

subcutaneous emphysema. Radiologic workup revealed pneumomediastinum, and bronchoscopy demonstrated a 2-cm-long laceration of the membranous trachea 3 cm below the vocal chords. Antibiotic therapy with amoxicillin and metronidazole was instituted. The patient was discharged on the tenth postoperative day.

The experience with our patient coupled with the patient outcomes in the previously noted case reports suggests the practicality of conservative management in select cases [5-7], but criteria to assess the potential for nonoperative management of intubation-related tracheal injury are needed. A stable patient is mandatory. Prompt evaluation of the extent of injury is critical. Fiberoptic bronchoscopy allows investigation of the injury and permits antibiotic irrigation and confirmation of tube placement. Bronchoscopy is recommended by many authors for routine placement of double-lumen endotracheal tubes and is a practice with which we concur [4, 8]. Smith and associates [8] found that when double-lumen endotracheal tubes were inserted without the aid of fiberoptic bronchoscopy 48% were found to be malpositioned. Tracheal lacerations have occurred after the placement of both double-lumen and single-lumen endotracheal tubes. To prevent tracheal injury, fiberoptic bronchoscopy should be used to aid intubation in all cases where intubation is difficult.

Evaluation of associated esophageal injury is important and consists of flexible esophagoscopy and thoracic computed tomography with oral contrast. Computed tomography allows assessment of esophageal leak and pneumomediastinum. In addition, computed tomography permits analysis of the volume of mediastinal fluid collection. A large collection suggests hemorrhage and has the possibility of becoming infected.

Antibiotic prophylaxis toward common endobronchial flora is prudent. The length of antibiotic administration should be tailored to the patient.

In summary, we propose the following criteria to be used as guidelines in deciding on nonoperative management of intubation-related tracheal lacerations:

1. Stable vital signs in the patient
2. No difficulty ventilating the patient while intubated or respiratory distress while extubated
3. No evidence of esophageal injury
4. Minimal mediastinal fluid collection
5. Nonprogressive pneumomediastinum or subcutaneous emphysema
6. No signs of sepsis

A low threshold for returning to the operating room for definitive treatment of the injury is warranted should complications develop.

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Anaphylactic Shock After Aprotinin Reexposure: Time Course of Aprotinin-Specific Antibodies

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We report a case of severe anaphylactic shock during a cardiac operation that occurred as a consequence of aprotinin readministration in the presence of preformed aprotinin-specific antibodies. Both immunoglobulin G (3 hours) and immunoglobulin E (5 minutes) antibody levels dropped early after the clinical event. Despite their possibly limited clinical significance, we still recommend the conductance of specific antibody screening tests before readministration of aprotinin.

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A 74-year-old woman was scheduled for mitral valve replacement. Three months before, an attempt to reconstruct the mitral valve using a 33-mm Duran ring had been performed elsewhere. After the previous procedure, the patient continued to present with significant hemolysis and grade III-IV mitral valve regurgitation. The decision was made to proceed to mitral valve replacement at our institution. We were not aware of any previous administration of aprotinin.

During this operation, aprotinin was administered at the time of skin incision. Shortly thereafter, tachycardia (140 beats/min) and arterial hypotension (systolic arterial blood pressure less than 50 mm Hg) developed and the

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Table 1. Serum Concentrations of Aprotinin-Specific Antibodies

Blood Sampling Time	CAP System		ELISA IgG (mg/L)	Western Blot		Hematocrit (%)
	RAST Class	FEIA IgG (mg/L)		IgE	IgG	
	IgE					
Preoperatively	II	>200	>200	+	+++	27.8
5 minutes after anaphylaxis	0	114	121	—	++	25.8
After 3 hours of CPB	0	59	68	—	—	31.8
1 day postoperative	0	32	43	—	—	31.0
14 days postoperative	0	183	177	—	+++	34.0

CPB = cardiopulmonary bypass; ELISA = enzyme-linked immunosorbent assay; FEIA = fluorescence enzyme immunoassay; IgE = immunoglobulin E; IgG = immunoglobulin G; RAST = radio-allergosorbence test.

inspiratory peak pressure increased. Within moments, the patient's heart arrested. At this time, 150,000 KIU (21 mg) of aprotinin had already been infused. Aprotinin administration was immediately stopped and cardiopulmonary resuscitation was commenced together with appropriate drug intervention. Under these measures, the patient's circulation was reestablished within minutes and maintained by high-dose catecholamine support. The left femoral artery was cannulated for the arterial return of cardiopulmonary bypass. At this time, circulatory collapse secondary to ventricular fibrillation occurred again, and the sternum was immediately reopened during ongoing resuscitative measures. Within the chest, dense adhesions between the heart and the mediastinal tissues were found as expected. Therefore, the right mediastinal pleura was quickly opened and the right atrium was cannulated after stab incision from the right pleural cavity, and extracorporeal circulation was established.

Mitral valve replacement using a 29-mm St. Jude Medical (St. Paul, MN) mechanical valve prosthesis was then performed in a routine fashion. Weaning from CPB could only be achieved under high-dose vasoconstrictor support, but there was adequate cardiac function. The patient gradually recovered from the operation over the following days and was extubated on postoperative day 13. However, she died 17 days after the procedure of unexpected sudden cardiac arrest.

Methods

Preoperative, intraoperative, and postoperative blood samples were drawn and screened for aprotinin-specific antibodies retrospectively by the immunologic tests described below (all assays were performed in triplicate).

CAP System

The aprotinin-specific antibodies were quantified with the CAP System (Pharmacia GmbH, Freiburg, Germany) using the radio-allergosorbence test fluorescence enzyme immunoassay for detection of immunoglobulin E (IgE) and specific fluorescence enzyme immunoassay tests for immunoglobulin G (IgG). Quantitative expression of the results was obtained with standard reagents

calibrated against World Health Organization reference preparations for human IgE (75/502) and for human serum immunoglobulins A, G, and M (67/86).

Enzyme-Linked Immunosorbent Assay

Ninety-six-well Immuno-Plates (Maxisorp; Nunc GmbH, Wiesbaden, Germany) were coated with aprotinin (Traisylol; Bayer AG, Leverkusen, Germany). After incubation with patient serum or control serum, aprotinin-specific IgG antibodies were detected by alkaline phosphatase-labeled goat anti-human IgG antibodies (Sigma Inc, St. Louis, MO).

Western Blot

Reduced samples of aprotinin were transferred to nitrocellulose membranes by electroblotting after running sodium dodecyl sulfate-polyacrylamide gel electrophoresis. The membrane was incubated with diluted patient serum (1:500) followed by addition of alkaline phosphatase-labeled goat anti-human IgG or IgE antibodies and then visualized by 5-bromo-4-chloro-3-indolyl phosphate/nitro blue tetrazolium substrate (all reagents are available from Sigma Inc). The intensity of the staining reaction was graded in a semiquantitative fashion.

Results

The results of the immunologic studies are summarized in Table 1 (values are not corrected for hematocrit). Preoperatively, the patient's serum contained both aprotinin-specific IgE and IgG antibodies. After aprotinin readministration, both IgG (after 3 hours) and IgE (as early as 5 minutes) antibody levels showed a significant decrease. Immunoglobulin G antibody levels almost fully recovered to reach preoperative values over the following 14 days (183 mg/L by fluorescence immunoassay), whereas the specific assays for IgE antibodies could not detect any reoccurrence of IgE within the same time frame.

Comment

In open heart operations a substantial reduction of blood loss and blood requirements after cardiopulmonary by-

pass have been reported after prophylactic treatment with aprotinin [1, 2]. Aprotinin, a protein foreign to the human immune system, represents a trespass into the patient's immunologic memory. A 0.7% incidence of untoward events was reported after the first exposure to aprotinin and up to 9% incidence after reexposure [3, 4]. However, most of the existing reports on aprotinin-mediated anaphylactic reactions are solely based on clinical observations.

In the case presented here, we were able to investigate the sequential changes in aprotinin-specific antibodies after the clinical event of severe anaphylactic shock in a patient with preexisting aprotinin-specific IgG and IgE antibodies. Our data indicate a decrease in IgG antibodies 3 hours after aprotinin reexposure, which is thought to occur secondary to formation of antigen-antibody complexes. Serum levels of IgG antibodies almost completely recovered to preoperative values within 2 weeks after the event. This is also true if the IgG antibody levels are corrected for hematocrit (see Table 1). Immunoglobulin E antibodies, in turn, could not be detected in the serum immediately after the anaphylactic event and remained absent for the following 2 weeks. Immunoglobulin E antibodies will bind to mast cells and basophils to precipitate histamine release by the latter [5], which contributes to the anaphylactic reaction.

In this case we studied the aprotinin-specific antibody response using all currently established immunologic assays. All of these tests showed corresponding results (see Table 1). According to our current knowledge, we still recommend the conductance of immunologic screening tests for aprotinin-specific antibodies in patients with a known history of aprotinin administration before reexposure to aprotinin is considered. However the clinical value of such tests remains unknown because the presence of aprotinin-specific antibodies does not necessarily result in an anaphylactic response to a further dose. If any preoperative test for IgG or IgE antibodies is positive and aprotinin administration is still indicated, we recommend administration of aprotinin only with the patient already on extracorporeal circulation starting with a small test dose after previous histamine receptor blockade.

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Stent Angioplasty for Renovascular Disease Associated With Acute Aortic Dissection

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Aortic replacement was performed in a patient with acute type A aortic dissection originating from the descending thoracic aorta. Postoperatively, a persistent false lumen resulted in severe stenosis of the right renal artery. Percutaneous stent angioplasty improved renovascular hypertension and renal function. This procedure is useful for management of patients with branch complications of aortic dissection.

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Renal artery complications associated with acute aortic dissection cause significant operative morbidity [1]. They also can affect the long-term outcome of surgical survivors, because stenotic lesions that persist or develop postoperatively can induce renovascular hypertension and renal failure.

Although percutaneous renal angioplasty has been extensively used for fibromuscular or atherosclerotic renovascular hypertension, few cases have been reported in patients with aortic dissection [2, 3]. The following case report describes stent angioplasty for severe stenosis of the right renal artery in a patient who underwent operation for an acute type A aortic dissection originating from the descending thoracic aorta.

In March 1995, a 57-year-old woman was diagnosed with an acute type A aortic dissection and underwent emergency partial replacement of the proximal aortic arch that had a false lumen. The intimal tear located in the descending thoracic aorta could not be resected through a median sternotomy.

Postoperatively hypertension developed that was difficult to control. Her systolic blood pressure frequently exceeded 180 mm Hg in spite of the intravenous administration of nicardipine. Her blood pressure was barely controlled (132/90 mm Hg on average) 1 month later with five antihypertensive drugs, including clonidine, prazosin, bisoprolol (β -blockers), nifedipine, and imidapril (angiotensin-converting enzyme inhibitor). Her serum creatinine and blood urea nitrogen levels remained normal, but the right glomerular filtration rate was de-

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