasound provided rapid intraoperative identification of the aortic lesion that was otherwise missed using direct inspection and TEE. This method of rapid intraoperative assessment of the ascending aorta may have further use in elective and emergent settings when conventional imaging fails.

**Conflict of Interest**

The authors have no conflicts of interest to disclose.

**References**


Abstract
A tree fell on the back of a 77-year-old male. A postmortem computed tomographic pan scan revealed systemic air embolism, multiple rib fractures with a penetrating injury to the aorta, pneumohemothorax, and air in the aorta. A massive amount of air entered the site of a penetrating injury of the aorta. This unique case adds one more cause to the list of documented etiologies of air in the aorta.

Key words
Penetrating injury of aorta • Rib fracture • Pneumothorax

Introduction
Migration of air into the aorta is rare. We report a rare case of a patient in whom a penetrating aortic injury caused by broken ribs, led to the migration of air into the aorta from a pneumothorax.

Case Presentation
A tree fell on the back of a 77-year-old male when he was cutting down trees. When emergency technicians (EMTs) reached him, he was in a state of cardiopulmonary arrest. The initial rhythm was asystole. Tracheal intubation, the insertion of a chest drain into the left thorax and the infusion of fluid and adrenaline were performed by the EMTs and an emergency physician. After his transfer to Shizuoka Hospital, Juntendo University, a monitor revealed that he remained asystolic in a state of cardiopulmonary arrest. The findings of a blood gas analysis (FiO₂ 1.0) on arrival were pH: 6.671, PCO₂: 44.6 mmHg, PO₂: 48.7 mmHg, HCO₃⁻: 4.9 mmol/l, base excess: -26.1 mmol/l and hemoglobin: 1.9 g/dl. Additional right thoracostomy was performed but a return of spontaneous circulation was not obtained. A postmortem computed tomographic (CT) pan scan performed 90 minutes after the patient’s collapse revealed systemic air embolism, multiple rib fractures with a penetrating injury to the aorta, lung contusion, pneumohemothorax, pneumomediastinum, air in the aorta and a lumbar fracture (Figure 1). The cause of death was unstable circulation due to massive hemorrhage from an aortic injury and air embolism, and respiratory failure due to lung contusion and pneumohemothorax.

Discussion
The main causes of migration of air into the aorta...
are iatrogenic, these include cardiac or aortic surgery, arterial endovascular management or transthoracic lung biopsy [1-5]. In rare cases, the suicidal connection of a peripheral venous catheter with oxygen gas has resulted in the migration of air into aorta [6]. In patients with decompression sickness, CT can demonstrate intra-arterial gas [7]. In the present case, a massive amount of air ventilated with positive pressure in a patient with pneumothorax, entered the site of a penetrating injury of the aorta (caused by rib fractures), which resulted in the aorta being filled with air. To the best of our knowledge, this is the first case to describe the introduction of air into the aorta by such a mechanism. The air in the aorta may have been the result of a massive hemorrhage and the introduction of air after the death of the patient as a result of no circulation and the presence of a hole in the aorta. However, distinguishing premortem from postmortem phenomena is difficult. Lung trauma involving laceration of air passages, lung parenchyma, and blood vessels may result in direct communication of these structures. Systemic air or gas embolism occurs when air or gas enters the pulmonary venous system as a result of a positive gradient caused by a low pulmonary venous pressure. Dedouit et al. [8] showed that a small amount of air in the ascending aorta due to a paradoxical embolism through patent foramen ovale or across the pulmonary capillary bed created tension pneumothorax. The present case also had lung contusion, and the gas in the aorta may have been produced by the paradoxical embolism. However, the amount of gas in the aorta in our case was massive, and we noted no gas at all in the left side of the heart. These findings therefore make it unlikely that the gas was produced by a paradoxical embolism.

This unique case adds one more cause to the list of documented etiologies of air in the aorta. The induction of microbubbles by arterial endovascular management, which can be detected by ultrasound, can be asymptomatic [9]. However, in one report, the detection of the air in the aorta by radiological studies indicated systemic air embolization, which tended to be associated with a poor outcome, similar to that which was observed in our case [5].

In the present case, a massive amount of air ventilated with positive pressure in a patient with pneumothorax, entered the site of a penetrating injury of the aorta (caused by rib fractures), which resulted in the aorta being filled with air. This unique case adds one more cause to the list of documented etiologies of air in the aorta.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

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Migration of Air with a Penetrating Aortic Injury
References


Because he underwent regular medical check-ups and his history did not indicate any cardiac problems, including murmur, I believe that the likelihood of an ASD is low. To our knowledge, no previous papers have reported such a large amount of aortic air due to lung injury, with high positive pressure air force.

3. How often are postmortem computed tomography (CT) scans performed in Japan? (They have shown that aortic dissection accounts for up to 7.5% of all lethal out-of-hospital cardiac arrests.)

The frequency of postmortem CT varies by medical institute in Japan; however, our hospital tries to perform whole-body CT for all patients who die of sudden cardiac arrest.

EDITOR’S QUESTIONS

1. How do you know that this patient did not simply exsanguinate, with the aortic air entering postmortem?

In my experience, including previous successful cases [1] (all others ultimately died and were not reported on), patients in whom the aorta has been penetrated by broken ribs due to blunt trauma do not immediately enter cardiac arrest. Witnesses saw the patient in the present case collapse, and he was already in cardiac arrest when the emergency technicians checked him. I, therefore, hypothesize that this patient suffered from some other trauma that resulted in immediate cardiac arrest.

2. How about pericardial embolism through an atrial septal defect (ASD)? How about penetrating lung injury, which under positive pressure forces air into the pulmonary veins and subsequently into the aorta, often with fatal results (diagram in Elefteriades JA, et al. House Officer’s Guide to ICU Care, 3rd Edition, 2013)? Please comment.

Because he underwent regular medical check-ups and his history did not indicate any cardiac problems, including murmur, I believe that the likelihood of an ASD is low. To our knowledge, no previous papers have reported such a large amount of aortic air due to lung injury, with high positive pressure air force.

3. How often are postmortem computed tomography (CT) scans performed in Japan? (They have shown that aortic dissection accounts for up to 7.5% of all lethal out-of-hospital cardiac arrests.)

The frequency of postmortem CT varies by medical institute in Japan; however, our hospital tries to perform whole-body CT for all patients who die of sudden cardiac arrest.

References

Is the Sac Waiting to Rupture? Sinus of Valsalva Aneurysm

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Abstract
Completely asymptomatic sinus of Valsalva aneurysms are rare entities, and there is no consensus regarding their management. We present the case of a patient who underwent atrial septal defect device closure at 5 years of age and was lost to follow-up, then presented 6 years later with unruptured sinus of Valsalva aneurysm and was closely followed. The aneurysm eventually ruptured and was successfully operated on with good outcomes.

Key Words
Unruptured sinus of Valsalva aneurysm • Atrial septal defect • Amplatzer device.

Case Presentation
An asymptomatic 11-year-old male presented for a routine evaluation. At the age of 5 years, he had undergone transcatheter closure (TCC) with a 16-mm Amplatzer Septal Occluder (ASO) (St. Jude Medical, St. Paul, MN, USA) for an atrial septal defect (ASD) with adequate rims except for a deficient aortic rim. Five months after the procedure, he underwent a clinical and echocardiographic follow-up that revealed normalization of the right heart chamber dimensions, ASO in situ straddling the aorta due to deficient aortic rim, and no residual interatrial shunting. Since then, the patient had felt well and had not sought any follow-up medical services.

Physical examination was unremarkable. Electrocardiogram (ECG) showed sinus rhythm with incomplete right bundle branch block, which was also seen in earlier ECGs. The PR interval was 0.14 s. Chest X-ray was normal. ECG showed a large saccular unruptured sinus of Valsalva aneurysm (SOVA) from the non-coronary sinus (NCS) of the aorta and protruding into the right atrium (RA), measuring 1.5 cm at the aortic end and 2 cm at the RA end, without any other associated lesions and not causing any obstruction (Figure 1A). The ASO was seen in situ without residual interatrial shunting. There was good distance between the two defects, the right atrial site of SOVA being lower than the anteroinferior margin of the ASD rim. The left ventricular function was normal. Computerized tomography (CT) scan was used to better profile the aneurysm (Figure 1B). Due to his asymptomatic status and lack of compressive features, such as conduction blocks, valvular regurgitation, or obstruction, the patient was asked to follow-up every 3 months. On follow-up, the size of the aneurysm was unchanged as assessed by ECG. After about 1 year, the patient developed acute gastroenteritis with severe frequent bouts of vomiting and cough with breathlessness. On physical examination there was a grade 3/6 continuous murmur heard best in the 3rd/4th parasternal area with signs of right heart failure. ECG showed the rupture of SOVA with the aneurysm draining from NCS to RA causing severe RA and right ventricular volume overload and pericardial effusion (Figure 1C). Because TCC of this aneurysm was not feasible due to the large aortic end of the sac measuring 1.5 cm, the patient was...
sent for surgery. The aorta was opened obliquely above the NCS. The ruptured SOVA sac was excised and closed with Sauvage filamentous dacron and pericardial patch on cardiopulmonary bypass (Figure 1D).

Discussion

SOVA is a rare cardiac anomaly that may be either acquired or congenital. A congenital lack of continuity between the aortic media and annulus fibrosis may initiate aneurysm formation. Less frequently, infections or degenerative processes may affect the aortic wall. SOVA is associated with multiple lesions, most commonly a ventricular septal defect. Rarely, SOVA may be associated with an ASD and staged TCC of both defects has been described [1]. SOVA may rupture into an extra- or intracardiac location [1]. Unruptured SOVA may cause right ventricular outflow tract obstruction, infective endocarditis, malignant arrhythmias, or myocardial ischemia/infarction due to severe distortion of coronary ostia or compression of the coronary trunks and conduction blocks [2]. The natural history of untreated, ruptured SOVA has a mean survival period of 3.9 years [3], so early surgery is indicated in such cases. Surgical intervention is also necessary in unruptured SOVA when the patient is symptomatic or there is evidence of obstruction or compression. Optimal management of asymptomatic, unruptured aneurysm is uncertain as no precise natural history is currently available. It is tempting to attribute the development of SOVA to the TCC of ASD procedure in our patient, especially because the aortic rim of the ASD was deficient. Intraoperatively, an indentation of ASO was noticed on the inner aspect of the aorta. However, the base of the aneurysm was away from the inferior rim of the device. It is possible that the mechanical force of the ASO may have distorted the aortic root, resulting in the development of the SOVA. In our patient, it might be attributable to a congenital weakness at the junction of the aortic media and annulus fibrosis. Indeed, device traction on the NCS has been hypothesized to cause aortic regurgitation following TCC of ASD [4], although this has been debated [5].

Vural et al. [5] described a detailed approach to SOVA, based on the clinical picture and the echocardiographic findings. In asymptomatic cases, surgery is indicated if the size of the aneurysm is larger than 50% of the average size of the other two normal Valsalva sinuses or is increasing in consecutive ECG examinations. This approach, however, is not based on any background data. The rationale for not surgically intervening in asymptomatic SOVA is the morbidity and mortality associated with the procedure itself. On long-term follow-up, a number of surgically operated patients require re-intervention for recurrence of SOVA rupture and aortic valve replacement for significant aortic valve regurgitation [6].

On the other hand, asymptomatic SOVA may expand, causing more severe symptoms and requiring more extensive corrective procedures in the future. Recent surgical advancements have significantly reduced mortality associated with the procedure. A “wait and watch” asymptomatic SOVA policy is therefore questioned by some authors [7]. Nevertheless, our case illustrates that conservatively managed asymptomatic SOVA needs careful follow-up with high anticipation for the diagnosis of rupture and prompt treatment.
Conflict of Interest

The authors have no conflict of interest relevant to this publication.

References


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Abstract

We describe the case of a 61-year-old male with a giant aortic root aneurysm associated with chronic aortic Type A dissection. The patient had been operated on 16 years before due to aortic annuloectasia with mechanical valve replacement. The patient underwent revision aortic surgery with a Bentall–De Bono operation with Svensson modification, using a #21 On-X Valsalva mechanical valve conduit. The postoperative course was uneventful.

Key Words
Aortic dissection • Aortic aneurysm • Annuloectasia

Introduction

Herein we illustrate the surgical management of a giant aortic root aneurysm with aortic dissection in a patient that had been operated on several years before due to aortic annuloectasia with an aortic valve replacement.

Case Presentation

A 61-year-old male patient had undergone aortic valve replacement due to aortic annuloectasia using a #23 St. Jude Medical mechanical valve prosthesis. A 4.5-cm aortic root aneurysm was not removed, probably because the aortic valve was trileaflet. By 2007, the aortic root had grown to 7 cm, but surgery was not performed at another institution due to the high risk, so the patient stopped follow-up.

In 2015, the patient was admitted to our hospital with thoracic pain. Computed tomography revealed a 11.3 × 10.7 × 10.1-cm aortic root aneurysm with a Stanford Type A dissection (Figures 1 and 2). The patient underwent aortic root surgery (Figure 3) with removal of the previous prosthesis and Bentall procedure using a #21 On-X Valsalva mechanical valve conduit. The distant left main coronary ostium was easily reattached to the ascending conduit via a 10-mm interposition graft (Svensson modification [1]). The right coronary artery button was easily mobilized and conventionally reattached (Figure 4).

Pathology showed a chronic dissection with an intimal tear close to the origin of the left main coronary ostium. Histology showed subendothelial fibrosis with calcified areas due to atherosclerosis.

The postoperative course was uneventful, and the patient was discharged home on postoperative day 8.
Discussion

Aortic root replacement should be considered at the time of aortic valve replacement in young patients with moderate enlargement of the ascending aorta and aortic annuloectasia, regardless of aortic valve phenotype.
Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Reference

Martijn van Dorp et al.: Local Anesthesia for Percutaneous Thoracic Endovascular Aortic Repair

In some cases, diseases of the aorta, the body’s main artery, have in recent years become treatable by “Thoracic Endovascular Aortic Repair”, or TEVAR. In this technique, the diseased vessel is not surgically replaced, but rather stabilized from the inside by a stent graft prosthesis that is inserted via a vessel in the groin. Usually, this vessel is surgically exposed to introduce the stent graft. In their study, Martijn van Dorp and colleagues investigated a technique that allows inserting the stent graft through a puncture through the skin into the groin vessel. They report their experience with 34 patients in whom they used this device for stent grafts whose introducing sheath were larger than previously recommended. With this puncture technique, they were able to perform these procedures under local anesthesia. With local anesthesia, some complications of these procedures, e.g. a stroke of the brain, can be recognized earlier. Furthermore, patients tend to be more comfortable and tend to be discharged home earlier after procedures under local anesthesia. In this study, the device mostly worked well, and there were no patients who needed surgical exposure of the vessel or general anesthesia. Only one patient had a complication concerning the access site vessel. The authors therefore conclude that with their puncture technique, this type of procedure can be performed under local anesthesia even when using larger diameter stent grafts.

Iakoubova et al.: “KIF6 719Arg Genetic Variant and Risk for Thoracic Aortic Dissection”

“KIF6” is the name of a gene that occurs in different variants in the human genome. One of the variants called “719Arg” previously has been associated with a high risk of developing coronary heart disease, which can cause a heart attack. In their study, Iakoubova and colleagues studied whether this variant of the gene might be associated with a higher risk of developing an aortic aneurysm or sustaining an aortic dissection. In aortic aneurysm, the aorta, the main artery that arises from the heart and distributes oxygenated blood in the body, dilates and can eventually rupture. In aortic dissection, a tear occurs in the vessel wall of the enlarged aorta through which blood enters in the vessel wall, creating a disruption of vessel layers. Both aneurysm and dissection are potentially life threatening diseases that often warrant major surgery. Since the disease usually presents with few warning signs, identifying risk factors is important to permit identification of patients at risk.

The authors tested 912 patients’ genes to determine which variant they had. These findings were then analyzed as to whether patients were being healthy or had aortic aneurysm or dissection. The results showed that the odds of aortic dissection were about two-fold higher in patients who carried the “719Arg” variant of the gene. The odds of having thoracic aortic aneurysm were not increased. The KIF6 gene contains the code for a group of molecules that are responsible for transportation within...
The mechanism by which the variant might cause a predisposition for aortic dissection is unclear. However, this study does not prove yet that the studied variant increases the risk of aortic dissection, because it does have some limitations. For studies of this type, the study group is quite small. Further trials are necessary to find out if the “KIF6 719Arg”-variant is indeed associated with aortic dissection and if testing for KIF6 is a helpful tool to identify patients at risk for aortic aneurysm and dissection.

Case reports

Ahmad Zeeshan et al.: Immediate Improvement in Severe Mitral Regurgitation After Aortic Valve Replacement for Severe Aortic Insufficiency

In patients with regurgitation (backward leakage) of the aortic valve, which serves as a gatekeeper between the heart and the aorta, the body’s main artery, leakage through the aortic valve can cause another valve in the heart, the mitral valve, to leak as well. When planning surgery in patients with leakage of both valves, it is a matter of judgement if only the aortic valve, or both valves need to be repaired or replaced. Operating on both valves might significantly increase the risk of surgery. Ahmad Zeeshan and colleagues describe a case of a patient who had a leaking aortic valve and secondary mitral valve leakage who after isolated aortic valve replacement showed full and immediate resolution of his mitral valve leakage. Correcting the leakage of the aortic valve “took the load off” the mitral valve, restoring its competency.

Yannick Deswysen et al.: Unusual Management of Thoracic Aortic Injury After Spinal Instrumentation: Just Glue It!

The aorta, the body’s main artery runs very close to the spine. Injury to the aorta is therefore a rare but possible complication of spinal surgery. In case of such injury, aortic repair with open replacement surgery or using a stent graft that is placed into the aorta via the groin vessels might be necessary. Yannick Deswysen and colleagues describe two cases of misplaced screws used for spinal surgery inadvertently penetrating the aorta, but without causing bleeding. Both cases were handled by removing the screw and gluing the injury with biological glue. Both patients recovered uneventfully. The suggested approach could significantly reduce the extent of aortic repair in asymptomatic and uncomplicated cases of aortic injury after spinal surgery.

Michelle Eddins et al.: Intraoperative Epiaortic Ultrasound for Traumatic Pseudoaneurysm of the Ascending Aorta

Ultrasound is a technique that uses sound waves that are echoed by body structures to create images of these structures. Usually, the ultrasound probe is placed on the skin or in the esophagus behind the heart. Michelle Eddins and colleagues present the case of a patient who suffered traumatic injury to his aorta, the body’s main artery in a car accident. The injury needed to be surgically repaired, but proved difficult to visualize. During surgery, the surgical team placed a sterile ultrasound probe on the aorta to produce images of the exact anatomy of the injury. This technique, called “epiaortic ultrasound”, helped them to plan the surgical procedure.

Youchi Yanagawa et al.: The Migration of Air into the Aorta from A Pneumothorax in a Patient with a Penetrating Injury of the Aorta

Youchi Yanagawa and colleagues describe the case of a man who died when a tree fell on his back. They performed a CT scan (a 3D X-ray study) of the man’s body that showed that several ribs had been broken, with one of them penetrating into the aorta. The unexpected finding was that air had not only entered through the man’s rib cage, but had also traversed into the aorta. The authors hypothesize that the patient did not die only from exsanguination, but also due to the air that entered the aorta.

Ankur Phatarpekar et al.: “Is the Sac Waiting to Rupture? Sinus of Valsalva Aneurysm”

The Sinuses of Valsalva are normal bulges of the wall of the aorta, the body’s main artery, at the level of the aortic valve, which serves as a gate between the heart and the aorta. Ankur Phatarpekar and colleagues present a case of a boy who underwent an intervention to close a hole in the wall between two of the heart’s chambers (atrial septal defect). He subsequently developed an enlargement of one of the Sinuses of Valsalva, called Sinus of Valsalva aneurysm, that lead to rupture and required
emergent surgical repair. Sinus of Valsalva aneurysm can be caused by a congenital weakness of the vessel wall, by an infection or by degenerative processes, leading to a variety of complications and symptoms. The authors discuss when patients with this type of anomaly need to be operated. If the patient has no complications or symptoms from the aneurysm, surgery is usually recommended when the aneurysm is very large or grows quickly. In all cases, close follow-up is necessary.

Andrés Enríquez Puga et al.: Chronic Type A Aortic Dissection and Giant Root Aneurysm After Aortic Valve Replacement

Andrés Enríquez Puga and colleagues describe a case of a patient who underwent replacement of his aortic valve, which represents the gate between the heart and the aorta, the body’s main artery. At the time of surgery, his aorta was already somewhat larger than expected, but was not replaced. Several years later, his aorta had enlarged to giant proportions (called aneurysm), and developed a dissection, which describes a tear in the inner wall of the vessel potentially leading to life threatening complications. He underwent surgical replacement of his aorta and recovered well. This case illustrates that if the risk is acceptable, replacement of a moderately enlarged aorta should be considered at the time of original aortic valve surgery to prevent further aortic enlargement.

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Upcoming Meetings

AORTA, June 2016, Volume 4, Issue 3:114
DOI: http://dx.doi.org/10.12945/j.aorta.2016.16.028

List of Upcoming Meetings

November 2016

1. **EACTS Academy: Aortic Valve Surgery**
   November 24-25, 2016
   Nancy, France
   Meeting information available at:
   www.eacts.org/academy/courses/aortic-valve-surgery

December 2016

1. **International Conference for Innovations in Cardiovascular Systems**
   December 4-6, 2016
   Tel Aviv, Israel
   Meeting information available at:
   2016.icimeeting.com

2. **13th European Cardiology Congress**
   December 5-6, 2016
   Madrid, Spain
   Meeting information available at:
   cardiology.conferenceseries.com/europe

January 2017

1. **35th Annual International Symposium: Clinical Update in Anesthesiology, Surgery and Perioperative Medicine**
   January 15-20, 2017
   Cancun, Mexico
   Meeting information available at:
   www.clinicalupdateinanesthesiology.org

2. **Controversies and Updates in Vascular Surgery**
   January 19-21, 2017
   Paris, France
   Meeting information available at:
   cacvs.org

3. **53rd Annual Meeting of the Society of Thoracic Surgeons and STS/AATS Tech-Con 2017**
   January 21-25, 2017
   Houston, Texas
   Meeting information available at:
   www.sts.org/education-meetings/sts-annual-meeting

4. **STS/CTSNet Career Fair at the 53rd Annual Meeting**
   January 22-24, 2017
   Houston, Texas
   Meeting information available at:
   www.ctsnet.org/events/2017-sts-and-ctsnet-career-fair