Pregnancy Does Not Cause Structural Bioprosthesis Alteration

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The twentieth century has seen a growth in basic concepts in cardiology and has also seen the creation of new areas of research. The dynamic process of constant information reassessment has eliminated nonscience-based myths and dogmas, for example Peter's aphorism [1].

In the 1950s, when the first successful case of pregnancy in a woman with a valvar prosthesis was reported by Canfield et al [2], the study of mutual influences of the valvar prosthesis/gestation binomium from a multidisciplinary viewpoint had its starting point [3].

Gestation success rates of over 80% have occurred in patients with a combination of a normal prosthesis, preserved ventricular function, and sinus rhythm; this triad has implied a good prognosis [4,5]. However, this optimistic evaluation has been questioned by those who believe that gestation is also an accelerating factor for bioprosthesis degeneration, particularly biological tissue calcification [6], and for reduction in the half life of the bioprosthesis.

An analysis in European cardiology centers reported 35% bioprosthesis malfunction, mostly due to calcification, as the single maternal complication. Hemodynamic deterioration occurred in about 80% of cases as well as some short-term reoperations, but maternal mortality rates were not reported [7]. Hanania et al. [8] reported biological prosthesis degeneration as the only maternal complication during the gravido-puerperal cycle in 17.5% of cases. Fifty percent of these cases of biological prostheses degeneration occurred more than nine years (average = 10.5) after implantation of the device. Badduke et al [9] reported a greater incidence of structural bioprosthesis degeneration (47.1% vs. 14.3%) and reoperation (59% vs. 19%) among young pregnant women, and assumed, therefore, that degeneration was age-dependent. On the contrary, Jamielson et al's [10] retrospective multicenter study found no difference in mortality rates and incidence of complications related to bioprosthesis, including calcification and reoperations, attributable to gestation.

These studies analyzed retrospectively a limited number of patients, whose ages varied greatly as did the time of implantation and postimplantation. Because the methods of analysis were not uniform within the studies, the conclusions observed should be carefully analyzed.

To overcome the obstacles resulting from retrospective and heterogenous studies in analyzing the interdependence between bioprosthesis/gestation, we developed a rigorous study design based on prospective evaluation of 85 patients, beginning at the time of biological prosthesis implantation, and limited to women ranging in age from 18 to 35 years [11].

In our study, 48 (56.6%) women with no structural abnormalities (bioprosthesis stenosis, calcification, rupture, leak and thickening) became pregnant between 12 and 36 months postimplantation of bovine pericardium bioprosthesis, and 37 (43.5%) women have not become pregnant. The cases, distributed therefore according to natural randomization, were followed until 60 months post-implantation in an attempt to rule out a time-related deterioration of prosthesis tissue characteristics (smaller probability of intrinsic structural failure of the prosthesis). The five-year period was long enough to make sound conclusions because, if the follow-up time period had been shorter, it might have compromised pre- and postgestational observation, and, if longer, it might have added obstacles related to the self-actuarial bioprosthesis curve [12]. Likewise, the exclusion of adolescents from the study has prevented the superimposition of the age factor as a manifest influence on the structural calcification of the prosthesis [13]. At the end of 60 months, we compared percentages and survival curves free from bioprosthesis structural alterations between the two groups (table I and figure 1) and no statistical differences were observed.

Our data have reinforced the line of thinking that considers the gravido-puerperal cycle as a transient change factor that may possibly even cause hemodynamic compromise, but should not necessarily be interpreted as a result of a direct influence on biological prosthesis structure [14].

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Table I - Structural alterations in pregnant (P - 47 cases) and nonpregnant women (NP - 38 cases)

	P		NP		Total		p value
	n	%	n	%	n	%	Descritivo
Leak	11	22.92	8	21.62	19	22.35	p=0.887
Thickening	7	14.58	5	13.51	12	14.12	p=0.888
Stenosis	4	8.33	5	13.51	9	10.59	p=0.494
Calcification	5	10.42	3	8.11	8	9.41	p=1.000
Rupture	3	6.25	1	2.70	4	4.71	p=0.629
Thrombus	0	0.00	2	5.40	2	2.35	p=0.187
Vegetation	1	2.06	0	0.00	1	1.17	p=1.000
Any alteration	22	45.8	13	35.3	35	44.71	p=0.498

In conclusion, we found no evidence to suggest that gestation has an accelerating influence on the development of structural alterations in bioprostheses implanted in the age group ranging between 18 and 35 years. The structural changes observed should therefore be attributed to the assumed natural history of biological prosthesis implantation in the age group analyzed.

We assume our conclusions may be helpful in family planning for the bioprosthesis carrier, applied to valvar substitution selection in the fertile age, and to the time for gestation counseling.

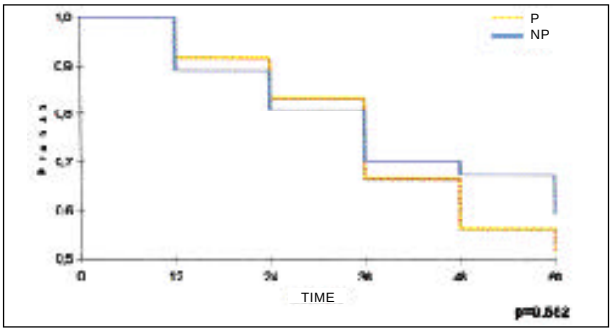

Fig. 1 - Survival free from structural alterations, thrombus and/or vegetation. P = pregnant women; NP = non pregnant women

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