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Behavior of mortality due to aortic dissection in Cuba

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AD: aortic dissection
SARCAP: Automated Registration and Control System of Anatomical Pathology (acronym in Spanish)

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ABSTRACT

Introduction: Aortic dissection is a disease of poor prognosis, with a high mortality rate, even when it is diagnosed early and the adequate surgery is performed.

Objectives: To describe some characteristics of the patients who died due to aortic dissection in Cuba.

Method: A retrospective cross-sectional study was conducted. It included 888 deceased patients with a diagnosis of aortic dissection, who were registered in the Automated Registration and Control System of Anatomical Pathology (SARCAP, for its acronym in Spanish) in Cuba, from 1962 to 2004.

Results: The age groups from 65 to 74 and from 75 to 84 years predominated, with 266 (30.0%) and 210 (23.6%) deaths, respectively. The largest number of deaths [496 (55.9%)] occurred in the first 24 hours. Stanford type A aortic dissection was the most frequent type [535 diagnoses were made (61.1%)]. The most common underlying cause of death was aortic dissection itself (61.6 %) and the most common direct cause of death was hemopericardium (43.9%). The rate of diagnostic agreement was 33.5 % in the underlying cause and 28.8 % in direct cause.

Conclusions: During the 42 years covered by the study, the patients who died from aortic dissection in Cuba predominantly had Stanford type A dissections, were over 55 years of age and had a hospital stay of less than 2 months. The largest number of deaths occurred in the first 24 hours and the rate of diagnostic agreement was low.

Key words: Aortic dissection, Necropsy, Diagnostic agreement, SARCAP

Comportamiento de la mortalidad por disección aórtica en Cuba

RESUMEN

Introducción: La disección aórtica es una enfermedad de pronóstico muy reservado, con una elevada mortalidad, aun cuando se diagnostique precozmente y se realice la intervención quirúrgica adecuada.

Objetivo: Describir algunas características de los fallecidos por disección aórtica en Cuba.

Método: Se realizó un estudio retrospectivo de corte transversal, que incluyó 888 fallecidos con diagnóstico de disección aórtica, incluidos en el Sistema Automatizado de Registro y Control de Anatomía Patológica (SARCAP) en Cuba, desde el año 1962 hasta el 2004.

Resultados: Predominaron los grupos etarios de 65-74 y 75-84 años, con 266 (30,0 %) y 210 (23,6 %) defunciones, respectivamente. El mayor número de muertes [496 (55,9 %)] ocurrió en las primeras 24 horas. La disección aórtica tipo A de Stanford fue la de mayor frecuencia [535 diagnósticos (61,1 %)]. La causa básica de muerte más encontrada fue la propia disección aórtica (61,6 %) y la causa directa, el hemopericárdico (43,9 %). El índice de coincidencia diagnóstica fue de 33,5 % en la causa básica y de 28,8 % en la directa.

Conclusiones: Los fallecidos por disección aórtica en Cuba, durante los 42 años estudiados, tuvieron predominantemente una disección tipo A de Stanford, más de 55 años de edad y una estadía hospitalaria menor de 2 meses. El mayor número de muertes ocurrió en las primeras 24 horas y el índice de coincidencia diagnóstica fue bajo.

Palabras clave: Disección aórtica, Necropsia, Coincidencia diagnóstica, SARCAP

INTRODUCTION

Aortic dissection (AD) was described by the renowned Italian anatomist and pathologist Geovani Batista Morgagni in 1761. It was studied and defined as dissecting aneurysm by the French physician René Théophile Hyacinthe Laenec in 1819, and was first diagnosed clinically by Swaine and Latham in 1885 and 1886, respectively[1]. It is a tear in the intima of the vessel, exposing the previously ill tunica media to intraluminal systolic blood force. Blood enters the media layer and splits the vessel wall into two layers. The blood-filled space in the dissected layers becomes the false lumen. This dissection extends along a variable length in the aorta, usually in a forward direction, and, at times, in a retrograde direction, from the site of intimal tear[2-4].

The term dissecting aneurysm is still used by some medical professionals nowadays; however, this concept has been replaced with the term aortic dissection for decades. The aneurysm and AD are independent diseases[5]. The latter may generally occur in an acute form[6], although, after the tear, there is a subsequent gradual expansion of the weakened outer wall of the aorta. On the other hand, the gradual deterioration of the aortic wall in aneurysms may cause a chronic dissection. Therefore, the term dissecting aneurysm should be reserved only for this possibility.

The concept of acute aortic syndrome was introduced late last century. It is defined as an acute process in the aortic wall that determines a high risk of rupture with a high morbidity and mortality. It includes AD, intramural hematoma, penetrating ulcer, and other diseases such as symptomatic or ruptured thoracic aneurysm, aortic transection and aorto-pulmonary fistula, as an erosive complication of the aneurism[7].

Currently, AD is considered a catastrophic illness, with an estimated incidence of 5 to 30 cases per million inhabitants. About 10 000 patients a year suffer from aortic dissection in the United States[8-10]. Without surgical treatment, its natural progression is fatal. A significant number of patients die without hospital medical attention. AD diagnosis is not made in 38% of patients and it reaches 28 % in necropsies[11]. Death by AD may be related to aortic rupture, cardiac tamponade, severe acute aortic regurgitation, or acute myocardial infarction by coronary involvement. The presence of recurrent pain, shock, cardiac arrest, and a pericardiocentesis with bloody fluid indicate the possible existence of an AD of the ascending aorta breaking into pericardial cavity[12, 13].

Several studies on AD have been conducted in our country[14,15], including clinical trials and post-mortem studies, which have shown the incidence and prevalence of the disease, as well as the fundamental aspects of its diagnosis and treatment. For this reason, it was decided to conduct this study, with the aim of covering an almost complete sample of our country, in more than four decades, thus providing continuity to previous research on this disease.

METHOD

A retrospective cross-sectional study was conducted. It included 888 autopsy reports with a diagnosis of AD from the Automated Registration and Control System of Anatomical Pathology (SARCAP, for its acronym in

Spanish) in Cuba, from 1962 to 2004.

The AD was classified according to the Stanford University anatomical and pathophysiological classification, proposed by Daily *et al*[16] in 1970, which includes:

- Type A: dissections involving the ascending aorta, which correspond to DeBakey types I and II[17].
- Type B: dissections that do not affect the ascending aorta, which includes DeBakey type III.

The following criteria were used for clinicopathological correlation:

- Yes: When the correlation was total or partial.
- No: When there was no correlation or data were insufficient.

Univariate and bivariate frequency distributions were built, with graphical representation. Chi-square homogeneity tests and proportion test for paired samples, with mutually exclusive characteristics, were used. Hypothesis tests and estimation required random samples. If the randomness of the sample cannot be justified, then the results of the tests are unreliable; however, their information may be used to help decide with what is observed in **Tables 4 and 5**.

RESULTS

Our study included all autopsies performed in patients over 15 years of age in 43 hospitals across the country, from 1962 to 2004, with a total of 101 082 autopsies, performed by dozens of specialists in Anatomical Pathology. In the first three years no cases of AD were reported, although it should be noted that the first five years included the deaths from a single hospital. Subsequently, other hospitals were incorporated to form the current network. The years with the highest incidence of this disease were 1994 with 133 and 2000 with 103 autopsies.

According to the type of AD, following the Stanford classification (**Table 1**), there was a predominance of type A, with 543 deaths (61.1%), of which 253 (28.5% of the total) were classified as DeBakey type I and 290 as type II (32.6% of the total). In 390 autopsies, pericardial effusion was found, due to the rupture of the dissection into that cavity. Stanford type B and DeBakey type III dissections were found in 345 deceased patients (38.9%), including 61 ruptures into pleura and 81 into the peritoneal cavity.

The mean age was 69 years, ranging between 20 and 100 years (**Table 2**). Moreover, there is a relationship between AD and increasing age, with a higher number of cases over 50 years of age. The most affected age groups were those from 65-74 and 75-84 years, with 266 (30.0%) and 210 (23.6%) deaths, respectively.

With regard to hospital stay (**Table 3**), the largest number of deaths occurred during the first 24 hours, with a total of 496 deceased patients (55.86 %), follow-

Table 1. Distribution of the sample according to Stanford and DeBakey classifications.

Stanford Type A			Stanford Type B			Total	
	Nº	%		Nº	%	Nº	%
DeBakey I	253	28,5	DeBakey III	345	38,9	888	100
DeBakey II	290	32,6					
Total	543	61,1		345	38,9	888	100

Source: SARCAP.

Table 2. Sample distribution by age groups.

Age groups (years)	Nº	%
15-24	1	0,1
25-34	3	0,3
35-44	26	2,9
45-54	76	8,6
55-64	**177**	**19,9**
65-74	**266**	**30,0**
75-84	**210**	**23,6**
85-94	108	12,2
≥ 95	5	0,6
Non-specified age	16	1,8
Total	888	100

Source: SARCAP

Table 3. Distribución de la muestra según estadía hospitalaria.

Days	Frequency	%	Cumulative Total	%
0 (< 24 h)	**496**	**55,9**	**496**	**55,9**
1-3	**209**	**23,5**	**705**	**79,4**
4-7	76	8,6	781	88,0
8-14	53	5,9	834	93,9
15-21	28	3,2	862	97,1
22-30	14	1,6	876	98,7
31-60	11	1,2	887	99,9
>60	1	0,1	888	100,00

Source: SARCAP

Table 4. Distribution of the sample according to the underlying and direct causes of death.

Causas de muerte	Nº	%
Underlying cause *		
Aortic dissection	**541**	**61,6**
Atherosclerosis	127	14,3
Cystic medial degeneration	119	13,4
Hypertension	82	9,2
Total	869	98,5
Direct cause **		
Hemopericardium	**390**	**43,9**
Shock	164	18,5
Hemothorax	38	4,3
Acute anemia	37	4,2
Bronchopneumonia	37	4,2
Total	666	75,1

* Z = 18.996; p = 0.000
** Z = 10.143; p = 0.000
Source: SARCAP

Table 5. Distribution of the sample according to diagnostic agreement.

Diagnostic agreement					
Direct cause of death*			Underlying cause of death**		
Agreement	Nº	%	Agreement	Nº	%
Yes	297	33,5	Yes	255	28,7
No	591	66,5	No	633	71,3

* $\chi 2 = 63.759$; $p < 0.05$
** $\chi 2 = 127.162$; $p < 0.05$
Source: SARCAP

ed by the deaths occurred between the first and third day of hospital stay, with 209 cases (23.86 %), making a cumulative of 79.39 %. This parameter increased with the course of days to reach 99.9% at 60 days, that is, 99.9 % of patients died within two months. One patient had a longer hospital stay. Mortality increased in the course of time.

Table 4 shows the distribution of the sample according to underlying and direct causes of death. Regarding the underlying cause, AD had the highest incidence. It was found in 541 death certificates (61.6%), followed by atherosclerosis (14.3%), cystic medial degeneration (13.4%) and finally hypertension (9.2 %). That is, 98.5% of the underlying causes of death were vascular alterations, which are closely related to the genesis of aortic syndromes.

With regard to the direct cause of death, hemopericardium was the most common one, found in 390 deaths (43.9%); followed by shock, which was diagnosed in 164 autopsies (18.5%). Pleural effusion was the third with 38 deaths (4.3%), and finally bronchopneumonia and acute anemia, found in 37 autopsies (4.2% each one).

Regarding the diagnostic agreement on the direct cause of death (**Table 5**), there was full and partial clinicopathological correlation only in 33.5% of the sample. It was similar with the underlying cause of death, with a correlation of 28.8 %. These results show high rates of diagnostic discrepancy.

DISCUSSION

With the decline in autopsies during the last three decades in most countries of the world, obtaining data from a large number of cases is very complicated. Despite the medical-legal and educational importance of this procedure, its use is becoming increasingly scarce[18-20]. In Cuba, it has been possible to maintain acceptable levels in postmortem studies, although we are not exempt from this crisis. From 1991 to 2011, the rate of autopsies in our country, not including fetuses, was greater than 33 %; and, in the case of hospital deaths, it has remained near 60 % over the same period[21].

At present, there are numerous classification systems to describe AD, although Stanford classification main-

tains hegemony due to its usefulness from a physiopathological point of view[22,23]. The involvement of the ascending aorta influences the prognosis and the subsequent course of action. That is, when the tearing includes the ascending aorta, surgery is always required. Conversely, if the dissection does not affect this segment, the course of action may be medical or endovascular treatment[24,25].

The AD is a disease that mainly affects elderly people. Our results are similar to those found in studies such as the prestigious IRAD[11,26,27] and RESA[28], where there is a prevalence of patients over 60 years, probably due to the loss of aortic elasticity and distensibility associated with aging. Histologically, senile aorta shows a fragmentation of elastin and a concomitant increase in collagen, which favors a physiological reduction of distensibility and increases myocardial oxygen consumption by 20 to 40 %. Recent experimental data from animal models suggest that impaired *vasa vasorum* flow to the aortic wall leads to increased rigidity with histological changes similar to ageing[4].

Without surgical treatment, AD mortality increases by 1% every hour after the onset of the symptoms[29]. As a result, more than one third of patients with this disease die within the first 24 hours, half of them in the first 48 hours, two thirds in the first two weeks and nearly 90 % in the first 3 months. The risk of death is increased in patients with complications such as aortic rupture, stroke, visceral ischemia, cardiac tamponade, and circulatory failure. Data from other important registries on acute AD show that in the absence of immediate surgical repair, medical treatment is associated with high mortality. Even with surgical repair, the hospital mortality rates are 10% after the first day, 12% at 2 days and almost 20% at 2 weeks[30,31].

In our study, in about 40 % of deaths, AD was not identified as the underlying cause of death. The cystic and atherosclerotic degeneration of the media and hypertension are included in this group. Cystic medial degeneration is defined as a degenerative process of the tunica media, with loss or fragmentation of elastic fibers and smooth muscle cells. Most dissections in young people are generally due to congenital abnormalities of the connective tissue, affecting the media of the aorta[32,33]. The atherosclerotic degeneration is the alteration of the intima, with formation of large plaques that end up destroying the elastic fibers and smooth muscle cells of the media, which in turn causes weakness and dilation of the arterial wall. Hypertensive disease increases the absolute stress on the arterial wall, the force of left ventricular ejection (dP/dt) and myocardial oxygen consumption. All this intensifies chronic damage of the arterial wall and the risk of dissection or rupture[4].

In this study, the increased incidence of hemopericardium is related to the predominance of Stanford type A dissections. The accumulation of blood in the pericardial sac has an anatomical relationship with the acute aortic syndrome, because the majority of dissections affect the ascending aorta. In 65 % of cases the site of intimal tear is located there, mostly within the first 5 inches. The tear may progress and break into the pericardial cavity, which increases the volume and pressure within it. These alterations, when they occur in an acute form, cause the pericardium to tense up and lose its elasticity. When the intrapericardial pressure exceeds intracavitary pressure, circulatory collapse occurs and death is inevitable if an emergency evacuation of fluids is not performed[34,35]. About 10 % of patients with a diagnosis of Stanford type A dissection suffer from cardiac tamponade with a high risk of death[36,37].

On the other hand, the shock is linked to blood loss, pain and heart failure is secondary to acute aortic regurgitation or acute myocardial infarction. Pleural effusion is related to the dissection of the thoracic aorta and is more likely to occur in the left pleura. Bronchopneumonia was found in patients who survived the early days, and is due to septic in-hospital complications, because is not directly related to the cause of the admission to hospital, in intensive care units most of the times.

The AD has well-defined clinical symptoms, but its acute onset may mislead the physician. In most cases, there is a diagnosis of myocardial infarction[14], pulmonary embolism, pericarditis[12] and other conditions. The clinicopathological discrepancies range between 25 and 52%, but when the aortic syndromes are analyzed, these percentages are close or even above 50%[38-41]. Gee[42] found a discrepancy rate of 58.8 % in the aortic aneurysm, Cameron and McGoogan[43], 57.9% and Fares *et al*[44], 64.2%; these results are in agreement with our study. Furthermore, in a study conducted in the province of Cienfuegos, on 55 deaths due to AD, the diagnosis made when the patient was admitted to hospital was confirmed in only 18.18% of cases[15]; however, Battle *et al*[45] found a discrepancy

rate of 28.1%.

CONCLUSIONS

During the 42 years covered by the study, those who died from AD in Cuba predominantly had Stanford type A dissections, were over 55 years of age and had a hospital stay of less than 2 months. The largest number of deaths occurred in the first 24 hours and the rate of diagnostic agreement was low.

REFERENCES

1. Carbonell Cantí C. Historia de la cirugía de la aorta torácica. En: Vaquero C, ed. Cirugía de la aorta torácica. Valladolid: Gráficas Andrés Martín SL, 2010; p. 15-32.
2. Tsai TT, Isselbacher EM, Trimarchi S, Bossone E, Pape L, Januzzi JL, *et al*. Acute type B aortic dissection: does aortic arch involvement affect management and outcomes? Insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation. 2007;116(11 Suppl):I150-6.
3. Ince H, Nienaber CA. Tratamiento de los síndromes aórticos agudos. Rev Esp Cardiol. 2007;60(5):526-41.
4. Isselbacher EM. Enfermedades de la aorta. En: Braunwald. Tratado de Cardiología. T II. 6ta ed. México: McGraw-Hill Interamericana, 2005; p. 1739-40.
5. Coselli JS, Conklin LD, LeMaire SA. Thoracoabdominal aortic aneurysm repair: rewiew and update of current strategies. Ann Thorac Surg. 2002; 74(5):S1881-4; discussion S1892-8.
6. Golledge J, Eagle KA. Acute aortic dissection. Lancet. 2008;372(9632):55-66.
7. Vilacosta I, San Román JA, Aragoncillo P, Ferreirós J, Mendez R, Graupner C, *et al*. Penetrating Atherosclerotic aortic ulcer: documentation by transesophageal echocardiography. J Am Coll Cardiol. 1998; 32(1):83-9.
8. Santo AH, Puech-Leão P, Krutman M. Trends in aortic aneurysm- and dissection-related mortality in the state of São Paulo, Brazil, 1985-2009: multiple-cause-of-death analysis. BMC Public Health [Internet]. 2012 [citado 2013 Nov 14];12:859. Disponible en: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3527140/pdf/1471-2458-12-859.pdf
9. Braverman AC. Aortic dissection: prompt diagnosis and emergency treatment are critical. Cleve Clin J Med. 2011;78(10):685-96.
10. Braverman AC. Acute aortic dissection: clinician update. Circulation. 2010;122(2):184-8.
11. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, *et al*. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA. 2000;283(7): 897-903.
12. Cury Rezende P, Borges Viana V, Benvenuti LA. Caso 2/2011 - Paciente joven, del sexo masculino, con cuadro de dolor torácico tipo pleurítico, hipotensión, sudoresis profusa, con ECG sin alteraciones isquémicas agudas y marcadores de lesión miocárdica negativos. Arq Bras Cardiol. 2011;96(4):e62-8.
13. Kelly BS. Evaluation of the elderly patient with acute chest pain. Clin Geriatr Med. 2007;23(2):327-49.
14. Valdés Dupeyrón O, Villar Inclán A, Nafeh Abiz-Reck M, Pedroso J, Guevara González L, Chao González N, *et al*. Tratamiento quirúrgico de las enfermedades de la aorta ascendente. Estudio de tres años. Rev Arg de Cir Cardiovasc. 2011;IX(1):47-59.
15. Olivert Cruz M, Romero Cabrera AJ, Bembibre Taboada R, Bermúdez López J. Disección aórtica. Estudio en un decenio (1987-1997). Rev Cubana Med. 2000;39(4):217-21.
16. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg. 1970;10(3):237-47.
17. DeBakey ME, Beall AC, Cooley DA, Crawford ES, Morris GC, Garrett HE, *et al*. Dissecting aneurysms of the aorta. Surg Clin North Am. 1966;46(4):1045-55.
18. Burton EC, Phillips RS, Covinsky KE, Sands LP, Goldman L, Dawson NV, *et al*. The relation of autopsy rate to physicians' beliefs and recommendations regarding autopsy. Am J Med. 2004;117(4):255-61.
19. Burton JL, Underwood JC. Necropsy practice after the «organ retention scandal»: requests, performance, and tissue retention. J Clin Pathol. 2003; 56(7):537-41.
20. Sanz-Ortiz J, Mayorga M, Martín A. Autopsia clínica en Oncología: ¿está en crisis? Med Clin (Barc). 2011; 137(7):317-20.
21. Hurtado de Mendoza Amat J, Montero González TJ, Ygualada Correa I. Situación actual y perspectiva de la autopsia en Cuba. Rev Cubana Salud Pública. 2013;39(1):135-47.
22. Ladouceur M, Fermanian C, Lupoglazoff JM, Edou-

ard T, Dulac Y, Acar P, *et al*. Effect of beta-blockade on ascending aortic dilatation in children with the Marfan syndrome. Am J Cardiol. 2007;99(3):406-9.
23. Contreras Zúñiga E, Zuluaga Martínez SX, Gómez Mesa JE, Ocampo Duque V, Urrea Zapata CA. Disección aórtica: estado actual. Rev Costarric Cardiol. 2009;11(1):19-27.
24. Kahn SL, Dake MD. Stent graft management of stable, uncomplicated type B aortic dissection. Perspect Vasc Surg Endovasc Ther. 2007;19(2):162-9.
25. Ford PF, Farber MA. Role of endovascular therapies in the management of diverse thoracic aortic pathology. Perspect Vasc Surg Endovasc Ther. 2007; 19(2):134-43.
26. Rampoldi V, Trimarchi S, Eagle KA, Nienaber CA, Oh JK, Bossone E, *et al*. Simple risk models to predict surgical mortality in acute type A aortic dissection: the International Registry of Acute Aortic Dissection score. Ann Thorac Surg. 2007;83(1):55-61.
27. Rogers AM, Hermann LK, Booher AM, Nienaber CA, Williams DM, Kazerooni EA, *et al*. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation. Results from the International Registry of Acute Aortic Dissection. Circulation. 2011;123(20):2213-8.
28. Evangelista A, Padilla F, López-Ayerbe J, Calvo F, López-Pérez JM, Sánchez V, *et al*. Registro Español del Síndrome Aórtico Agudo (RESA). La mejora en el diagnóstico no se refleja en la reducción de la mortalidad. Rev Esp Cardiol. 2009;62(3):255-62.
29. Song KJ, Kang SJ, Song JM, Kang DH, Song H, Chung CH, *et al*. Factors associated with in-hospital mortality in patients with acute aortic syndrome involving the ascending aorta. Int J Cardiol. 2007;115(1): 14-8.
30. Suzuki T, Mehta RH, Ince H, Nagai R, Sakomura Y, Weber F, *et al*. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation. 2003;108(Suppl 1): II312-17.
31. Mehta RH, Suzuki T, Hagan PG, Bossone E, Gilon D, Llovet A, *et al*. Predicting death in patients with acute type A aortic dissection. Circulation. 2002; 105(2):200-6.
32. Senay S, Alhan C, Toraman F, Karabulut H, Dagdelen S, Cagil H, *et al*. Endovascular stent-graft treatment of type A dissection: case report and review of literature. Eur J Vasc Endovasc Surg. 2007;34(4): 457-60.
33. Ince H, Nienaber CA. Diagnosis and management of patients with aortic dissection. Heart 2007;93(2): 266-70.
34. Nienaber CA, Eagle KA. Aortic dissection: new frontiers in diagnosis and management. Part II: Therapeutic management and follow-up. Circulation. 2003;108(6):772-8.
35. Santini F, Luciani GB, Montalbano G, Messina A, Faggian G, Mazzucco A. Acute type A aortic dissection: an update on a still challenging disease. J Cardiovasc Med (Hagerstown). 2007;8(2):102-7.
36. Braverman AC, Thompson R, Sanchez L. Diseases of the aorta. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Philadelphia: Elsevier Science, 2011; p. 1309-37.
37. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, *et al*. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266-369.
38. Shojania KG, Burton EC, McDonald KM, Goldman L. Changes in rates of autopsy-detected diagnostic errors over time: A systematic review. JAMA. 2003; 289(21):2849-56.
39. Royal College of Pathologists of Australasia Autopsy Working Party. The decline of the hospital autopsy: a safety and quality issue for healthcare in Australia. MJA. 2004;180(6):281-5.
40. Spiliopoulou C, Papadodima S, Kotakidis N, Kuotselinis A. Clinical diagnoses and autopsy findings. A retrospective analysis of 252 cases in Greece. Arch Pathol Lab Med. 2005;129(2):210-4.
41. Arce FP, Ondiviela R, Val Bernal JF. Discordancias clínico-patológicas en la autopsia. Experiencia del Hospital Marqués de Valdecilla. XXIX Reunión de la SEAP. Madrid, España; 6 Febrero 2007.

42. Gee WM. Causes of death in a hospitalized geriatric population: an autopsy study of 3000 patients. Virchows Arch A Pathol Anat Histopathol. 1993; 423(5):343-9.
43. Cameron HM, McGoogan E. A prospective study of 1152 hospital autopsies: II. Analysis of inaccuracies in clinicial diagnoses and their significance. J Pathol. 1981;133(4):285-300.
44. Fares AF, Fares J, Fares GF, Cordeiro JA, Nakazone MA, Cury PM. Clinical and pathological discrepancies and cardiovascular findings in 409 consecutive autopsies. Arq Bras Cardiol. 2011;97(6):449-55.
45. Battle RM, Pathak D, Humble CG, Key CR, Vanatta PR, Hill RB, *et al*. Factors influencing discrepancies between premortem and postmortem diagnoses. JAMA. 1987;258(3):339-44.