Isolated Chylopericardium after Intrapericardial Procedures

Possible Role of Inadvertent Right Efferent Lymphatic Trunk Injury

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Chylopericardium after an intrapericardial procedure is rare, and satisfactory explanations of its possible causes are lacking.

Herein, we present 4 cases of chylopericardium that developed after intrapericardial surgery, and we review the literature.

Our literature review revealed 29 cases of chylopericardium that complicated intrapericardial operations, to which we added our 4 cases for analysis. The 33 surgical procedures involved repair for congenital heart disease (n=21), valve surgery (n=5), coronary artery bypass grafting (n=6), and other (n=1). Causes were verified in 7 patients: small lymphatic injury in 3 and high venous pressure or venous thrombosis in 4. Of the 26 patients with chylopericardium of unknown origin, 15 had congenital heart disease. Ten of these 15 had chromosomal abnormalities, especially trisomy 21 (Down syndrome); these patients typically had increased lymphatic permeability, which raised the likelihood of chylopericardium. Five revascularizations for coronary artery disease required harvesting of the left internal thoracic artery for reconstruction, incurring a risk of damage to the drainage site of the right efferent lymphatic trunk. In addition, all 26 patients with chylopericardium of unknown origin underwent dissection of the ascending aorta and the main pulmonary artery, near the right efferent lymphatic trunk. Inadvertent injury to the trunk during the dissection would have increased the risk of chylopericardium. Accordingly, even though the overall incidence of chylopericardium during intrapericardial procedures is low, we recommend a meticulous dissection of the ascending aorta from the main pulmonary artery. **(Tex Heart Inst J 2007;34:82-7)**

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Chylopericardium is rare after an intrapericardial procedure, because the sites of surgical dissection are usually anatomically remote from the thoracic duct. Even though some procedures may impede thoracic duct drainage to the subclavian vein, with the resultant elevation in venous pressure, the usual manifestation is chylothorax rather than chylopericardium. A 2002 cadaver study of the intrathoracic tributaries of the thoracic duct revealed that tributaries from the heart arrive at the thoracic duct via 2 routes.[1] Injury to these tributaries along their course within the pericardium may account for postoperative chylopericardium. Nevertheless, chylopericardium arising from post-intrapericardial procedures is described in few reports in the medical literature.[2-20] Herein, we present our experience with 4 patients, review the relevant literature, and refer to the cadaver study as we speculate on probable injury sites.

Case Reports

Patient 1

A 3-year-old boy was admitted with a diagnosis of a secundum-type atrial septal defect. To facilitate accurate cross-clamping of the aorta during the operation, we performed limited dissection of the ascending aorta (AAo) from the main pulmonary artery (MPA), separating the connecting soft tissues for clamping purposes. Forty-eight hours postoperatively, the mediastinal fluid drainage increased and became opalescent. The cholesterol and triglyceride levels of the effusion were 53 and 1,515 mg/dL, respectively. A diet of milk rich in mid-chain fatty acids was prescribed.

By day 6, the chylous fluid turned transparent, and its total daily volume decreased substantially. On the basis of these observations, the mediastinal tube was removed on day 7. No recurrence of the fluid accumulation was noted during regular echocardiographic follow-up over the next 5 years.

Patient 2

A 2-year-old boy was admitted for corrective surgery for tetralogy of Fallot. The AAo was dissected from the MPA extensively because of the expectation that a transannular patch would be required. The ventricular septal defect was repaired, and a transannular patch was used for right ventricular outflow tract reconstruction. The persistence of a moderate amount of serosanguineous pericardial effusion (>400 cc/day) was noted immediately after the operation. The effusion turned milky in color on day 14, and the levels of cholesterol and triglycerides therein were 42 and 202 mg/dL, respectively. A diet of mid-chain fatty-acid milk was administered to the patient until the effusion decreased; the mediastinal tubes were removed on day 20.

Patient 3

A 1-year-old boy with known Down syndrome and severe pulmonary hypertension (Qp/Qs=5.3) was admitted for surgical correction of an atrioventricular septal defect (complete form), and a small patent ductus arteriosus. The routine dissection of the AAo from the MPA was extended to encircle the patent ductus arteriosus before cardiopulmonary bypass was instituted. The atrioventricular septal defect was repaired by means of a 2-patch technique, with the coronary sinus draining into the left atrium. On postoperative day 10, massive pericardial effusion was observed on echocardiography. Subxiphoid pericardial drainage was begun immediately. The cholesterol and triglyceride levels of the milky fluid were 33 and 278 mg/dL, respectively. Chylopericardium was diagnosed, and a diet rich in mid-chain fatty acids was promptly implemented. After 18 days of dietary control, the drainage volume decreased, with no recurrent fluid accumulation noted during follow-up.

Patient 4

A full-term newborn boy was transferred to our hospital due to an imperforate anus, and an urgent colostomy was performed. On the basis of the infant's facial dysmorphism, Down syndrome was suspected, and trisomy 21 was confirmed upon subsequent chromosomal examination. Echocardiography and cardiac catheterization revealed a large ventricular septal defect, severe tricuspid valve regurgitation, and pulmonary artery hypertension. At the age of 4 months, the infant underwent surgical correction of the ventricular septal defect. The AAo was dissected from the MPA in standard fashion. Although the postoperative course was

smooth, the pericardial discharge turned milky-yellow on day 3. The levels of cholesterol and triglycerides in the effusion were 28 and 185 mg/dL, respectively. A diet rich in mid-chain fatty acids was prescribed. Seven days later, the drainage volume decreased, and the pericardial tubes were removed.

Review

We reviewed the English-language medical literature, searching for the key word "chylopericardium." Studies that provided insufficient individual patient data were excluded; we considered only the 29 cases from 19 reports that exclusively dealt with isolated chylopericardium that had developed after intrapericardial procedures. Including our 4 patients, our review encompassed 33 cases.

Table I summarizes the demographic data. Twenty patients (61%) were male, and 19 were children (58%). Surgical procedures comprised repair for congenital heart disease (n=21), valve surgery (n=5), coronary artery bypass surgery (n=6), and correction of idiopathic hypertrophic subaortic stenosis (plus a myomectomy) (n=1). After we excluded 7 patients for whom no clinical symptoms were mentioned, the most prevalent presentations were cardiac tamponade (10/26) and persistent or increased drainage volume with the fluid becoming opalescent (9/26) after postoperative food intake. Twenty-seven patients were diagnosed with chylopericardium within 30 days of surgery; the late-onset average for the other 6 patients was 62.5 days. Eighteen patients were prescribed dietary regimens involving foods rich in medium-chain fatty acids, low-fat intake, or nothing by mouth. Five patients underwent low thoracic duct ligation or additional pericardioperitoneal shunt surgery, 2 underwent re-exploration for suture repair, and 1 received conservative therapy with additional somatostatin treatment. The causes of the chylopericardium were determined to be small lymphatic injury (n=3; cases 7, 14, and 29) and high venous pressure or venous thrombosis (n=4; cases 1, 5, 15, and 19). The remaining 26 cases were of unknown origin.

We explored the possible causes of chylopericardium in these 26 patients. Ten of the 15 congenital heart disease patients (cases 9, 21-27, 30, and 32) had chromosomal abnormalities; 9 of these patients had trisomy 21. Increased lymphatic permeability due to congenital lymphatic dysplasia probably led to the postoperative chylopericardium that was reported in the Down syndrome patients and in the other patient with a chromosomal anomaly. Five of the 6 patients who underwent coronary artery bypass had the left internal thoracic artery harvested as a conduit (cases 8, 10, 11, 13, and 20), and these dissections were extended to the 1st intercostal space near the drainage site of the right efferent lymphatic trunk (RET)—a possible site of injury that

TABLE I. Summary of Cases of Chylopericardium after Intrapericardial Surgery

Year	Reference	Case No.	Diagnosis; Operation	Pt. Age/ Sex	ChrA	Management	Postop Onset (d)	Cause
1971	Thomas CS and McGoon DC[2]	1	PA; PDA/VSD repair, homograft reconstruction	31 y/F	–	TDL	60	Thrombosis at jugular–subclavian venous confluence
1977	Kansu E, et al.[3]	2	AR, AS; AVR	53 y/F	–	Conservative	35	Unknown
1981	Pollard WM, et al.[4]	3	IHSS; myotomy, myomectomy	23 y/M	–	Conservative	3	Unknown
		4	PS; pulmonic valvotomy	4.5 y/M	–	Conservative	10	Unknown (mediastinal lymph increase)
		5	CAD; CABG	57 y/M	–	Conservative	4	Left subclavian vein obstruction
		6	—; VSD repair	22 y/M	–	TDL	3	Unknown
1982	Rose DM, et al.[5]	7	Infundibular PS; MRS	20 y/F	–	Suture repair	12	Lymphatics of periaortic fat transected
1982	Fudge TL, et al.[6]	8	CAD; CABG	57 y/M	–	Conservative	9	Unknown
1984	Papaioannou Y, et al.[7]	9	TOF; total correction	9 y/M	tri 21	Conservative	5	Unknown
1984	Bakay C and Wijers TS[8]	10	CAD; CABG	41 y/M	–	PP fistula	35	Unknown
1984	Schiessler A, et al.[9]	11	CAD; CABG	45 y/M	–	Conservative	1	Unknown
1985	Tchervenkov CI and Dobell AR[10]	12	AS; aortic valvotomy	12 y/M	–	Conservative	34	Unknown
1987	Lee Y, et al.[11]	13	CAD; CABG	49 y/M	–	TDL, DPS	8	Unknown
1988	Pereira WM, et al.[12]	14	—; ASD II repair	4 y/F	–	TDL	17	Lymphatics injured at IVC
1989	Denfield SW, et al.[13]	15	TGA; Mustard	9 m/M	–	Conservative	180	SVC obstruction
		16	—; ASD repair	1 y/M	–	Conservative	5	Unknown
1989	Bar-El Y, et al.[14]	17	MS; MVR	39 y/F	–	Conservative	31	Unknown
1994	Schactman M, et al.[15]	18	AS; AVR (concomitant CABG)	77 y/M	–	Conservative	4	Unknown
1998	Rimensberger PC, et al.[16]	19	TGA; atrial switch	4 m/M	–	Conservative + somatostatin	2	Increasing central venous and left atrial pressure
1999	Sharpe DA, et al.[17]	20	CAD; CABG	63 y/F	–	Conservative	11	Unknown
2001	Campbell RM, et al.[18]	21	—; TOF repair	3 m/M	22q–	Conservative	7	Unknown
		22	—; AVSD repair	3 m/F	tri 21	Conservative	23	Unknown
		23	—; AVSD repair	4 m/F	tri 21	Conservative	19	Unknown
		24	—; AVSD repair	5 m/F	tri 21	Conservative	13	Unknown
		25	—; AVSD repair	5 m/F	tri 21	Conservative	6	Unknown
		26	TOF; AVSD repair	2 y/M	tri 21	Conservative	28	Unknown
		27	TOF; AVSD repair	2 y/F	tri 21	Conservative	22	Unknown
2002	Sasaki A, et al.[19]	28	MR; MVR	49 y/M	–	Conservative	2	Unknown
2003	Shanmugam G, et al.[20]	29	—; ASD repair	8 y/F	–	Suture repair	3	Thymic lymphatic leakage
2007	Kan C-D, et al.*	30	AVSD; PDA repair	1 y/M	tri 21	Conservative	10	Unknown
		31	—; ASD repair	3 y/M	–	Conservative	3	Unknown
		32	—; VSD repair	4 m/M	tri 21	Conservative	4	Unknown
		33	—; TOF repair	2 y/M	–	Conservative	14	Unknown

*Current study

AR = aortic valve regurgitation; AS = aortic valve stenosis; ASD = atrial septal defect; AVR = aortic valve replacement; AVSD = atrio-ventricular septal defect; CABG = coronary artery bypass grafting; CAD = coronary artery disease; ChrA = chromosomal anomaly; DPS = Denver pleuroperitoneal shunt; IHSS = idiopathic hypertrophic subaortic stenosis; IVC = inferior vena cava; m = months; MR = mitral regurgitation; MRS = muscular resection; MS = mitral stenosis; MVR = mitral valve replacement; PA = pulmonary atresia; PDA = patent ductus arteriosus; PP = pericardioperitoneal ; PS = pulmonary valve stenosis; SVC = superior vena cava; TDL = thoracic duct ligation; TGA = transposition of great vessels; TOF = tetralogy of Fallot; tri 21 = trisomy 21 (Down syndrome); 22q– = chromosome 22q deletion syndrome; VSD = ventricular septal defect; y = years

would cause chylopericardium. Of the remaining 6 patients, cases 2, 3, 12, and 18 underwent an aortotomy, and cases 17 and 28 underwent mitral valve replacement. No single procedure appeared to jeopardize the lymphatic trunk in these 6 patients. However, all 26 patients underwent varying degrees of dissection of the AAo from the MPA, in proximity to the RET. An inadvertent injury to the RET during blunt dissection would engender chylopericardium.

Discussion

Chylopericardium that complicates intrapericardial surgery is extremely rare. Thomas and McGoon[2] first reported this problem in 1971 after procedures involving cardiopulmonary bypass. Chylopericardium has been reported after valve replacement, myocardial revascularization, and corrections of congenital heart anomalies[2-20]; the overall incidence in the literature is less than 0.22%.[4,7,18,21] Upon retrospective review of all of the surgical patients in our hospital from September 1988 through February 2004, we calculated an incidence of 0.12% (4 of 3,332).

Normally, the lymphatic system drains chylous fluid, which is filtered by the capillaries from the interstitial spaces into the thoracic duct. The thoracic duct passes through the chest near the aorta and azygos vein and then terminates at or near the junction of the left subclavian artery and the jugular veins, into which the chyle drains. It is generally accepted that direct injury to the thoracic duct results in chylothorax rather than chylopericardium because the thoracic duct is located in the region of the descending aorta, and direct injury to the thoracic duct during a purely intrapericardial procedure is unlikely. Identifying the mechanism responsible for the development of chylopericardium is of interest to surgeons; however, due to the paucity of clinical experience with chylopericardium, few satisfactory explanations have been forthcoming to account for its occurrence.

Causes for chylopericardium could be established in only 7 of the patients who underwent intrapericardial procedures. Injury to the lymphatic system near the inferior vena cava during re-exploration was diagnosed in case 14. Small lymphatic leaks in the thymic tissues or periaortic fat were noted during reoperation in cases 7 and 29; both were treated by direct suture ligation. In cases 1, 5, 15, and 19, the chylopericardium was related to obstruction of normal thoracic ductal flow, as confirmed by venography, actual symptoms, or pressure tracing. Both direct obstruction of the systemic venous return (as seen in cases 1, 5, and 15) and abnormally elevated systemic venous pressure transmitted into the lymphatic channels (case 19) may increase the pressure within the thoracic duct; the same is true when patients undergo a Fontan procedure for tricuspid atresia and develop chylothorax, which results in pericardial chyle accumulation. Whereas in adults the thymus is nearly devoid of lymphatic tissue, in children the thymus is rich in lymphatic tissues. This may explain why chylopericardium develops more often after pediatric surgery, and why most such instances involve the anterior mediastinum of the thymic tissue.

Although many patients in our literature review were surveyed for possible pathogenesis, in most it remained undetermined. Previous researchers[18,22] have concluded that an important cause of postoperative chylopericardium is the increased lymphatic permeability due to congenital lymphatic dysplasia that contributes to the chylous effusions present in Down, Noonan, and Turner syndromes. Ten patients in our survey were found to have a chromosomal anomaly, mainly trisomy 21. However, the pathogenesis was unclear in the other 16 patients. We analyzed the general clinical manifestations in this subgroup. All 16 patients had undergone either some degree of dissection of the AAo from the MPA (to facilitate accurate aortic cross-clamping) or harvesting of the left internal thoracic artery (for coronary artery bypass).

In a study of the intrathoracic tributaries of the thoracic duct in 530 cadavers, 8.9% of lymphatic vessels were found to arise from the heart and connect with the thoracic duct.[1] There are 2 routes for these tributaries—the RET and the left efferent lymphatic trunk (LET). The RET, which often drains lymph from the right ventricle, ascends cephalad between the AAo and the MPA and joins the upper part of the left anterior mediastinal node chain, which is located at the level of origin of the internal thoracic artery. The RET then travels to the left of the thymus gland and drains into the arch of the thoracic duct (Fig. 1). The LET, which drains lymph from the left ventricle, ascends behind the MPA, joins the right paratracheal node, and drains into the thoracic duct at the mediastinum or arch. This confirms the observation of Pollard and colleagues,[4] who demonstrated the phenomenon of increased numbers of superior mediastinal lymphatics in 1 patient with chylopericardium (case 4).

Riquet and associates[1] have suggested that injury to an incompetent RET along its course within the pericardium is a possible cause of isolated postoperative chylopericardium.[1] (Note: We frequently dissect the AAo from the MPA in order to cross-clamp the AAo accurately after cardiopulmonary bypass. Inadvertent injury to the RET might be unavoidable during this dissection or the subsequent clamping; however, backflow from the thoracic duct actually occurs only if the integrity of the RET is compromised and results in leakage of chyle. This may explain why the incidence of isolated chylopericardium is so low even though dissection of the AAo from the MPA is a routine procedure.)

In patients who have undergone coronary artery bypass grafting, harvesting of the left internal thoracic artery above the left subclavian vein with ligation of
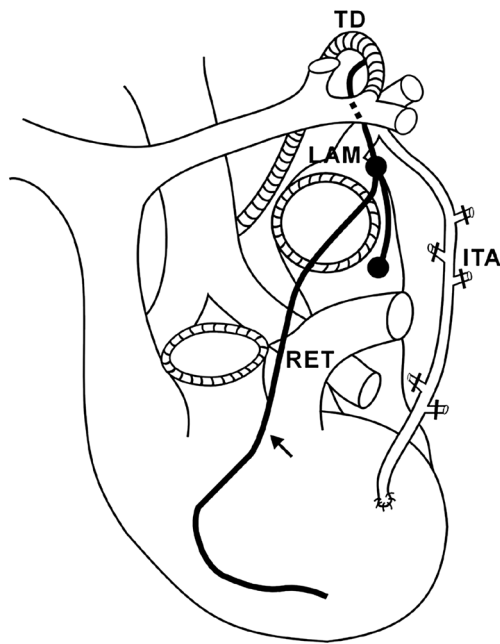
Fig. 1 *The right efferent lymphatic trunk (RET), which drains the lymph from the right ventricle, arises between the ascending aorta and the main pulmonary artery (arrow), joins the upper part of the left anterior mediastinal node chain (LAM) located at the origin of the internal thoracic artery (ITA), and empties into the arch of the thoracic duct (TD).*

the upper intercostal branch may incur an additional risk of chylopericardium or chylothorax if the pleural membrane is opened. The risk may be increased because the anterior mediastinal node chain is easily jeopardized during proximal dissection of the internal thoracic artery, and chylous leakage may ensue. This complication is under-reported and shows the danger of high dissection of the internal mammary artery. The intrapericardial collection of pericardial effusion after cardiac surgery is primarily related to not opening the pleural membrane.

Two research groups[7,23] showed improvements in patients with chyluria and chylothorax who were prescribed a medium-chain triglyceride diet. This regimen has since proved effective in many cases, under varied conditions. Therefore, we recommend treating chylothorax and chylopericardium with 2 weeks of conservative therapy: decompression of the thoracic lymphatics by parenteral hyperalimentation or oral medium-chain triglycerides, together with decompression of the pericardial space by closed chest-tube suction. To avoid prolonged hospitalization, we recommend outpatient drainage of a chylous effusion with the use of a soft silastic catheter (Pleurx; Denver Biomedical, Inc.; Golden, Colo) that is tunneled to prevent infection. More than half of the patients whose cases we reviewed were successfully treated by use of this method.

Control of chyle output with somatostatin has also been advocated.[25,26] Surgical reintervention was required in only 6 of 34 patients so treated. Generally, the indications for surgery are drainage of more than 1,000 mL/day in adults, or 100 mL × years of age in children, for the first 7 days, or persistent chylous drainage for longer than 2 weeks.[26-29] Ligating the thoracic duct at the diaphragmatic level confers the advantage of stopping flow from accessory ducts that may be unrecognized[30,31]; however, sometimes the thoracic duct cannot be found intraoperatively, or ligation fails to control the leak.[28,32] Other surgical options include pleurodesis, pleurectomy, low ligation of the thoracic duct by video-assisted thoracoscopic surgery, and pericardial–peritoneal shunting,[28,30,33,34] but the results are not always satisfactory.[24]

Summary

Chylopericardium after simple intrapericardial surgery is a rare entity, but one that is associated with potentially serious problems. Chylopericardium can result from injury to the lymphatic branches of the thymus, increased lymphatic permeability associated with the lymphatic dysplasia in patients with Down syndrome, or thrombosis of the great venous system. An inadvertent injury to the RET during dissection can also lead to chylopericardium. We advise caution when dissecting the tissues between the AAo and the MPA, and we discourage harvesting the internal mammary artery as far as the upper intercostal branches. Although surgical reintervention may be required for refractory chylopericardium, a conservative treatment strategy is still the 1st choice.

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