

Issue 78 September 2024

A Knowledge Sharing Initiative by Medanta

Innovative Embolization Solves Rare Chylopericardium

Persistent cardiac effusion treated with minimally invasive technique

Chylopericardium is an uncommon disorder marked by the build-up of chyle, a triglyceride-rich fluid, in the pericardial cavity. This accumulation can exert significant pressure on the heart, frequently leading to tamponade and posing a therapeutic challenge.

Chyle is transported by the thoracic duct. Primary (idiopathic) chylopericardium is diagnosed by excluding other causes, while secondary chylopericardium typically results from injury, disruption, or blockage of the thoracic duct due to surgery, radiation, trauma, malignancy, or infection. Although most patients present without symptoms, typical signs include cough, chest pressure, chest pain, lightheadedness, and syncope. Rapid fluid accumulation can lead to cardiac tamponade.

Idiopathic chylopericardium is extremely rare, with limited cases reported. This report details our experience in managing a case of idiopathic chylopericardium.

Case Study

A 17-year-old boy with no known health issues was in good health until November 2022, when he developed a fever, dry cough, and rhinitis, resulting in a diagnosis of an upper respiratory tract infection. A persistent cough lasting about a month led to routine chest X-ray, revealing cardiomegaly. An echocardiogram performed at a peripheral center confirmed pericardial effusion.

Suspecting tuberculosis (TB), anti-tubercular treatment was initiated empirically, without a pericardiocentesis, and the patient continued this treatment from January 2023 to July 2023. In this period, he found symptomatic improvement in cough but had persistent intermittent palpitations, lethargy, weight loss, chest heaviness.

These persisting symptoms led him to another cardiology centre in August 2023. Echocardiography revealed massive pericardial effusion for which pericardiocentesis was done, showing fluid rich in triglycerides and chylomicrons, indicative of chylopericardium. The chylopericardium was stubborn in nature and hence was further evaluated.



Magnetic resonance lymphangiography, whole-body lymphoscintigraphy, conventional lymphangiography, and whole-body PET scan were performed, which were inconclusive. The patient underwent multiple pericardial taps for this recurrent effusion, and considerations regarding the creation of a pericardial window were made.

In May 2024, the patient visited Medanta Gurugram for further evaluation. Echocardiography showed massive pericardial effusion with signs of cardiac tamponade. He was admitted to the ICU, where pericardiocentesis was performed through an anterior thoracic approach. The effusion was chylous, with a high triglyceride level (1234 mg/ dL) and lymphocyte predominance. Work-up for secondary causes of pericardial effusion, including autoimmune and inflammatory causes, was done and found to be negative.

Due to the recurrence of significant chylous pericardial effusion, an interventional radiology consultation was sought. A conventional lymphangiogram was performed under general anesthesia. Bilateral inguinal lymph nodes were punctured, and Lipiodol was injected to delineate the cisterna chili. Using Dyna CT, the cisterna chyli was punctured



Successive images showcasing the lymphatic system. Images C and D indicate a lymphatic leak into the pericardium (marked by a yellow arrow).

and the thoracic duct was opacified. The patient had a type-3 pattern of the thoracic duct with two outflow tracts. A plan to embolize the right portion of the niche of abnormal lymphatic vessels was made using coils and sclerosant. The patient continued on a fat-free diet. However, follow-up did not show remarkable improvement. Due to persistent pericardial effusion, a re-intervention was planned.

Using fluoroscopic guidance, the cisterna chyli was reaccessed, and two wires were placed in the thoracic duct after performing a lymphangiography. Retrograde access of the left thoracic duct was achieved using a microcatheter. A repeat lymphangiogram demonstrated a pericardial leak of contrast. A plan to perform embolization of the remaining trunk was made without hampering the thoracic duct. Temporary gelfoam occlusion of the right outflow was made, following which balloon-assisted glue embolization (1:8 dilution) was performed.

Post-procedure, octreotide and total parenteral nutrition were initiated. Follow-up imaging showed complete symptom resolution and negligible pericardial effusion on echocardiography.

This case provides valuable insights into the diagnosis and treatment of idiopathic chylopericardium.

Discussion

Understanding the lymphatic system's structure is crucial for comprehending chylous leak. This system consists of lymphatic vessels, nodes, and the cisterna chyli. The primary vessel, the thoracic duct, collects roughly 75% of the body's lymph and channels it into the venous bloodstream. Acting as a vital fluid circulation network, it connects the lymphatics across different organs. The cisterna chyli, a triangular expansion in the lymphatic system, is situated in the retroperitoneum, just behind the abdominal aorta near the second lumbar vertebra. The thoracic duct originates from the cisterna chyli, starting at the level of the second or third lumbar vertebra, and merges into the junction of the left subclavian and jugular veins. Chyle, primarily composed of chylomicrons, contains longchain triglycerides, cholesterol esters, and phospholipids, and is abundant in lymphocytes—mainly T lymphocytes with cell concentrations ranging from 400 to 6800. Though its electrolyte concentration is similar to plasma, chyle is rich in immunoglobulins and fat-soluble vitamins.

Chylous leaks are rare conditions resulting from the escape of chyle from the lymphatic system. They can occur anywhere along the chyle pathway, starting in the intestinal lymphatic ducts and extending through the cisterna chyli into the thoracic duct. These leaks can lead to various clinical conditions, such as chylothorax, chylopericardium, postoperative chylous wound leaks, chylous ascites, chyloptysis, and chyluria.

Typically, the pericardial space contains 25ml-35ml of fluid, similar to lymph fluid, produced by the epicardium. The pericardium does not produce a significant amount of fluid. Lymphatic vessels of the heart drain pericardial fluid into the left subclavian vein via mediastinal lymphatics, lymph nodes, and the thoracic duct.

The primary lymphatic drainage from the pleura, the lower left lung, the entire right lung, and the pericardium converges at the bronchomediastinal lymphatics.

Conditions that can be mistaken for chylous pericardium include cholesterol pericarditis and purulent pericarditis. Cholesterol pericarditis usually presents with an orange effusion filled with cholesterol crystals. Purulent pericarditis generally presents more severely than chylous pericardium. Diagnosis of pericardial effusion caused by cholesterol or purulent pericarditis depends on cell count, biochemical analysis, cytology, and fluid culture. Diagnosis of chylopericardium is confirmed by pericardial fluid analysis showing: 1) a triglyceride level greater than 500 mg/dL (5.65 mmol/L) and a cholesterol/triglyceride ratio of less than 1, 2) fat globules identified on Sudan III staining, 3) negative cultures and no malignancy identified on cytology, 4) lymphocyte predominance (primarily T lymphocytes) on cytological examination, 5) protein content exceeding 3 g/dL and high specific gravity.

Idiopathic chylopericardium, a rare disorder, is characterized by milky white or pink pericardial fluid with elevated triglyceride levels. Low-output chylothorax (<1000 mL/day) is typically managed with dietary modifications, while high-output chylothorax often requires surgical thoracic duct ligation, performed through open surgery, video-assisted, or robot-assisted means. This procedure usually involves ligating the thoracic duct in the lower thorax, often combined with pericardectomy or pericardial fenestration to ensure sufficient drainage. Even after medical or surgical intervention, there is a high recurrence rate of chylopericardium.



A: Lymphangiography revealing coil placement in the right collateral outflow. B: CT scan illustrating a large pericardial effusion with hyperdense Lipiodol at the posterior, indicative of a lymphatic leak. C: Imaging from the second intervention showing two access wires navigating the lymphatic system. D: Combined antegrade and retrograde conventional lymphangiography.

Thoracic duct embolization, introduced by Dr. Constantine Cope, offers a minimally invasive alternative to ligation. Over the past 20 years, image-guided embolization of leaking lymphatic vessels has become a primary treatment for chylothorax, albeit with varied success rates due to the complexity of the condition. MR/CT lymphangiography followed by conventional lymphangiography has also proven useful in detecting and treating this condition. Case reports and series have highlighted the role of percutaneous embolization in managing this rare condition. Combining embolization with octreotide and total parenteral nutrition can provide significant relief.

Conclusion

Percutaneous embolization offers a valuable, minimally invasive treatment option for the challenging and rare diagnosis of chylopericardium. It should be considered as a primary intervention tool in the management of chylous leaks over surgical or medical management.

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Medanta@Work

Half-Match HSCT Cures Child of Infections and Bleeding in a Case of Wiskott Aldrich Syndrome

Wiskott Aldrich Syndrome (WAS) is a type of primary immunodeficiency disorder (PID), characterised by bleeding manifestations, recurrent infections, eczema, autoimmunity, and malignancy. Over the last decade, improved awareness and better in-house diagnostic facilities have aided increased timely recognition of WAS cases across India.

Case Study

A 3.5-year-old boy presented to Medanta Gurugram. He had a history of cradle cap and seborrheic dermatitis in early infancy. Later on, on-and-off episodes of bleeding, epistaxis and bleeding per-rectal, were reported along with frequent fever and cough because of which the patient's treating paediatrician ordered a Complete Blood Count test.

The result showed thrombocytopenia. Initially, the paediatrician thought it to be a case of Immune thrombocytopenic purpura (ITP). However, proper evaluation led to the diagnosis of Wiskott-Aldrich Syndrome (WAS) as the boy had eczema, small platelets and frequent infections. Genetic mutation testing confirmed WAS gene mutation.

He was kept on monthly IVIG therapy and septran prophylaxis. Even after this, the patient developed two episodes of major infections—pneumonia and shigellosis—requiring hospitalisation. So, the child was referred to Medanta Gurugram's Division of Paediatric Hemato-Oncology and Bone Marrow Transplant for further management. Since he was having bleeding episodes because of thrombocytopenia and repeated infections, hematopoietic stem cell transplant (HSCT) was needed for definitive treatment. The patient was a single child and there was no matched sibling donor, he was planned for an alternative donor HSCT.

At 4.5 years of age, he underwent half-matched HSCT with his mother as the donor. Blood group of the patient was O-positive, and the mother was A-positive. Busulfan, fludarabine and ATG (Anti-thymocyte globulin) was used for conditioning. After the transplant, cyclophosphamide was used for graft versus host disease (GVHD) prophylaxis along with mycophenolate mofetil (MMF) and tacrolimus.

Post-transplant course was uneventful except for hyperactive airway disease, which was managed with steroids and antihistaminics and inhaled bronchodilators. The patient was discharged on Day 20 after transplant. On chimerism, 100% donor cells were found. There was no viral reactivations or GVHD. Initially, for the first six months, the child required monthly IVIG infusions. After that, his serum IgG level normalised, and he did not require any IVIG thereafter. His blood group has changed to A-positive (same as the donor). He has completed his vaccination schedule.

The patient is now 6 years post-transplant, and is living a normal life. He attends school regularly with no major infections and no hospitalisations.

At Medanta Gurugram, we are a high-volume centre for transplants for various primary immunodeficiency diseases, including WAS, from all types of donors, including matched sibling donors, matched unrelated donors and Haploidentical donors with high success rate. Nowadays, haploidentical HSCT are increasing in numbers with small family norms and low availability of matched sibling donor.

Discussion

This Wiskott-Aldrich Syndrome is a complex and severe X-linked disorder characterised by microthrombocytopenia (small platelets), eczema, immunodeficiency, and increased risk in developing autoimmunity and lymphomas. The gene responsible for WAS is located on the short arm of the X chromosome at Xp11.22–p11.23. The WAS gene encodes the WAS protein (WASp). WASp is involved in actin polymerization and associated coupling of receptor engagement, signaling events, and cytoskeletal rearrangement.

The diagnosis of WAS should be considered in any male presenting with eczema, ecchymosis, petechiae, and mucosal bleeding—easy bruising, epistaxis, hematochezia, haematuria—with recurrent and severe sino-pulmonary infections, viral infections, fungal infections. Sometimes, features of autoimmunity like haemorrhage, cytopenia, vasculitis, inflammatory bowel disease, arthritis, renal disease, are also present. On investigation, there is low haemoglobin with microcytosis, thrombocytopenia, low mean platelet volume (microthrombocytes), low IgG, IgA, IgM, IgE levels. There is T-cell lymphopenia.

Without definitive treatment, it is difficult for these children to reach the adult life. Hematopoietic stem cell transplantation (HSCT) is the treatment of choice. In general, HSCT outcomes have improved over time for all donor types. For WAS patients, who lack matched sibling donor, haploidentical donors are being used for HSCT. After transplant, the patients are able to live a normal life.

When to Suspect Wiskott-Aldrich Syndrome

Doctors should suspect WAS in male patients presenting with:

- Recurrent and severe infections (especially sino pulmonary, viral, and fungal)
- Eczema
- Bleeding tendency (easy bruising, petechiae, ecchymosis, epistaxis, hematochezia, hematuria)
- Thrombocytopenia with small platelet size (microthrombocytopenia)
- Features of autoimmunity (e.g., hemorrhage, cytopenia, vasculitis, inflammatory bowel disease)

Early diagnosis and referral to a transplant center is crucial for improved patient survival.

Conclusion

If a boy presents with recurrent infections, eczema and bleeding, then WAS should be suspected. Not all thrombocytopenia cases are immune thrombocytopenia (ITP). Early diagnosis of WAS with early referral to a transplant centre leads to improved survival in patients.

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Spotlight

Not all Filling Defects are Bland Thrombus

Tumoural pulmonary embolism (TPE) is a rare but potentially life-threatening condition characterised by the embolization of tumour fragments to the pulmonary vasculature. TPE most commonly occurs in patients with advanced malignancies, particularly adenocarcinomas, and can manifest as acute or subacute respiratory distress. Imaging techniques, such as computed tomography pulmonary angiography (CTPA) and ventilation-perfusion (V/Q) scans, are pivotal for diagnosis. Treatment options include anticoagulation, systemic therapy for the underlying malignancy, and surgical interventions in select cases.

Despite advancements in management, TPE remains challenging due to its unpredictable clinical course and high mortality rate. It is characterised by the migration of tumour cells or tumour-derived emboli to the pulmonary vasculature, resulting in acute or subacute respiratory compromise. While venous thromboembolism (VTE) is a well-recognized complication in cancer patients, TPE represents a distinct entity with unique clinical features, diagnostic challenges, and therapeutic considerations. Histopathological analysis of pulmonary emboli in TPE often reveals tumour cells within the thrombus, confirming the neoplastic origin of the embolic material. Hereby, we highlight one such case of sub massive pulmonary embolism which was managed by pharmaco-mechanical thrombectomy.

Case Study

A 65-years old female, who was a known case of adenocarcinoma colon (post-extended right hemicolectomy) presented to the emergency unit of Medanta Lucknow with exertional dyspnoea. She was on immunotherapy for recurrent disease and lung metastasis. She was on therapeutic dose of rivaroxaban for prior pulmonary thromboembolism. In view of presenting complains and past history, biochemical and imaging work up was done including D-dimers, cardiac biomarkers, echocardiography and CT-pulmonary angiography. Her D-dimer was 920 ng/ml, HS troponin I (35.14 ng/L) and NT pro-BNP (1210 pg/mL). 2D- echocardiography was normal except for grade 1 left ventricular diastolic dysfunction and trace MR/TR. CT pulmonary angiography showed diffuse multifocal intraluminal hypodense filling defects involving left pulmonary artery just extending to left pulmonary trunk and involving all the distal segmental branches of upper and lower lobe with no post contrast opacification. Filling defects were also noted in right upper lobe segmental branches and posterior distal segmental branches of right lower lobe. Poor pacification of left pulmonary vein and its branches was also noted.



CT pulmonary angiogram showing acute pulmonary thrombosis (a) Axial sections showing non- enhancing hypoattenuating eccentric thrombus in right pulmonary arterial branches (blue arrowheads) and complete thrombosis in left branch of pulmonary artery (yellow arrow). (b) Coronal section showing non-enhancing complete thrombosis in left interlobar artery (yellow arrowheads).

Opinion of critical care medicine and pulmonary medicine teams were taken and in view of sub-massive pulmonary embolism with deteriorating clinical status of the patient, patient was taken in DSA lab for mechanical pulmonary thrombectomy after discussing with family and taking their informed consent.

Under aseptic conditions, access was taken via right common femoral vein, 16 F long sheath inserted and 5F Picard catheter was advanced into the right atrium over floppy-tip guidewire. The sheath was advanced over the catheter-wire assembly and parked at cavoatrial junction. Catheter-wire assembly was negotiated through the right ventricle into main pulmonary artery and angiogram was taken.



Pre-thrombectomy pulmonary angiogram showing cut-off and filling defect in left branch of pulmonary artery (blue solid arrow) without any opacification of intrapulmonary vessels. Normal contrast opacification of right branch of pulmonary artery (yellow solid arrow) and interlobar artery is noted with attenuation of upper lobe segmental branch (yellow arrowheads).

Over the wire, computer assisted vacuum thrombectomy (CAVT) was done with CAT-16 and Lightning-12 aspiration

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catheters (Penumbra) which were advanced in the pulmonary arteries and multiple passes were taken. The initial few passes resulted in some reduction of the clot burden from left branch of pulmonary artery.

Post thrombectomy angiogram showed decent recanalization of the left main pulmonary trunk, however distal perfusion was not adequate. In view of the same, pharmacological thrombolysis was done and a small dose of alteplase (total of 5 mg) was administered after cannulating descending pulmonary artery. Further passes were taken in interlobar arteries afterwards using Lightning-12 catheter which resulted in decent recanalization.

At this point of time, the aspirated clot was examined and suspicion of acute thrombotic with tumoral pulmonary embolism was raised (image on the right). Subsequently, catheter directed thrombolysis was done using multi side hole infusion catheter with infusion at the rate of 1 mg per hour of Alteplase in normal saline along with 1000 U heparin per hour for 15 hours. The aspirated clot was sent for histopathological evaluation.



A: Fluoroscopic image showing aspiration catheter with wire in left interlobar pulmonary artery (blue arrowheads). B: Post-thrombectomy DSA image showed recanalization initially with stasis in left branch of pulmonary artery and ipsilateral interlobar artery (yellow arrowheads) which could be due to the residual mucin content resulting from the tumoral thrombus.

The patient was shifted back to the ICU and monitored closely. Overnight improvement was noted in vital parameters and oxygen requirement. The next day, the histopathological analysis of the thrombus was done when the patient was taken for check angiogram and removal of the infusion catheter. It confirmed tumoural thrombus containing metastatic adenocarcinoma with extracellular mucin.

Over the next few days, her clinical status improved with progressively decreasing oxygen requirement. After two days of ICU admission, the patient was shifted to the ward and then discharged after a week of the surgery. On follow-up, the patient was clinically stable, and had resumed her normal daily activities without any significant limitation.



Gross specimen of thrombus aspirated post mechanical thrombectomy containing soft tissue component with hemorrhagic / necrotic areas. Histopathological evaluation confirmed its metastatic nature.

Discussion

Presently, there is no reliable modality to differentiate a bland thrombus from tumour thrombus unless there is vascularity of the clot. The presence of a tumour clot within the pulmonary artery may necessitate more aggressive techniques to ensure vessel patency such as stent placement. The management of TPE involves a multidisciplinary approach, incorporating anticoagulation, systemic therapy for the underlying malignancy, and supportive care. Systemic therapy, including chemotherapy, immunotherapy, or targeted therapy, should be tailored to the tumour type and stage with the goal of controlling tumour burden and reducing the risk of further embolic events. In select cases, catheter-directed thrombolysis may be considered as an adjunctive therapy.

The prognosis of TPE is generally poor, reflecting advanced stage of malignancy and the inherent thromboembolic risk associated with cancer. A novel entity described in literature and recognised more frequently in the post-COVID era is de-novo pulmonary embolism arising in absence of deep venous thrombosis. It has been attributed to systemic inflammation, endothelial dysfunction and acute lung injury. Management and prognosis differs for these two clinical entities and therefore, must be distinguished.

Dr. Rohit Agarwal

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Dr. Vritika Bhardwaj

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Spotlight



Dr. Avichala Taxak Associate Consultant Peripheral Vascular and Endovascular Sciences Medanta - Gurugram

For securing Third place at the National ISVIR (Indian Society of Vascular and Interventional Radiology) Mid Term Complication Meet 2024, for presenting an Endovascular Salvage in a Complex Ch-EVAR (Endovascular Aortic Repair with 3-vessel chimney).



CONGRATULATIONS

Dr. Amit Agarwal (Director), Dr. Roma Pradhan (Associate Director) & the entire Team of Endocrine and Breast Surgery for being at the forefront of breast cancer care in the region.

Welcome Onboard



Dr. Neeraj Rastogi

Director - Radiation Oncology Medanta - Lucknow

A renowned oncologist with a career spanning over 3 decades, Dr. Rastogi has been instrumental in establishing comprehensive cancer treatement centres across India. His expertise is treating a wide range of cancers, including urogenital, hepatobiliary, gastrointestinal, breast, cervical, head and neck cancers.





Dr. Atahar Jamal

Associate Director - Critical Care Medicine (PICU) Medanta - Lucknow

A paediatric critical care specialist, he has trained in critical care nephrology, neurology, postoperative care of congenital heart diseases, ECMO, envenomation, intoxication, burns, trauma, severe and refractory ARDS, tropical infections, noninvasive ventilation, HFOV and APRV ventilation, and MODS.





Dr. Ashish Vilas Ukey

Senior Consultant - Aesthetic, Plastic & Reconstructive Surgery Medanta - Lucknow

With 12 years of experience, he is proficient in performing a wide range of aesthetic, reconstructive maxillofacial surgeries, and hair transplantation.





Dr. Sujeet Kumar

Consultant - Haemato Oncology & Bone Marrow Transplant Medanta - Lucknow

He has done more than 100 BMT (autologous and allogenic), worked as principal investigator in multinational trials on Chronic Myeloid Leukemia therapy, and published research papers on haematology (benign and malignant), and BMT.



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Dr. Jitendra Kumar Singh

Associate Consultant - Neuro Anesthesia and Critical Care Medanta - Patna

Trained in general anaesthesia with difficult airway intubation (fiberoptic intubation), tele-anaesthesia, USG-guided block for surgical anaesthesia, central venous line, arterial cannulae, HD catheter insertion, TPI, epidural anaesthesia, PICC line insertion, pain management, monitored anaesthesia care (MAC), critical care management and ICU procedures.





Dr. Kailash Mohitey

Consultant - Internal Medicine Medanta - Ranchi

Specialist in management of diabetes, hypertension, and inflammatory bowel diseases.





Dr. Anshul Agrawal Associate Consultant - Kidney and Urology Medanta - Indore

Trained in onco-surgery of kidney, prostate, urinary bladder and adrenal gland; minimally invasive surgeries for kidney and ureteric calculi and prostate (PCNL, Mini-PCNL, flexible RIRS, URSL Bipolar TURP) with LASER; DVIU and urethroplasty; TRUS-guided biopsy; TRUS aspiration. Experience in laparoscopic nephrectomy, adrenalectomy and pyeloplasty. Trained in vascular access surgeries (AV- Fistula) for chronic kidney disease, and renal transplantation.



Dr. Mala Sinha

Associate Consultant - Gynaecology and Gynaeoncology Medanta - Patna

Specialist in cytoreductive surgery and HIPEC, Dr. Sinha is trained in laparoscopic and robotic surgery. Her areas of focus include preventive gynaecology (cervical cancer screening, post menopausal health), pregnancy and cancer, fertility preserving treatments, vulva dermatosis, vulval surgery, recurrent endometriosis and radical hysterctomy.



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