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Fatal Bleeding due to Segmental Mediolysis of Inferior Vena Cava

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Abstract

This is the first report of a fatal bleeding caused by segmental mediolysis of the inferior vena cava (IVC) at the transition through the diaphragm in a 74 year old female patient. As evaluated by the missing vital reaction, the abdominal bleed occurred three days after the placement of 2 XTW clips reducing a grade III-IV tricuspid insufficiency to a grade II. In the immediate vicinity of the rupture, an acute thinning and ultimately a segmental loss of the smooth muscle layer without any sign of repair, inflammation or necrosis is present. In the surrounding area, individual venues as well as single arterioles show segmental changes in their muscular layer being previously mentioned as defect in the literature. The patient suffered from a long history of cardiac insufficiency due to infarction involving the anterior papillary muscle of the left ventricle as well as degenerative valve disease of the aortic and mitral valve. 6 weeks prior to the TriClip placement, a MitraClip had been placed successfully using the same access via the IVC to the left ventricle without any complication, making any consideration of a vascular leak at the diaphragmatic site even more unlikely. Changes in hemodynamics following a reduced tricuspid regurgitation (now grade II) may have been the reason for an increased wall pressure and the observed rise of transaminases ultimately leading to the rupture of the IVC wall. Thus, in cases of suspected segmental mediolysis, venous vessels should also be considered as source of an acute abdominal bleeding.

Introduction

Segmental mediolysis, an overall rare condition, was first defined by Slavin et al. in 1976. 1 Since it has been primarily described in arterioles and medium-sized splanchnic branches of the aorta along with renal, carotid, cerebral, and coronary arteries, it was called segmental arteriolar mediolysis (SAM) [references in 2 table 2; 3]. The clinical characteristics of SAM have been summarized as follows 4, 5, 6, 7, 8, 9: (1) middle-aged and elderly people, (2) onset due to sudden intra-abdominal bleeding, (3) no underlying diseases, such as inflammatory changes or arteriosclerotic changes, and (4) irregular dilation and narrowing of arteries on angiography. Pathologically, an injurious phase with vacuolization and lysis of smooth muscle cells of the outer media and separation of the adventitia has been differentiated from a reparative phase with granulation tissue and healing fibrosis. 9

In contrast, similar alterations of vessels and even larger veins have only been mentioned as lesions observed alongside segmental arteriolar mediolysis without further clinical consequences reported, such as subendothelial vacuole formation, the deposition of acid mucopolysaccharide in the wall, and small foci of nodular intimal proliferation. 10, 11

Functional tricuspid regurgitation (TR) is the most common form of TR and is due to dilation of the tricuspid valve (TV) annulus from right ventricle or right atrial remodeling. The most common etiologies for functional TR are left

heart failure, aortic or mitral valve disease, or pulmonary disorders with pulmonary hypertension. 12 Women are 4.3 times more likely to be affected with TR than men. 12 In the past, the function of the tricuspid valve has been considered by the medical community to be significantly less important than the function of the aortic and mitral valves. Moreover, severe TR was previously thought to be well tolerated by patients who were treated with medical management. The current thinking about the tricuspid valve has significantly changed, and TR has been recognized as a disease entity with independent prognostic implications. 13, 14, 15 Irrespective of left ventricular function and pulmonary hypertension, TR is associated with increased morbidity and mortality, partly due to the development of right heart failure 16, 17 , and decreased survival over four years regardless of the left ventricle (LV) ejection fraction, right ventricle (RV) size, or pulmonary artery pressure. 18 As a consequence, cardiothoracic surgeons and interventional cardiologists have begun focusing on tricuspid valve repair or re-placement. Recent studies 19, 20 have shown that the effect of transcatheter edge-to-edge tricuspid valve repair for severe tricuspid regurgitation is associated with a reduction of hospitalization for heart failure and improved clinical outcomes. Procedural success was achieved in 72% of 3-leaflet TV. 21 In particular, the TriClip system appears to be safe and effective at reducing tricuspid regurgitation by at least one grade. 22 Compared to conservative management, an early and successful surgical or transcatheter intervention improved 2-year survival in patients at low and, to a lower extent, intermediate TRI-SCORE; however, no benefit was observed in the high TRI-SCORE category . 23, 24

Here, we report here an unusual case of a segmental mediolysis of the inferior vena cava leading to rupture and fatal bleeding at the transition through the diaphragm three days after a repair of TR by cardiologic intervention of successfully placing 2 XTW clips.

Case Report

A 74 year old female patient was admitted to the hospital in cardiac and multi-organ failure (anuria, metabolic/lactate acidosis). Her past history is characterized by multiple cardiac events being caused by an arterial hypertension, such as: i) coronary three vessel arteriosclerosis treated with a right coronary artery (RCA-) stent after a successful drugeluting balloon (DEB-) - dilatation of the RCA; ii) medium grade aortic stenosis (area 1,2cm2); iii) high grade mitral insufficiency (III-IV in March 2023) treated successfully with a MitraClip 6 weeks prior to this admission, leaving a remaining mitral insufficiency of grad I-II; iv) a cardiac insufficiency (New Year Heart Association, NYHA, III; ejection fraction, EF, of 30%; pro- B-type-natriuretic peptide (BNP) of 17583 in February 2023); v) an ischemic and a tachy-cardiomyopathy vi) an atrial fibrillation with a normo-frequent transmission (78/min), vii) a postcapillary pulmonary hypertension, viii) high grade tricuspid insufficiency with a significant coaptation defect (III-IV), and ix) chronic renal insufficiency (estimated glomerular filtration rate, eGFR of 31ml/min1.73m2: Kidney Disease: Improving Global Outcome (KDIGO) stage G3bA1: moderately to severely decreased GFR and albuminuria <30mg/g, mildly increased). On her admission (April 10), a remaining pump function (heart insufficiency with preserved left ventricular ejection fraction: HFpEF), an improved EF of 56% and a CHA2DS2-Vasc-Score of 5 (chronic heart failure, CHF; age, coronary heart disease, chronic heart insufficiency, arterial hypertension, Diabetes mellitus; female) were diagnosed.25 The patient was anticoagulated with a reduced dose of Eliquis (due to borderline GFR, and low body weight) and treated with antiarrythmic medication (Amiodarone, Metoprolol). Temporarily, a hemodynamic and respiratory stabilization was achieved and the patient received the placement of two XTW clips on April 10, reducing the degree of tricuspid insufficiency to a grade II. On April 12, the patient's health deteriorated leading to cyanosis and asystole. Hemodynamic stability could only be achieved by increasing the dosage of catecholamines. At that point, the systolic pump function was moderately decreased, both atria dilated, and a highly reduced right ventricular pump function (tricuspid annular plane systolic excursion, TAPSE, 0.6cm) was detected. Transaminases increased (LDH 2499 U/ml; GOT 1156 U/ml), and the patient was anemic (Hb from initially 15.2 [on April 10] to 6.9 g/d [on April 12]; Ery count from initially 5.08 x 106 down to 2.27 x 106). The patient' s health deteriorated rapidly in spite of all therapeutic measures initiated and died in the morning of April 13.

Autopsy presentation

The autopsy

The abdomen showed 2.2 liters of partially liquid, partially clotted blood spreading sheet-like in a 3mm thick layer over the liver and upper abdominal organs. The only visible site of bleeding was an area of 5cm diameter at the abdominal site of the diaphragm surrounding the passage of the inferior vena cava immediately prior to its entrance in the right atrium. Earlier segments of this vein, and other larger venous/arterial vessels in the abdomen did not show any signs of damage to the endothelial layer or any bleeding. The heart showed a left ventricular hypertrophy, weighing 455g (patient's expected norm was 350g). The left ventricle was moderately dilated. The MitraClip had divided the sclerosed mitral valve in two slit-like orifices of 0.4 and 0.3cm. The anterior papillary muscle was highly scarred. The aortic valve had calcified cusps with a circumference of 6.5cm. The right ventricle was dilated, the TriClips coapted the non-sclerosed leaflets. Both atria were massively dilated and showed endocardial fibrosis but no signs of damage or hemorrhage, no thrombus formation in the auricles. The

entrance of the upper and lower vena cava was open and undamaged.

Histological findings

The microscopic examination was performed after longitudinal dissection of the hemorrhagic area of the diaphragm (Figure 1b, 1c). This area revealed a rupture of the IVC wall with disintegration of the adventitia. Immune histologic analysis of the smooth muscle layer (Figure 2) showed a thinning and regional disappearance (Figure 2a, 2d) compared to normal (Figure 2c). Regionally, a phlebosclerosis of the intimal layer was observed. To analyze any vital reaction, macrophages, neutrophils, and the endothelial layer as well as erythrocyte reabsorption by macrophages (iron loading) were studied. Only individual macrophages (CD68) without iron loading (Berlin blue reaction) could be observed. An initial chemotaxis of neutrophiles was identified (CD15). These results support the interpretation of a bleeding as a late event prior to death, and timewise unrelated to the placement of the TriClip. Single arterioles showed a subendothelial vacuolization of smooth muscle cells and thinning of the media (Figure 3a). Individual venules showed a hemorrhagic distortion of the destroyed muscular media (Figure 3b).

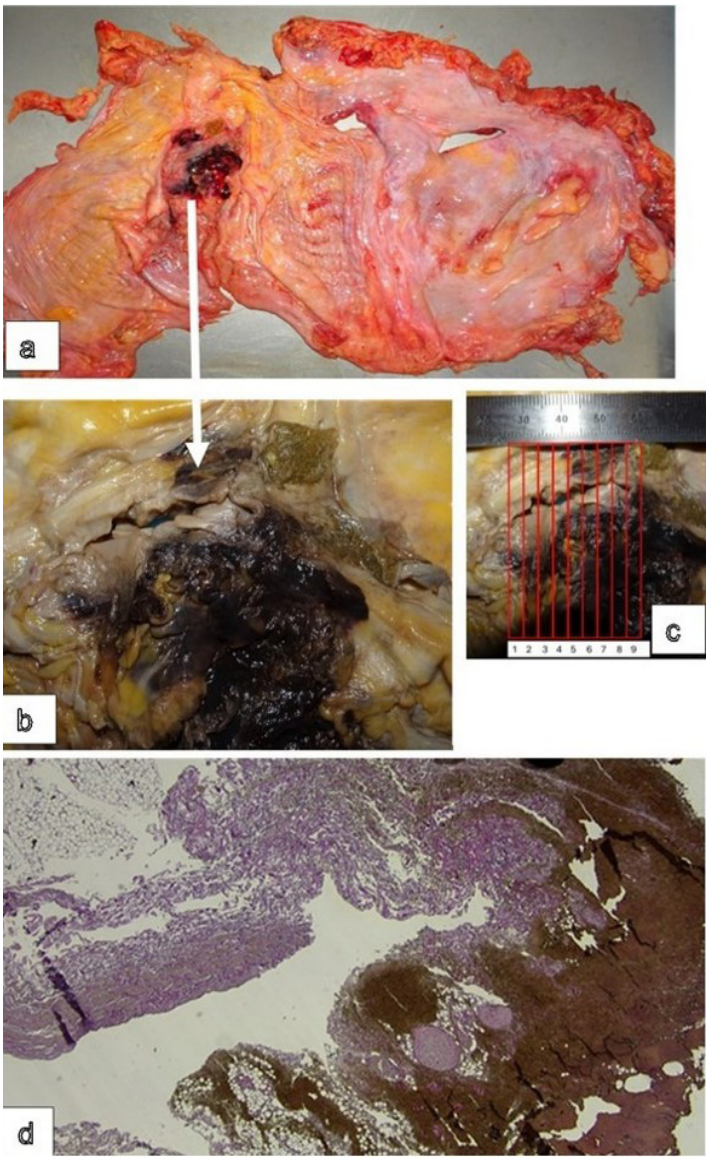

Figure 1: *a) abdominal side of diaphragm with bleeding at transition of IVC; fresh specimen; b) transition of IVC, formalin-fixed; c) method of investigation by cross-sectioning; d) section plane showing disruption of IVC wall*

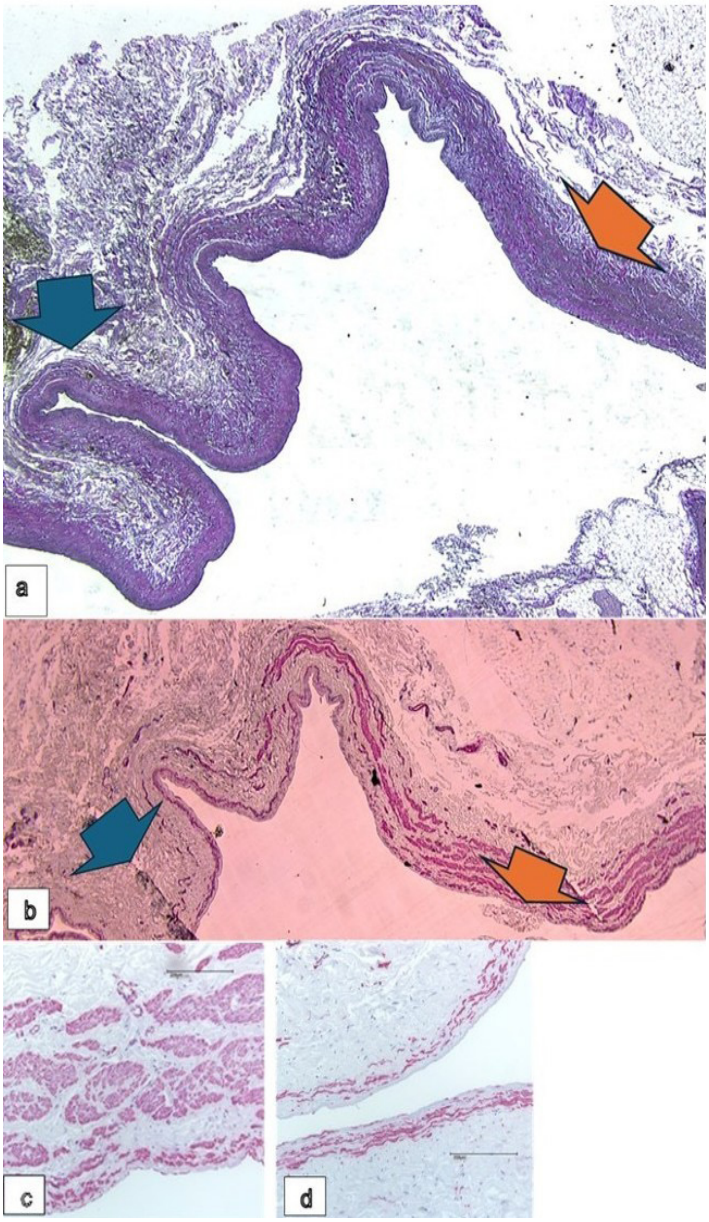

Figure 2: *a) section plane of IVC showing normally wide (yellow arrow) vs. highly reduced (blue arrow) muscular wall; b) identical section plane with immune histologic demonstration of smooth muscle actin (SMA); normal muscular layer (yellow arrow) vs. highly reduced muscular layer; c) SMA normal wall; d) SMA highly reduced layer. Barr = 200µm*

The infarcted area of the heart was fibrosed affecting the anterior wall and the anterior papillary muscle. The liver was characterized by a network of fulminant necrosis in zone 2 and 3 sparing only liver cells in zone 1 surrounding Glisson's triangles.

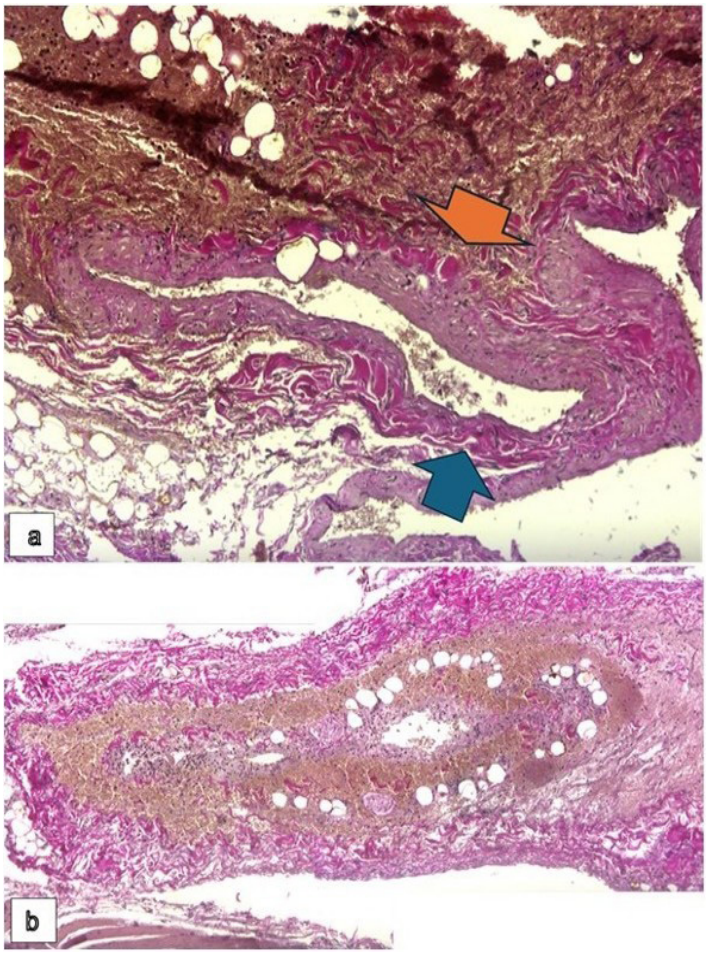

Figure 3: *a) arteriole with vacuolization and normal (yellow arrow) vs. reduced (blue arrow) muscular layer ; b) venule with hemorrhagic destruction of muscular layer*

Discussion

By criteria of the Tricuspid Valve Academic Research Consortium (TVARC) 26, the presented case is characterized by a severe TR regurgitation (TAPSE 6mm) 26; secondary to a mitral valve regurgitation which resulted in a fatal hemorrhage classified as subacute bleeding (24 h > x > 30d), being confirmed by autopsy (type 5b) 26 due to a vascular rupture of the inferior vena cava at the transition through the diaphragm. The unusual aspect of this case is the newly reported segmental mediolysis of the inferior vena cava as cause of a fatal hemorrhage.

Segmental mediolysis can occur in both, the arterial and the venous system. The vast majority of reports refer to the arterial system (see references in 2, 3). Slavin 1 originally described for these lesions four different types of alterations: (1) mediolysis, (2) a tearing separation of the outer media from adventitia, (3) arterial gaps, and (4) a florid reparative response that replaces zones of mediolysis and fills areas of medial adventitial separation. The reparative process may make these lesions indistinguishable from common types of fibromuscular dysplasia (FMD). 27, 28

In contrast, of involvement of the venous systems has been mentioned incidentally. 10,11 Alterations in medium and larger-sized veins reported have been a vacuolar change with lysis leading to apparent separation of residual muscle bundles but no rupture or fatal bleeding.

This case report demonstrates lesions found at the rupture site of the IVC, which are much more profound than the ones previously observed in any vein (Figure 1 and 2). The segmental mediolysis caused areas of an almost complete disappearance of the muscular layer (Figure 2a, 2b, 2d; versus normal: Figure 2c). There is an advanced dissection of the adventitial layer by the occurring hemorrhage.

In addition, there are lesions found in individual arterioles and in venules surrounding the rupture site similar to the ones reported before (Figure 3a, 3b)10. The adventitial alteration, however, is more pronounced than previously reported: the adventitial connective tissue is regionally dissected and subsequently lost, denuding the remaining media. In addition, these alterations are only observed in the vicinity of the bleeding site, and are not found in any other tissue analyzed by autopsy/histology.

Other diseases mimicking mediolysis as reported previously could be excluded. 6, 8, 28, 29, 30 However, since there is a history of a cardiologic intervention (placement of MitraClip) predating the occurrence of rupture, additional mechanisms may have contributed to a progressive pressure to this region of an altered IVC wall, ultimately leading to the rupture. The delayed reaction with bleeding on day 2/3 after the intervention and its accompanying rise of transaminases is not consistent with a direct interventional event but rather with a combination of different mechanisms, such as i) the enlargement of the left atrium (atrial cardiomyopathy), 31 blocking the venous return through protrusion of the atrial septum to towards the entrance of the IVC; ii) the preexisting, circumscribed phlebosclerosis; iii) the reduction of mitral valve and tricuspid valve regurgitation with subsequent increased atrial congestion, and finally iv) acute vasospasms due to inappropriate reactions to catecholamine or endothelial dysfunction, 8,11 while even endothelin-1 as surrogate vasoconstrictor may play a role.32

Is there a potential connection between the degree of mitral valve regurgitation influencing TR? Percutaneous mitral valve repair (PMVR) using the MitraClip device was observed to improve functional TR severity in approximately one third of patients, particularly in those who experience a significant systemic pulmonary artery pressure (SPAP) reduction after the procedure 33 but its effect on RV dimensions and function, tricuspid annular geometry and TR has not been extensively investigated as yet. 34 Thus, Matsunaga and Duran have reported functional TR occurring on structurally normal tricuspid valves frequently observed

in patients with mitral valve regurgitation.35 Furthermore, TR has been found to progress after mitral valve surgery particularly in ischemic or dilatative cardiomyopathy. 36 Almost 50% of patients present with TR despite successful surgery lowering the pulmonary pressure. 35 Furthermore, the correction of tricuspid regurgitation can paradoxically be worsened by the repair of mitral regurgitation although hemodyamics are improved, as Toselli et al reports. 37 Thus, in analogy to our case, the successful repair of mitral valve regurgitation could have resulted in an increased postcapillary hypertension and aneurysmal dilatation of the left atrium, causing an increased pulmonary pressure and subsequent worsening of TR. Repairing the TR with a decreased regurgitation from grade III-IV to a grade II will have consequently reduced blood flow and caused an increased the pressure in the IVC. While under normal conditions the function of the IVC as capacity vessel would have been able to handle the increased blood volume, the mediolytic vessel wall was obviously insufficient to withstand the pressure and ruptured, leading to a fatal bleed enhanced by the (albeit lower dose of) anticoagulant. Since the degree of regurgitation was so high, this pathomechanism may have played an additional negative role in this case.

Since there is no known genetic trait or disease associated with a segmental mediolysis, the question remains which clinical sign or symptom could be taken as warning sign of its possible existence. For arterial lesions, Slavin et al. 38 reported six potential angiographic presentations (a) arterial dilatation, (b) single aneurysm, (c) multiple aneurysms, (d) dissecting hematomas, (e) arterial stenosis, and (f) arterial occlusions. From the pathologic process being involved, it seems probable, that arterial dilatation is caused by lytic loss of medial muscle; dissections may develop from reparative alterations, while thrombi formation leads to stenosis / occlusion. Not surprisingly, the initial common radiologic findings which are consistent with histologic analyses are arterial dilatation, aneurysms and occlusion, while dissections and stenotic lesions are considered late lesions. In a review of 85 cases of SAM, Shenouda et al noticed that sixty-nine percent of cases were diagnosed histologically of which 24% by means of autopsy only. 3 SAM mortality ranged by 26%. When treated with open surgery, there was a 9% mortality rate, while endovascular intervention most commonly by coil embolization of aneurysmatic alteration had a reported success of 88%. While there is no report regarding the frequency or extent of venous mediolysis, the number of such alterations can be regarded higher than expected considering the simultaneous presentation of both arterial and venous lesions in several reported cases as well as in this present case. Thus, in cases of segmental mediolysis unexpected bleeding may be caused not only by arterial lesion but also by concomitantly occurring venous lesions.

Conclusion

In summary, we report the first case of a vascular mediolysis affecting the IVC at the critical passage through the diaphragm. For future cases, the spectrum of segmental mediolysis can be expected to affect both, large and small arteriolar and venous vessel alike. Potentially, different methods of imaging such as ultrasound or angiography may help to identify mediolytic lesions in order prevent cases of fatal bleeding.

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Compliance with ethical standards: The case was originally considered a legal one due to unknown cause of death. Since the deceased had no next of kin, and an external cause of death was highly unlikely, the case was transferred to Pathology with orders to send copies of the report to forensics. Since human tissue was investigated, procedures were followed as outlined in accordance with ethical standards, formulated in the Helsinki Declaration 1975 (and revised in 1983). The CARE protocol for case reports was followed.

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Conflict of interest: The authors declare that they have no conflict of interest.

References

1. Slavin RE, Gonzalez-Vitale JC. Segmental mediolytic arteritis: a clinical pathologic study. Lab Invest. 1976; 35(1):23-9.
2. Feld K, Mellin W, Melzer B , Rothschild MA, Fries JWU. Fatal hemorrhage after tonsillectomy. Rechtsmedizin 2002; 32, 52–57.
3. Shenouda M, Riga C, Naji Y, Renton S. Segmental arterial mediolysis: a systematic review of 85 cases. Ann Vasc Surg. 2014; 28(1):269-77
4. Peng KX, Davila VJ, Stone WM, Shamoun FE, Naidu SG, McBane RD, et al. Natural history and management outcomes of segmental arterial mediolysis. J Vasc Surg. 2019;70:1877–86.
5. Ko M, Kamimura K, Ogawa K, Tominaga K, Sakamaki A, Kamimura H, et al. Diagnosis and management of fibromuscular dysplasia and segmental arterial mediolysis in gastroenterology field: A mini-review. World J Gastroenterol. 2018 Aug 28;24(32):3637-3649.
6. Michael M, Widmer U, Wildermuth S, Barghorn A, Duewell S, Pfammatter T. Segmental arterial mediolysis: CTA findings at presentation and follow-up. AJR Am J Roentgenol. 2006 Dec;187(6):1463-9.
7. Kalva SP, Somarouthu B, Jaff MR, Wicky S. Segmental arterial mediolysis: clinical and imaging features at presentation and during follow-up. J Vasc Interv Radiol. 2011 Oct;22(10):1380-7.
8. Baker-LePain JC, Stone DH, Mattis AN, Nakamura MC, Fye KH. Clinical diagnosis of segmental arterial mediolysis: differentiation from vasculitis and other mimics. Arthritis Care Res (Hoboken). 2010 Nov;62(11):1655-60.
9. Minten L, Messiaen P, Van der Hilst J. Acute abdominal pain: a challenging diagnosis. Acta Gastroenterol Belg. 2022 Oct-Dec;85(4):646-647.
10. Shintaku M, Ohta M, Tatsumi K, Yutani C. Segmental arterial mediolysis of the middle colic artery: report of a case with

special reference to lesions of small arteries and veins. Int J Clin Exp Pathol. 2021; 14(4):508-513.
11. Slavin RE, Inada K. Segmental Arterial Mediolysis With Accompanying Venous Angiopathy: A Clinical Pathologic Review, Report of 3 New Cases, and Comments on the Role of Endothelin-1 in Its Pathogenesis. International Journal of Surgical Pathology 2007;15(2):121-134.
12. Badano L, Muraru D and Enriquez-Sarano M. Assessment of functional tricuspid regurgitation. Eur Heart J 2013; 34: 1875-1885.
13. Wang N, Fulcher J, Abeysuriya N, McGrady M, Wilcox I, Celermajer D, et al. Tricuspid regurgitation is associated with increased mortality independent of pulmonary pressures and right heart failure: a systematic review and meta analysis. Eur Heart J. 2019 Feb 1;40(5):476-484.
14. Bartko PE, Arfsten H, Frey MK, Heitzinger G, Pavo N, Cho A, et al. Natural History of Functional Tricuspid Regurgitation: Implications of Quantitative Doppler Assessment. JACC Cardiovasc Imaging. 2019 Mar;12(3):389-397.
15. Benfari G, Antoine C, Miller WL, Thapa P, Topilsky Y, Rossi A, et al. Excess Mortality Associated With Functional Tricuspid Regurgitation Complicating Heart Failure With Reduced Ejection Fraction. Circulation. 2019 Jul 16;140(3):196-206.
16. Topilsky Y, Maltais S, Medina Inojosa J, Oguz D, Michelena H, Maalouf J, et al. Burden of Tricuspid Regurgitation in Patients Diagnosed in the Community Setting. JACC Cardiovasc Imaging. 2019 Mar;12(3):433-442.
17. Chorin E, Rozenbaum Z, Topilsky Y, Konigstein M, Ziv-Baran T, Richert E, et al. Tricuspid regurgitation and long-term clinical outcomes. Eur Heart J Cardiovasc Imaging. 2020 Feb 1;21(2):157-165.
18. Lurz P, Stephan von Bardeleben R, Weber M, Sitges M, Sorajja P, Hausleiter J, et al. TRILUMINATE Investigators. Transcatheter Edge-to-Edge Repair for Treatment of Tricuspid Regurgitation. J Am Coll Cardiol. 2021 Jan 26;77(3):229-239.
19. Orban M, Rommel KP, Ho EC, Unterhuber M, Pozzoli A, Connelly KA, et al. . Transcatheter Edge-to-Edge Tricuspid Repair for Severe Tricuspid Regurgitation Reduces Hospitalizations for Heart Failure. JACC Heart Fail. 2020 Apr;8(4):265-276.
20. Sorajja P, Whisenant B, Hamid N, Naik H, Makkar R, Tadros P, et al. TRILUMINATE Pivotal Investigators. Transcatheter Repair for Patients with Tricuspid Regurgitation. N Engl J Med. 2023 18;388(20):1833-1842.
21. Kitamura M, Kresoja KP, Besler C, Leontyev S, Kiefer P, Rommel KP, et al. Impact of Tricuspid Valve Morphology on Clinical Outcomes After Transcatheter Edge-to-Edge Repair. JACC Cardiovasc Interv. 2021 26;14(14):1616-1618.
22. Nickenig G, Weber M, Lurz P, von Bardeleben RS, Sitges M, Sorajja P, et al. Transcatheter edge-to-edge repair for reduction of tricuspid regurgitation: 6 month outcomes of the TRILUMINATE single-arm study. Lancet. 2019 Nov 30;394(10213):2002-2011.
23. Anguita-Gámez M, Giraldo MA, Nombela-Franco L, Eixeres Esteve A, Cuerpo G, Lopez-Menendez J, et al. Validation of the TRI-SCORE in patients undergoing surgery for isolated tricuspid regurgitation. Heart. 2023 Aug 24;109(18):1401-1406.
24. Dreyfus J, Galloo X, Taramasso M, Heitzinger G, Benfari G, Kresoja KP, et al. TRIGISTRY investigators. TRI-SCORE and benefit of intervention in patients with severe tricuspid regurgitation. Eur Heart J. 2024 Feb 21;45(8):586-597.
25. Lip GY, Frison L, Halperin JL, Lane DA. Identifying patients at high risk for stroke despite anticoagulation: a comparison of contemporary stroke risk stratification schemes in an anticoagulated atrial fibrillation cohort. Stroke. 2010 Dec;41(12):2731-8.
26. Hahn RT, Lawlor MK, Davidson CJ, Badhwar V, Sannino A, Spitzer E, et al. TVARC Steering Committee. Tricuspid Valve Academic Research Consortium Definitions for Tricuspid Regurgitation and Trial Endpoints. J Am Coll Cardiol. 2023 Oct 24;82(17):1711-1735.
27. Slavin E, Saeki K, Bhagavan B, Maas AE. Segmental arterial mediolysis: a precursor to fibromuscular dysplasia? Mod Pathol 1995; : 287-294.
28. Lie JT. Segmental mediolytic arteritis. Not an arteritis but a variant of arterial fibromuscular dysplasia. Arch Pathol Lab Med 1992;116:238–241.
29. Brinza EK, Gornik HL. Fibromuscular dysplasia: Advances in understanding and management. Cleve Clin J Med. 2016 Nov;83(11 Suppl 2):S45-S51.
30. Molloy ES, Langford CA. Vasculitis mimics. Curr Opin Rheumatol 2008;20:29– 34.
31. Goette A, Kalman JM, Aguinaga L, Akar J, Cabrera JA, Chen SA, et al. EHRA/HRS/APHRS/SOLAECE expert consensus on atrial cardiomyopathies: Definition, characterization, and clinical implication. Heart Rhythm. 2017;14(1):e3-e40.
32. Hunley TE, Kon V. Update on endothelins - biology and clinical implications. Pediatr Nephrol. 2001 Sep;16(9):752-62.
33. Frangieh AH, Gruner C, Mikulicic F, Attinger-Toller A, Tanner FC, Taramasso M, et al. Impact of percutaneous mitral valve repair using the MitraClip system on tricuspid regurgitation. EuroIntervention. 2016 8;11(14):e1680-6.
34. Ohno Y, Attizzani GF, Capodanno D, Cannata S, Dipasqua F, Immé S, et al. Association of tricuspid regurgitation with clinical and echocardiographic outcomes after percutaneous mitral valve repair with the MitraClip System: 30-day and 12-month follow-up from the GRASP Registry. Eur Heart J Cardiovasc Imaging. 2014 Nov;15(11):1246-55.
35. Matsunaga A, Duran CM. Progression of tricuspid regurgitation after repaired functional ischemic mitral regurgitation. Circulation. 2005;112: 1453-1457.
36. De Bonis M, Lapenna E, Sorrentino F, La Canna G, Grimaldi A, Maisano F, et al. Evolution of tricuspid regurgitation after mitral valve repair for functional mitral regurgitation in dilated cardiomyopathy. Eur J Cardiothorac Surg. 2008; 33:600 - 606.
37. Toselli M, Benatti G, Mele D, Rapezzi C, Giannini F. Approccio transcatetere nei pazienti affetti da insufficienza mitralica severa e concomitante insufficienza tricuspidale severa [Transcatheter approach of concomitant mitral and tricuspid regurgitation]. G Ital Cardiol (Rome). 2021 Sep;22(9 Suppl 1):9S- 20S. Italian.
38. Slavin RE. Segmental arterial mediolysis: course, sequelae, prognosis, and pathologic radiologic correlation. Cardiovasc Pathol. 2009 Nov-Dec;18(6):352-60.

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