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Outlook of Non Operated Type B Aortic Dissection with Special Reference to the Incidence of Degenerative Abdominal Aortic Aneurysm (AAA). One Center Study

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Key words. Dissecting aortic aneurysms ; abdominal aortic aneurysms ; thoracic aorta.

Abstract. *Objectives :* to determine the value of pharmacological treatment of type B aortic dissection (B AD) in face of new forms of treatment.
Design : this is a retrospective study of the period from 1990 to 2000. Files of 81 patients have been reviewed and completed by questionnaires.
Results : Two B AD died after admission without any treatment, 10 were operated on with 7 discharged alive (group I) ; 69 received hypotensive agents and β-blockers, 65 were discharged alive (group II). Late mortality of the group I is 3/7, not related with B AD. Late mortality after mean follow-up of 56.8 months is 27/65 with 4/27 related to B AD (4 ruptures, 2 operated on). Non fatal secondary surgery amounts 5 in 4 patients. Total B AD aortic events comprise 8/65 patients. Type A AD were operated on successfully (8 : 4 before B AD, and 4 after B AD). Degenerative abdominal aortic aneurysms were present, operated (9) or not (3), in the history of patients and 3 more appear subsequently. At 10 years, actuarial survival is 40% ± 18.
Conclusion : in non-complicated cases of B AD, medical treatment is a reasonable choice, provided that a strict follow-up of the thoracic abdominal aorta is performed.

Introduction

Success of pharmacological treatment of type B Aortic Dissection (B AD) introduced by Myron WHEAT (1) has reinforced the concept of chronic aortic stress in the pathogenesis of this vascular catastrophe. B AD would be an evenemential disease. However, some of the affected patients do not exhibit any increased aortic stress and belong to the group of inherited diseases characterized by anomalies of aortic wall macromolecules (Marfan, Ehlers-Danlos). Besides, the pathogenesis of degenerative AAA has been increasingly attributed both to genetically induced aortic collagen disorders and to acquired atherosclerosis (2). As the use of endo-vascular treatment type B AD is growing (3), the choice will be weighted not only with open surgery but also with medical treatment. By reviewing retrospectively a consecutive series of B AD in one center, a attempt to define a natural outcome will be drawn and compared with already published series ; incidentally we report the incidence of type A AD and previous and metachronous degenerative infra-renal abdominal aortic aneurysm (AAA).

Material and methods

Files of 81 patients admitted between January 1st, 1990 and December 31, 2000, with the diagnosis of B AD have been reviewed. Diagnosis was obtained through CT scan. General policy was to start immediate treatment with β-blocking and hypotensive drugs and to proceed with surgery only if signs of impeding rupture or visceral ischemia were present. No endovascular treatment was performed during this period. Data on hospital survivors have been completed from outpatients clinic data or oral and written information obtained from referring physicians, family doctors, and families. Several patients had not been submitted to systematic follow-up.

Results

Patients (n = 81) were in the sex ratio of 4M:1F (66:15). Mean age was 62.8 ± 10.4 (extremes 41-85). Two patients died shortly after admission. Seventy nine patients were either operated on (n = 10) by partial descending aorta replacement or submitted to continue pharmacological treatment (n = 69) ; one of them

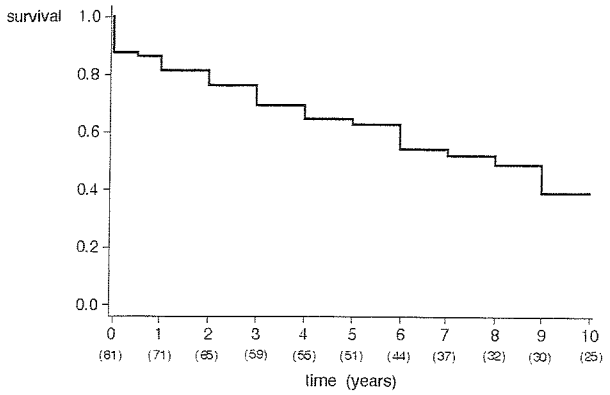

Fig. 1
Actuarial survival curve of 81 patients admitted with the diagnosis of B AD.

received semi-electively AAA treatment surgery one month later, because of significant enlargement of the diameter ; he subsequently died from sepsis.

Early mortality

Two patients died prior to any treatment (one cerebral massive infarction, one complete mesenteric infarction, at autopsy). Four out of 69 patients (6%) with medical treatment died (one tamponnade due to secondary retrograde dissection at day 10, one cerebrovascular accident at day 5, one descending aorta rupture after the patient's discharge from Intensive Care Unit at day 4, and one was operated on for mesenteric infarction at day 5 and died shortly thereafter). Out of the 10 patients operated on for partial descending aorta replacement, three died (30%) (one from acute myocardial infarction combined with CVA, one from mesenteric infarction, and one from intractable recurrent thoracic hemorrhage). Total early mortality is 10 (15%), two prior to treatment, four under medical treatment, three after thoracic surgery, and one delayed death after non-urgent AAA surgery during the same in-hospital stay following admission for B AD.

Late mortality

Late mortality (at December 31, 2000) after a mean follow-up time of 56.8 months amounted 30 (27M, 3F). In the seven hospital survivors operated on for descending aorta partial replacement, three died (43%) (two from heart failure, one from obstructive bronchopneumopathy). In the 64 medical treatment hospital survivors, 27 died (42%) (eight from acute myocardial infarction, four from descending aortic rupture (two despite emergent aortic surgery), three cerebrovascular accidents, 12 died from other causes than cardiovascular (four pulmonary failures, three gastrointestinal carcinomas, five miscellanous). Actuarial survival curves displays 40% survival rate ± 18 at 10 years. According to sex, the numbers are 25% in males and 69% in females (Fig. 1).

Late descending aorta surgery (n patients = 8)

Due to increasing aortic diameter, four patients (6%) experienced successfully five partial descending aortic replacements, 3 to 9 years after B AD occurrence. Six patients experienced descending aorta rupture, two died in the admitting hospital prior to transfer for urgent surgery, and four were operated on emergently with two postoperative deaths ; descending aorta diameters of operated on patients ranged from 8 to 10 cm.

Incidence of type A aortic dissection

Four patients had experienced type A aortic dissection prior to type B AD occurrence (4 to 7 years) and had been successfully operated on ; two of them had Marfan disease ; four additional patients experienced type A aortic dissection (2 to 9 years after B AD) ; and survived surgery.

Occurrence of degenerative AAA

Prior to B AD diagnosis, 12 patients (15%) had either a history of AAA surgery (n = 9) or an intact AAA (n = 3). Two of the three were operated on one month and one year after B AD, one is still left unoperated). After B AD occurrence, AAA appeared in three patients and were operated on 2, 5 and 7 years thereafter ; hence, nearly one B AD patient over 5 has or will have an AAA.

Discussion

Results of medical treatment of B AD have obviously a beneficial bias due to the process of selection of most ominous cases for immediate surgery ; hence the comparison of 6% mortality of the medical treatment with 30% mortality of urgent surgical treatment is not fair. Besides, this one-centre retrospective study comprises a limited number of cases. Published series dealing with outcome of medically treated B AD (4-9) differ by the number of patients, by the criteria for immediate surgery and the fact that they are uni- or multicentric. Besides, they all underscore the fact that operative mortality has been decreased with time. Nevertheless, they provide us with a significant amount of medically treated patients ; so that, we can roughly appreciate the limits and the benefits of pharmacological treatment (Table 1). In four series, the proportion of medically treated type B AD varies from 76 to 87% ; it reaches 87% in our series. In-hospital mortality varies from 2.5 to 17.6% (with 5% early cross-over to surgery in one series (7). It is 6.8 in our series. As far as late mortality linked with initial type

Table I

	[3]	[4]	[5]	[6]	[7]	[8]	[9]	this series
n patients admitted for BAD	56	53		88	235	68	86	81
death before treatment	0			0		3		2
initial surgery	28***	12 12%		27 30%	38+9* 24%	17	10 13%	10
operative mortality	3 11%	4 33.3%		5 18%	8/38 21%	0	3 30%	1 10%
medically treated	28	44 77%	50**	61 70%	187-9 76%	48 78%	76** 87%	69 87%
in hospital mortality medically treatment	0	6 14%		4 7%	33 17.6%	1 2.5%		4 5.8%
discharged alive	53			57	30+154*	64		74
AD related mortality	1/28 4%		9 18%	4 7%		4 6.2%		2 3%
AD related late surgery	0		10 20%		med47 36%	med12 23%	10	8 12%
operative mortality late surgery			NM		4 0 0	0	3/10 36% ?/11 ND	2 (rupt.) 25%
total AD related mortality			18%	7%	2%	6.2%	4%	5.5%

* operative mortality not mentioned for the 8 cross-over and supposed to be 0
** Marfan excluded
*** endovascular

B AD (the link is not always evident), its importance varies from 3 to 18.8% at different follow-up time ; it is 5.6% after a mean follow-up of 56.8 months in our series. Late AD related mortality must be viewed together with related late aortic surgery ; it ranges from 2 to 36% and is 12% in our series. One part of late mortality due to AD is made up of the operative mortality of late thoracic surgery ; it ranges from 0 to 30% ; it is 25% in our series.

One group (9) evidences a link between the amount of late events linked with AD and the fact that the false lumen remained patent while JUVONEN *et al.* (5) do not. One recent Japanese study (10) provides firm statistical evidence that the patency of the false lumen plays a positive role in the growing of descending anterior aorta and consequently, the incidence of its rupture. A fair comparison between the two forms of treatment must imply that medical treatment survivors have a late excess mortality (4/64 in this series) linked to aortic rupture that could have been prevented by immediate surgery. Besides, medical treatment alone implies a meticulous follow-up in order to electively perform surgery in dilating chronic dissecting aneurysms ; our follow-up was missing or incomplete since six patients experienced rupture short of timed radiological prealable diagnosis.

As far as the mechanisms of B AD are concerned, two different etiopathogenetic groups can be discerned. The one with obvious genetic alteration of aortic wall macromolecules (Marfan, Ehlers-Danlos), the other with chronic aortic stress (longstanding hypertension) and without recognized primary parietal macromolecular defects. It is impressive to see the prevalence of degenerative AAA far more frequent in the B AD group than in the general population of the same age and sex. Atherosclerosis, i.e., environmental cause, plays a role in the development of AAA, increasing number of data suggests that genetically depending enzymatic actions alter directly the elastin and collagen components of aortic wall (11). The coincidence of type B AD with a history of operated on degenerative infrarenal aneurysm (AAA) or the presence of unoperated AAA had been mentioned by LEE *et al.* (12) ; they reported three operated on AAA in a group of 86 type B AD, the mere presence of 11 more non-operated AAA not operated (16%). The authors stressed therefore that abdominal and not only thoracic aorta scanning must be performed regularly in the follow-up. CAMBRIA *et al.* (13) had previously reported that among 300 type B AD, 18 had been previously diagnosed to have a degenerative thoracic or abdominal aortic aneurysm : 5 had been operated on before, 5 were present but not affected by the extension of the dissection and 8 were involved by the dissection (5 AAA, 3 thoraco-AAA). Our series is characterized by the importance of AAA preexisting type B AD (12%) and the additional metachronous development of three more degenerative AAA.

According to some, recommendations for imaging's follow up and imaging's schedule is at 1, 3, 6, 9 and 12 months after discharge from the hospital and annually thereafter (14-15). Systemic hypertension, baseline aortic size and presumably presence of a patent false lumen identify either risk of recurrent aortic dissection or/and increase of the aortic size and consequently risk of rupture (16, 17). Apart from the radiologic follow-up, medical treatment is recommended to prevent or slow down the increasing size of aorta : β-blockade, calcium channel inhibitors and statins. SHORES *et al.* (17) demonstrated that the mean slope of the regression line for the aortic root dimensions (rate of dilatation) is significantly lower in the β-blocker group than in the control group.

The conclusion probably should apply to dissected descending thoracic aorta. It is admitted that agents must be increased to achieve a blood pressure < 135/80 mmHg (130/80 in Marfan's disease). The benefits definitely evidenced in Marfan patients could be extended non-Marfan patients. Calcium channel blockers are a reasonable alternative for those patients intolerant to β-blockers. The beneficial effects of statins after successful aortic abdominal surgery are characterized by a lesser cardiovascular mortality. Moreover, KERTAI (18) demonstrated that statins suppress MMP-9 production in the aortic wall, whose action to promote aortic dilatation has often been demonstrated (19). Statins have not yet been proposed for prevention of aortic dilatation in type B AD survivors. It has been proposed to use statins with the objective to reduce LDL at less than 70 µg/dl (20).

Close radiologic follow-up, both abdominal and thoracic, and administration of active agents are essential for patients having survived type B aortic dissection. While type A dissections, which are routinely operated on, have an understandably better medical compliance, unlike type B because of the lack of a surgical scar and because the patients have often been moved from a surgical intensive care unit to a non-surgical service, the patients and their general physicians do often neglect a close follow-up either by the surgeon or by the cardiologists. Patients and physicians should be clearly informed of the importance of follow-up.

Conclusion

As far as this retrospective study is concerned, it lacks the answer to specific questions about usefulness of sequential imaging, role of a patent false lumen and role of pharmacological agents. But it confirms that a 40% 10 years survival rate can be expected with cardiovascular events accounting for 50% of the late mortality ; moreover, it evidences the importance of existing AAA and the propensity of B AD patients to have new aortic dissection (A or B) and new degenerative AAA formation.

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