Virchows Arch (2000) 437:560–563
DOI 10.1007/s004280000279

CASE REPORT

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Cardiac vein thrombosis and haemorrhagic myocardial necrosis; report of a case with review of the literature

Received: 25 May 2000 / Accepted: 22 June 2000 / Published online: 7 September 2000

Abstract A 29-year-old woman, addicted to heroin since the age of 15 years, presented with a 4-day history of acute inspiratory chest pain, dyspnoea and vomiting associated with hypoventilation. She died 3 h after admission to the intensive care unit in spite of active resuscitative measures. The main autopsy findings were limited to the heart, which showed widespread cardiac vein thrombosis, and both ventricles and the atria were associated with multiple areas of haemorrhagic myocardial necrosis. We review the literature of this uncommon pathological entity and discuss its possible pathogenesis.

Keywords Thrombosis · Cardiac veins · Coronary sinus · Thebesian veins · Haemorrhagic myocardial necrosis

Introduction

Thrombosis of the venous system of the heart is rare. It may be an isolated finding or occasionally associated with thrombosis of the coronary sinus [4, 7, 10, 11, 15]. The condition has also been documented in cases of myocardial infarction, cardiac transplantation, infections and tumours [5, 7, 10, 14, 17]. We describe the case of diffuse myocardial venous thrombosis associated with haemorrhagic myocardial necrosis and review the literature.

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Case report

Clinical summary

A 29-year-old mother who had given birth to three children, one of whom died 3 years ago, was admitted to the intensive care unit complaining of sudden onset chest pain and dyspnoea. Her symptoms had begun 4 days previously when she experienced acute inspiratory chest pain, dyspnoea and upper abdominal pain associated with vomiting. She was treated before admission at another centre with nonsteroidal antiinflammatory drugs and omeprazole without improvement. She was known to have been addicted to heroin since the age of 15 years, but has been on methadone, 80 mg/day for 2 weeks. Since the age of 12 years, she smoked one package of cigarettes daily (17 years of one pack per day) and was not on oral contraceptives. Over the last 4 years, she had been experiencing episodes of dyspnoea which, 3 years ago, necessitated her hospitalization and was evaluated then for pulmonary embolism. Negative D-dimers excluded a thrombotic event. Upon admission, her temperature was 36°C, blood pressure 75/50 mmHg, pulse irregular at 93/min, with a respiration rate of 16 and an oxygen saturation of 90%. She was restless and sweating profusely. Auscultation revealed a third heart sound on the left sternal border, crepitant rales on the right lung field and hypoventilation. There was mild epigastric tenderness but no guarding effect. Her haemoglobin was 130 g/l and her white cells were 17.3 g/l. The test for human immunodeficiency virus (HIV) was negative, and toxicological examinations revealed only opiate metabolites in the urine. Chest X-rays showed cardiomegaly, while transthoracic echocardiography was consistent with left ventricular wall thickening and global dysfunction of both ventricles. Serum enzymes (creatinine kinase, MB isoenzymes. tropinin I) were all markedly elevated, indicating myocardial damage. The electrocardiogram was consistent with acute myocardial infarction. Fluids were administered in the form of 2000 ml NaCl and 500 ml plasmasteril i.v. together with dopamine and metoclopramide. While awaiting transfer to a University Coronary Care Unit, 3 h later, she suddenly presented a pulseless electrical activity. Active resuscitative measures were unsuccessful.

Results

Autopsy

Gross findings

Postmortem was performed 12 h after death. The body weighed 64 kg for a height of 163 cm (BMI 24.0). There

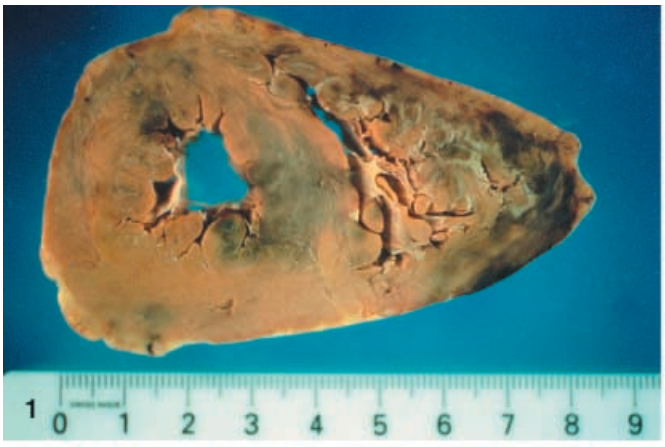
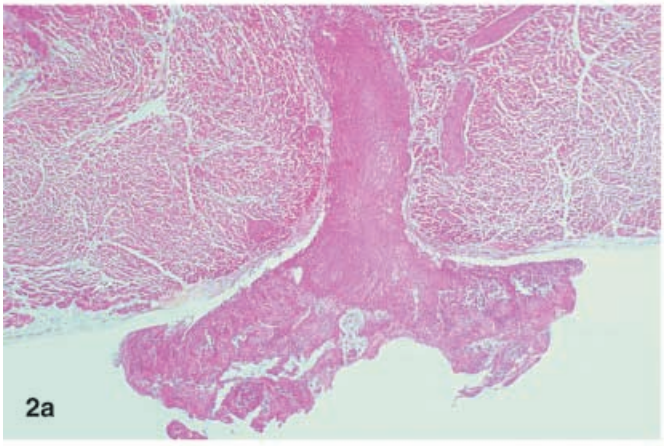
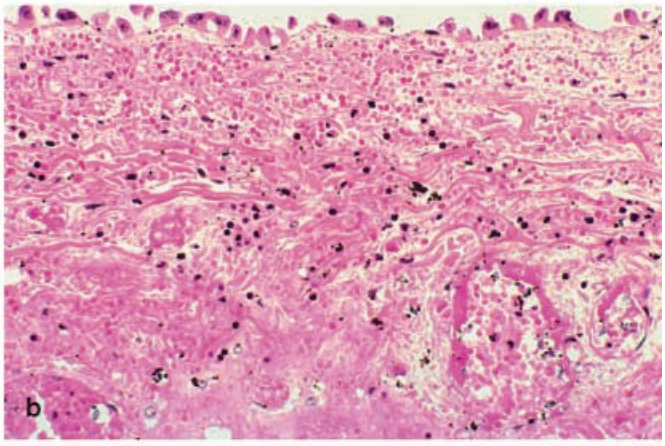


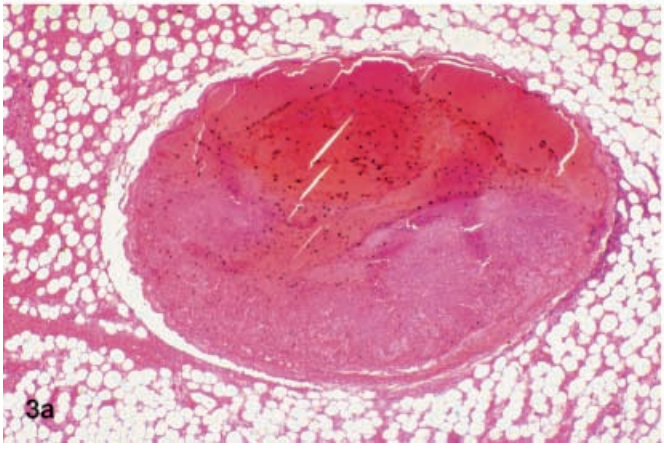
Fig. 1 Transversal section of the heart showing mural thrombosis of both ventricular cavities, moderate ventricular wall hypertrophy and bilateral patchy transparietal brownish-grey areas

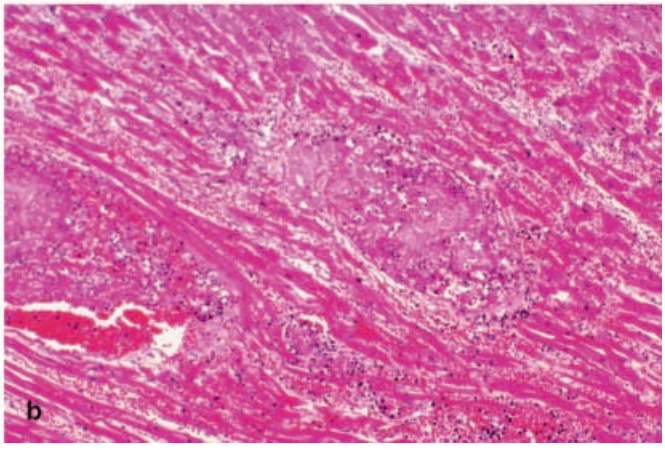
Fig. 2 a Right ventricular endocardial thrombosis extending into the dilated thebesian vein. Note the interstitial myocardial oedema associated with dispersed polymorphonuclear leucocytic infiltration. (haematoxylin and eosin; ×11.2) **b** Diffuse thrombosis of the dilated myocardial veins with phlebitis in places within the myocardium. Note dissection of the myocardial fibres. (haematoxylin and eosin; ×28)

Fig. 3 a Thrombosis of the dilated main right anterior cardiac vein and diffuse haemorrhages in the surrounding fatty tissue. (haematoxylin and eosin; ×11.2) **b** Left ventricular wall showing hyperplasia of the mesothelial cells, multiple small venous thrombosis and interstitial haemorrhages within the epicardial fatty tissue. The arteries are normal. (haematoxylin and eosin; ×70)









were numerous scars on both arms and legs. The heart weighed 400 g, with moderate concentric biventricular hypertrophy. The pericardial cavity contained 150 ml of haemorrhagic fluid, while the epicardium was brilliant with some brownish areas of variable sizes. The heart valves were normal in appearance. The organ was sectioned transversally and these showed several ill-defined, irregular, darkish-red areas. Both ventricles were occupied by mural thrombi which were more important in the right (Fig. 1; magnifications refer to the original slide format). Both atria and their appendages were also thrombosed. On the right, it extended into the pulmonary trunk. The coronary arteries were normal, free of atherosclerosis. The lungs, congested, weighed together, were 1290 g. The cut surface showed extensive anthracosis but otherwise appeared normal. The pulmonary arteries contained recent thrombotic material, mainly in the peripheral branches.

Microscopic findings

Heart. Both ventricles presented extensive mural thrombosis. These were situated predominantly on the right where they englobed the trabeculi and were in continuity with the endocardium in areas.

The thrombi were made up essentially of platelets and some fibrin, associated in areas with various quantities of polymorphonuclear leucocytes accompanied, here and there, by erythrocytes. These thrombi extended into the thebesian veins, completely or partially obliterating their lumen (Fig. 2a). The deeper myocardial veins, for the most part distended, contained similar thrombotic material partially surrounded by erythrocytes, capped by polymorphonuclear leucocytes and eosinophils which infiltrated and extended beyond the venous wall (Fig. 2b). The arteries were congested and somewhat dilated but free of thrombi. There was no arterial lesion. The myocardium of both ventricles presented intramyocardial haemorrhages of variable degrees sometimes separating the myocardial fibres into bundles. In one area, there was a small group of eosinophilic fibres, but no contraction band necrosis or evidence of infarction were observed.

The main epicardial venous trunks and their tributaries were markedly distended and filled with thrombotic material and a mixture of erythrocytes and some polymorphonuclear leucocytes (Fig. 3a). There were widespread haemorrhages in the epicardial fatty tissue attaining both the epicardium and myocardium in places. The epicardial mesothelial cells were hyperplastic (Fig. 3b). Both auricular appendages were thrombosed, accompanied by necrosis of their walls.

The wall of the coronary sinus was oedematous, but its endothelium was intact and there was no thrombosis. The surrounding epicardial fatty tissue, haemorrhagic, contained widespread venous thrombi but no arterial lesions.

Lungs. Numerous sections showed, besides peripherial organizing and recent thromboemboli, areas of distended alveoli filled with pigment-laden macrophages, some of which contained Prussian blue-stained iron particles.

Anatomopathological conclusion

Diffuse cardiac vein thrombosis with haemorrhagic myocardial necrosis, pulmonary thromboembolism and emphysema were observed.

Discussion

Isolated thrombosis of the heart veins is a rare condition. We have collected only seven reported cases from the literature, all of which were incidental findings at postmortem and had led to the death of the patients [4, 7, 15]. One of those presented with thrombosis of a trabecular splenic vein [15]. The clinical symptoms were similar in all and were characterised by sudden onset of chest pain, irradiating to the back, dyspnoea and occasionally vomiting, leading to death within 2–4 days, like the patient presented here. The clinical diagnosis was always illusive, and only in one was it established by subtraction coronary angiography [6]. The condition has also been described in association with infections or sepsis [7, 10, 14, 15], tumours [7] and cardiac transplantation [12, 18]. Coronary vein thrombosis has often been documented in cases of thrombosis of the coronary sinus as a result of inadvertant implantation in/or damage to it during central venous catheterization, especially by way of the left internal jugular vein and more so, in cases with anatomical variations of the coronary sinus [3, 6, 8, 13, 16, 17]. It has also been described in cases of ventriculoatrial shunts [19] or in cases where a pacemaker lead was mistakenly implanted in the coronary sinus [2]. A relatively high percentage of epicardial coronary vein thrombosis has also been documented in association with myocardial infarction, in which more than 30% of the left ventricular mass was involved, in combination with valvular heart disease and a post-attack survival time of at least 24 h [5]. However, isolated cardiac vein thrombosis, other than these conditions, remains an enigma. Some authors have postulated that trauma to the chestwall could be a contributing factor, while others have suggested the possibility of circulatory stasis secondary to the formation of ventricular mural wall thrombosis with extension into the thebesian veins [4, 7].

In the absence of clinical indications, and especially because the cardiac venous thrombosis was an incidental histological finding and there were no lesions which suggested a possible coagulation abnormality, no studies were undertaken to investigate coagulation abnormalities.

Recently, ventricular thrombosis, right or left, has been documented in association with raised titres of anticardiolipid antibodies of the antiphospholipid group of connective tissue disorders [1, 9]. However, to the best of our knowledge, none of those presented with thrombosis of the cardiac veins and/or their tributaries.

Acknowledgements The authors are indebted to Drs A. Sutter and P. Zhang for performing the autopsy, to Drs P. Monnier (La Chaux-de-Fonds) and R. Jenni, PD (Zurich) for the interpretation of the echocardiogram, and to Mrs M. Cox, Mr J. Ribeiro and R. Wyler for preparing the manuscript.

References

1. Bruce D, Bateman D, Thomas R (1995) Left ventricular thrombi in a patient with the antiphospholipid syndrome. Br Heart J 74:202–203
2. Cox JN (1994) Pathology of cardiac pacemakers and central catheters. In: Berry CL (ed) The pathology of devices. Springer, Berlin Heidelberg New York, pp 198–271 (Current topics in pathology, vol. 86)
3. Figuerola M, Tomas M-T, Armengol J, Bejar A, Adrados M, Bonet A (1992) Pericardial tamponade and coronary sinus thrombosis associated with central venous catheterization. Chest 101:1154–1155
4. Flynn JE, Mann FD (1946) The presence and pathogenesis of endocardial and subendocardial degeneration, mural thrombi, and thromboses of the thebesian veins in cardiac failure from causes other than myocardial infarction. Am Heart J 31:757–768
5. Hansen BF (1979) Thrombosis of epicardial coronary veins in acute myocardial infarction. Am Heart J 97:696–700
6. Hazan MB, Byrnes DA, Elmquist TH, Mazzara JT (1982) Angiographic demonstration of coronary sinus thrombosis: a

potential consequence of trauma to the coronary sinus. Cathet Cardiovasc Diagn 8:405–408
7. Lake B (1958) Cardiac vein thrombosis with myocardial hemorrhage. Am Heart J 55:157–160
8. McMichael J, Mounsey JPD (1951) A complication following coronary sinus and cardiac vein catherization in man. Br Heart J 13:397–402
9. O'Hickey S, Skinner C, Beattie J (1993) Life threatening right ventricular thrombosis in association with phospholipid antibodies. Br Heart J 70:279–281
10. Ong ML (1993) Endocarditis of the tricuspid valve associated with congenital coronary arteriovenous fistula. Br Heart J 70: 276–278
11. Philips JB III, Ruiz-Castaneda N, Setzer ES (1981) Coronary sinus thrombosis: a central venous catheter complication. J Pediatr Surg 16:733–734
12. Pomerance A, Stovin PGI (1985) Heart transplant pathology: the British experience. J Clin Pathol 38:146–159
13. Ramsaran EK, Sadigh M, Miller D (1996) Sudden cardiac death due to primary coronary sinus thrombosis. South Med J 89:531–533
14. Ross EM, Macher AM, Roberts WC (1985) Aspergillus fumigatus thrombi causing total occlusion of both coronary arterial ostia, all four major epicardial coronary arteries and coronary sinus and associated with purulent pericarditis. Am J Cardiol 56:499–500
15. Rywlin AM, Greenberg JJ, Gordon HW, Piccinini JC, Lehr D (1968) Hemorrhagic myocardial infarction due to cardiac vein thrombosis. Report of two cases. Am J Cardiol 21:269–272
16. Sheep RE, Guiney WB Jr (1982) Fatal cardiac tamponade. Occurrence with other complications after left internal vein catheterization. JAMA 248:1632–1635
17. Smith WW, Albert RE, Rader B (1951) Myocardial damage following inadvertent deep cannulation of the coronary sinus during right heart catheterization. Am Heart J 42:661–666
18. Urbanova D (1989) Coronary sinus thrombosis in patients after heart transplantation. Cor Vasa 31:231–237
19. Wells CA, Senior AJ (1990) Coronary sinus thrombosis and myocardial infarction secondary to ventriculoatrial shunt insertion. J Pediatr Surg 25:1214–1215