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Morphometry of the human splenic artery: muscular columns, morphofunctional aspects and developmental implications

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Summary. There is a paucity of studies in the literature concerning the structural characteristics of the arterial wall in the abdominal region using human material and specialized morphometric techniques. In the present study we carry out the morphometric study, describing a series of structural peculiarities in 12 segments of the human splenic artery. Among these the presence of length-wise or spiral-shaped muscular columns in the medial layer which mark and reduce the diameter of the arterial lumen is of major importance. In its underlying intima small localized thickenings appear which, with age may become generalized. We also analyze the different intimal thickenings and such indices as the Intimal Thickening Index, Lumen Reduction Index and Pathologic Thickening Index, with differences among the groups we have considered. The study of elastin in the various parietal structures help us to understand the possible pathogenesis of the thickenings, and to clarify the important morphological-functional correlation for the regulation of blood flow which exists in this arterial region.

Key words: Splenic artery, Intimal thickening, Muscular columns, Atherogenesis, Arterial morphometry

Introduction

The accepted definition of an artery depicts a simple pattern of three layers (the intimal endothelial, media and an outer or adeventitia) in terms of its structure. The artery is predominantly elastic or muscular due to its proximity to or distance from the heart (Fawcett, 1992). Further research has shown a series of structural pecularities which alter this notion and are conditioned by age, species, the individual and even depend on the different arterial regions.

Considerable study has been done into the structure of the cerebral and coronary arterial wall. There is less reported research concerning the celiac trunk (Díaz et al., 1987) and branches, although this is an area of physiological and clinical importance. Investigators tend to concentrate on the post-trauma changes of the spleen (Ogurzkurt et al., 1996) and arterial repercussions and its usefulness as a blood source in hepatic disease or in localized chemotherapy treatment (Ohta et al., 1993).

The splenic artery is the widest branch of the celiac trunk. It vascularizes the spleen, a unique organ from the others in this region of the body given its hematopoyetic function. Its blood flow is abundant and variable, able to provide 350 liters in 24 hours. It is a spongy organ with considerable capacity of retention and excretion of blood where the splenic artery possesses a wall which may respond at any time to the needs of the organ.

Thus, this artery undergoes strong fluctuations in blood flow and its arterial wall is constantly being reorganized. These adjustments are seen largely in the medial layer of the artery, with columnar formations of muscular material following a different direction from the rest of the smooth muscular cells of concentricity lined up to the arterial lumen. Furthermore the intimal thickenings which have been classified by the American Heart Association SAC (1991) complement the information we have on pads or intimal cushions (Velican and Velican, 1977). Although the elements described are physiological and regulators of flow, the adaptive zones could, over time, become sensitive to atheromatose pathologic development (Glagov and Zarins, 1989).

We have conducted a morphometric study of the human splenic artery to add to those performed to date in this field. We have used the QUANCOUL® program to measure the surface, contours and quantify color images. In this case we have studied elastin to ascertain whether

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it varies significantly in the different areas of the artery.

Materials and methods

A total of 12, 1-cm-long distal segments of human splenic arteries were studied, removed during splenectomy, in individuals ranging in age from 16 to 54 years. The cases were selected where the basic disease were not related to arteriosclerosis nor to any hematologic illness associated with chronic splenomegalia, aneurysm or anatomic variations.

The extracted pieces were set immediately in 10% formol solution, washed in water and dried in alcohol at progressively increased concentrations. They were dipped in paraffin and cut with a microtome 6 μm thick. Finally they were stained with the Orceina and Verhoeff technique to bring out elastic structures.

The arteries were divided in three non-exclusive groups (normal arteries, with muscular columns in the medial layer and with generalized intimal thickening) to study the medial layer, the muscular columns and the intimal layer .

The morphometric study was carried out in Unit 441 INSERM of Pessac-France with the QUANCOUL® program at an increase of 400x, with sufficient measurements to make the study statistically significant. The study was divided into two sections:

1.- General morphometry, in which the following parameters were obtained: 1) Surface of the intimal and medial layer and muscular columns. 2) Theoretical and actual lumen of the artery. 3) Index of Intimal Thickening, of Reduction of the Arterial Lumen and of Pathologic Thickening. 4) Contour of the internal elastic membrane and Elastolysis Index.

2.- Special Morphometry. The elastin concentration was studied in different sites: normal medial and intimal layers, intimal thickenings and muscular columns.

In the statistical study we analyzed the mean, standard deviation, variance, co-variance and the q of Newman Keuls for significant mean analysis, with values for significance of $p<0.05$, $p<0.01$ and $p<0.001$

We define the Intimal Thickening Index (I.T.I.) as the quotient between the surface of the intima 100x and the surface of the media; the Pathologic Thickening Index (P.T.I.) as the quotient between thickened and normal intima; the Lumen Reduction Index (L.R.I.) as the value obtained from the difference of 100 less the quotient between the actual lumen 100x and the theoretical lumen; the Theoretical Lumen (T.L.) as the sum of the actual lumen and the surface of the intima; the Elastolysis Index (E.I.) as the quotient of the average distance between two fragments of the Internal Elastic Membrane (I.E.M.) or elastolysis and the contour that the I.E.M. presents in a single surface area and the Total Area of the artery as the sum of the medial and intimal layers, and arterial lumen (excluding the adventitia).

Results

1.- Qualitative study

The splenic artery is defined as a visceral vessel, muscular in character and sinuous in its path to the spleen. In the medial layer there was considerable muscle and smooth muscle cells (SMC) organized configuring a series of oblique directional strips forming a spiral-shaped muscular element referred to as the muscular column of the medial tunic. The presence of elastic fibers was not very marked except in the internal limiting membrane, variegated in form and frayed throughout. The endothelium offered no special characteristics. We observe the presence of subendothelial spaces where there were muscular fibers, collagen and amorphous material. The intimal thickenings were more frequent than the large generalized thickenings which cover the arterial circumference.

The *muscular columns of the medial tunic* (Fig. 2) appeared in the transverse cuts of the obliquely sectioned vessel, and provided a speckled aspect to this part of the arterial wall. This obliqueness is compatible with some of the single direction length, following the axis of the vessel. This column stretched the I.E.M. altering the vessel lumen and forming an endoarterial cushion which gave the medial layer a papilomatose aspect. At this level, the intima presented a localized thickening, with well-defined subendothelial space. Some of these areas were sites of more advanced atheromatose lesion (Fig. 4).

The *internal elastic membrane (I.E.M.)* of the splenic artery presented considerable polymorphism, appearing at times linear and sinuous (Fig. 1) and other times frayed, in the form of multiple parallel lamina, but without degenerative signs in most cases. The I.E.M. was most altered and frayed in the areas where there were intimal thickenings, directly related to the intensity and surface of the thickening (Fig. 3).

The *elastic material* of the medial tunic varied and could take different forms. We had a greater or lesser number of elastic fibers between the two limiting membranes, at times with a very fine reticule connecting the adventitia with the I.E.M. and providing a coherence in its parietal structure. These elastic fibers generally followed the concentric direction parallel to the smooth muscular cells. Its polymorphous presentation ranged in size from small undulated fibers to actual knots and elastic fibrous rolls.

2.- Morphometric study

The data are presented in table 1.

2.1. General morphometry

In the *normal arteries*, (Fig. 1) we would underline

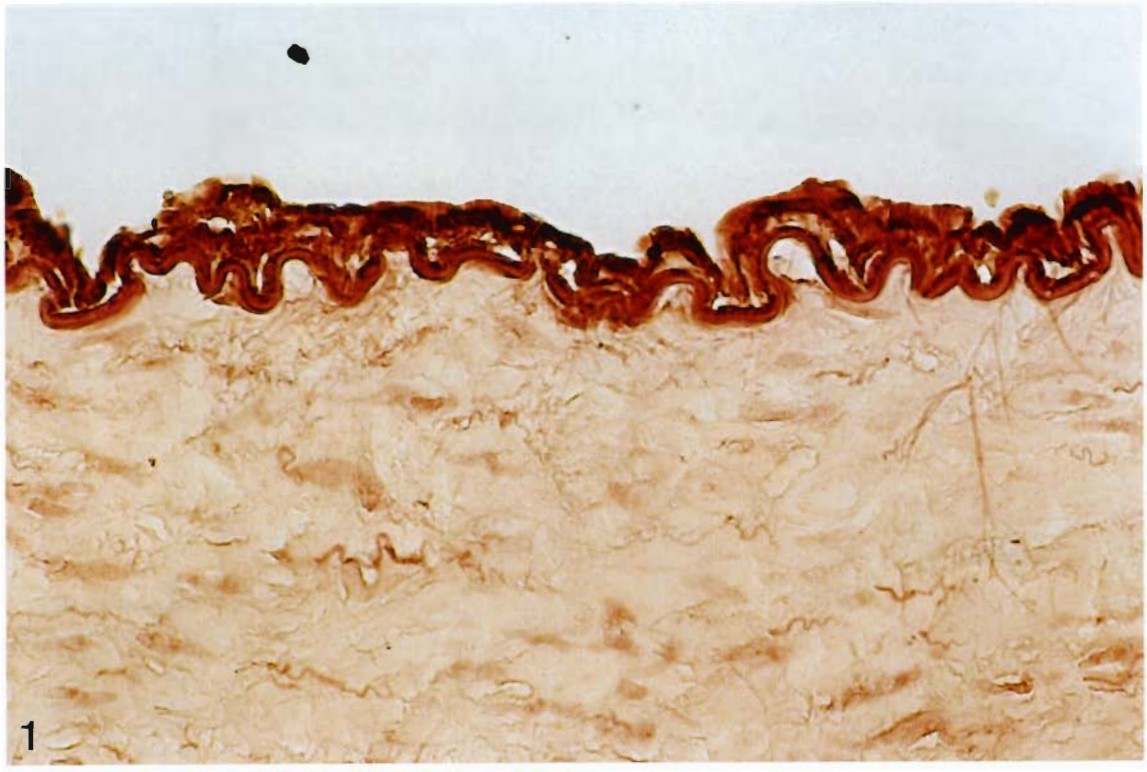


Fig. 1. Internal Elastic Membrane and medial tunic of the human splenic artery with normal appearance. Medial Tunic shows polymorphism of the elastic fibers, undulated, radial or forming knots. Concentric in direction of the smooth Muscular Cells of the arterial wall. Orcein. x 350

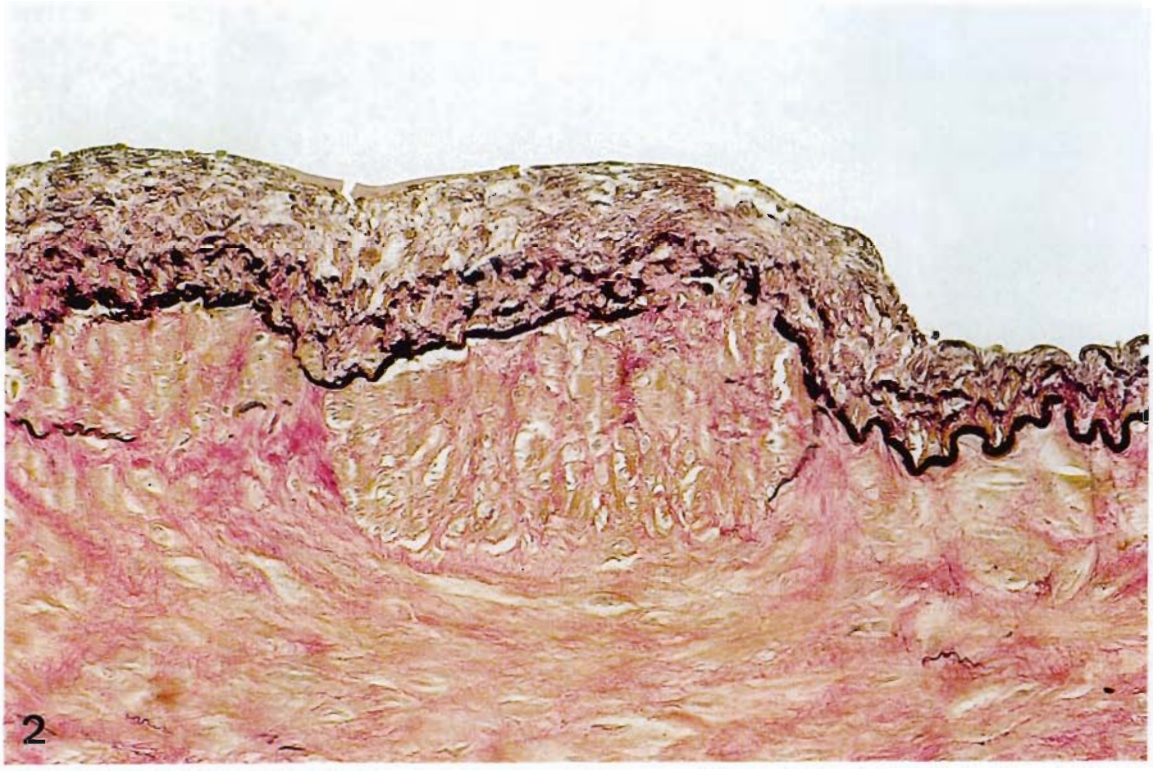


Fig. 2. Presence of lengthwise Muscular Columns in the medial layer into the arterial lumen. Formation of a localized intimal thickening. The scarce elastic content and the change of the direction of the Smooth Muscle Cells within the unserlying medial layer are evident. Verhoeff-Van Gienson. x 350

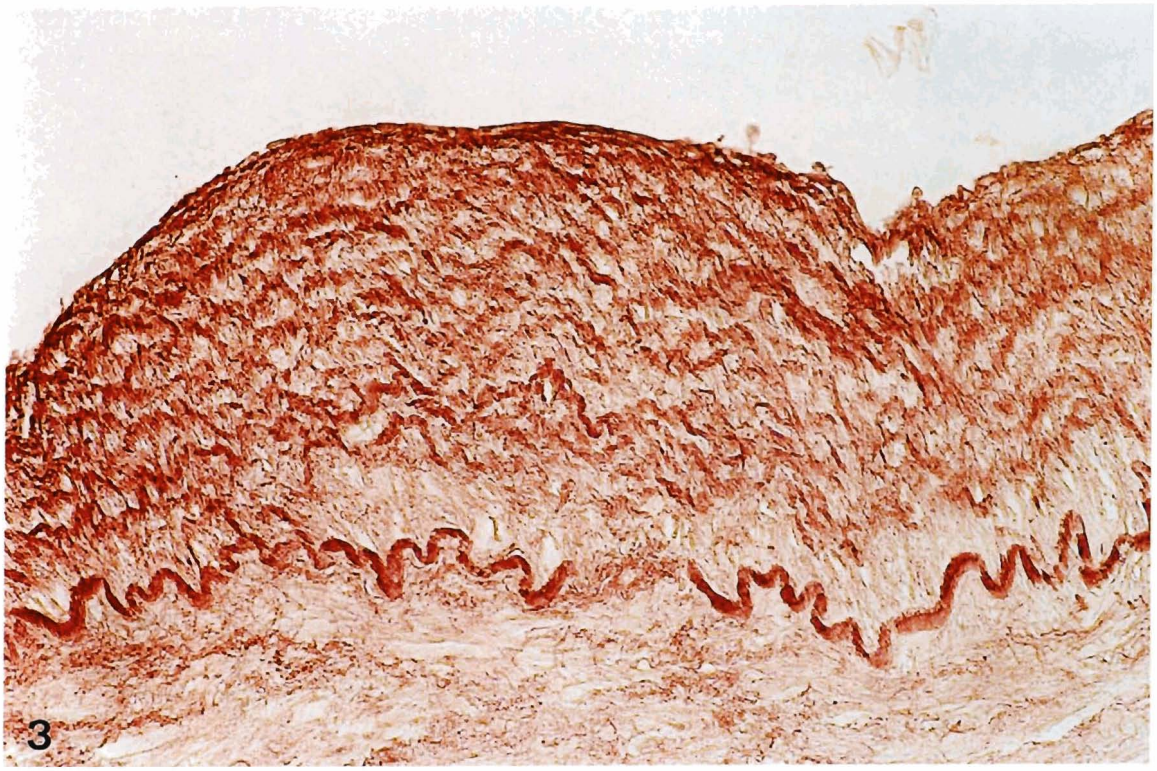


Fig. 3.
Fragmented and de-structured Internal Elastic Membrane in multiple lines of fraying. Orcein. x 350

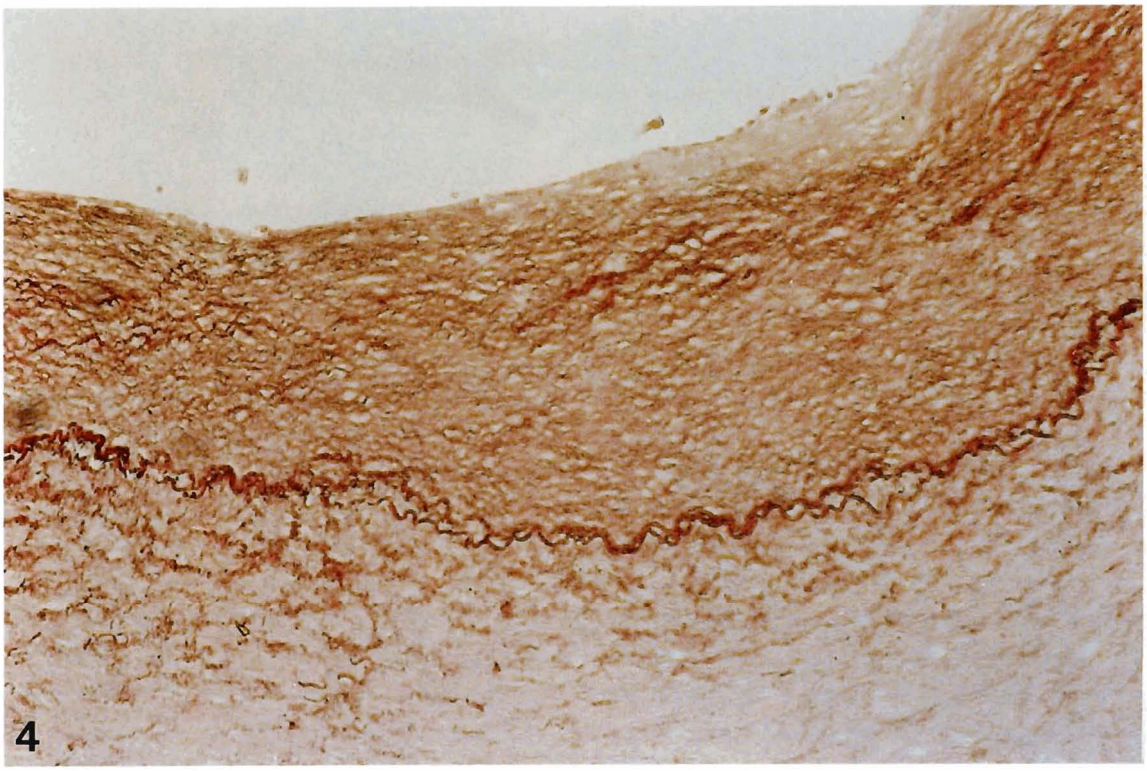


Fig. 4.
Development of an atheroma plaque on a eccentric intimal thickening. Orcein. x 350

Table 1. Summary chart of morphometric data in the human splenic artery.

	NORMAL ARTERY	ARTERY WITH MUSCULAR COLUMN	ARTERY WITH GENERALIZED THICKENING
Total area	4.08±2.25	3.33±1.12	6.43±2.68
Medial layer	2.56±1.74	1.99±1.19	3.52±1.13
Intimal layer	0.09±0.04	0.16±0.03	1.52±0.65*
Height of media	333±83	482±35	512±67
Height of intima	30±10	50±15	312±72*
Actual lumen	1.13±0.40	0.74±0.24	1.40±0.62
Theoric lumen	1.22±0.41	1.19±0.51	2.92±0.55*
R.L.I.	7.32±4.89***	46,57±10.58	52.26±9.25
I.T.I.	3.46±1.20	7.95±2.94*	43.22±7.86***
P.T.I.	1.36±1.02	2.17±1.26	5.63±1.06**
Elastolysis	19±9.0	25±12	69±18**
E.I.	5.87±0.16	5.61±2.61	17.99±4.32**
Elastin of media	15.08±7.03	10.62±0.90*	8.25±1.30**
Elastin of intima	74.28±8.25**	30.76±6.57	45.40±7.64

Surface of measurements in mm^2. Index and quantification color in %. Elastolysis in μm. *: $p<0.05$; **: $p<0.01$; ***: $p<0.001$.

that the indices (I.T.I. = 3.46%, P.T.I. = 1.36% and R.L.I. = 7.32%) are lower in this group compared with the arteries presenting muscular columns and generalized intimal thickening.

In the *arteries with muscular columns* (Fig. 2) the large I.T.I (7.95%) was greater than that of the normal arteries but considerably less than those which appeared in the generalized thickenings (43.22%). The P.T.I. (2.17%) resembled those of the normal arteries but less than in the arteries with generalized thickening (5.63%). The efficacy of reducing the arterial lumen was similar in this group to the arteries with generalized thickening, nearly 50%.

The muscular columns had a mean surface area of 396.206 μm^2 not included in the medial layer described above.

In the *arteries with generalized intimal thickening* (Fig. 4), we would point out the raised pathologic indices, specifically the I.T.I., P.T.I. and L.R.I. The rest of the values which were significantly raised compared to the other groups which appear in table 1, are shown in the appropriate sections.

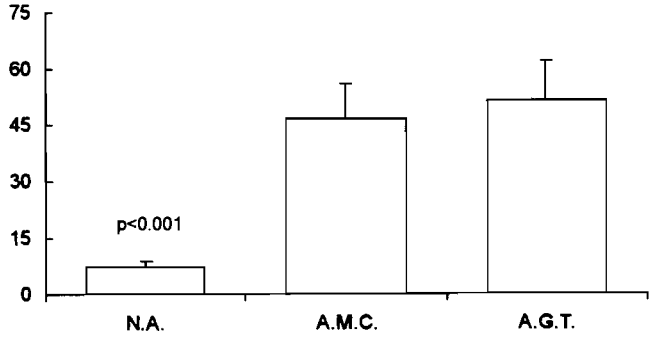
The *statistical study* of these values provides the following results:

Regarding the *total area of the artery* and *the thickness of the medial layer*, there was no significant statistical difference between the values of each group studied. The arterial size did not influence the appearance of muscular columns in the generalized intimal thickenings. There was a tendency for these thickenings to appear in the segments of the wider artery and in the most tortuous areas.

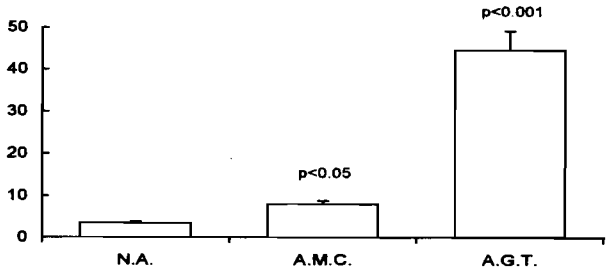
When we studied the behavior of the L.R.I. (Graph. 1) we found significant differences ($p<0.01$) between the L.R.I. of normal arteries compared to the arteries of the other two groups.

Regarding the *I.T.I.* (Graph 2) significant differences were established ($p<0.001$) between the non-diseased intimal arteries and with muscular columns compared to those that present generalized intimal thickening. The significance was less ($p<0.05$) between the arteries with and without muscular columns in the media.

The (*P.T.I.*) which compares the normal intima with the thickened one (Graph 4), confirmed significant differences ($p<0.01$) between the values for the arteries with generalized intimal thickening (5.63%) and the other two arterial groups (2.17% for the arteries with



Graph 1. LPI in the different arterial groups. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.



Graph 2. ITI in the different arterial groups. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.

muscular columns and 1.36% in the normal arteries).

2.2.Internal Elastic Membrane (IEM)

The *I.E.M.* normally presents a continuous, undulating form, with hardly an orifice or alteration in its structure. In disease conditions, this membrane appeared fragmented (Figs. 3, 4).

Elastolysis is defined as the distance between two fragments of altered I.E.M. which has lost its continuity. Elastolysis values obtained varied significantly ($p<0.01$) between the group of arteries with generalized intimal thickening (69±18 μm) and the arteries with muscular columns and without intimal disease (25±12 and 19±9 μm respectively).

The mean *contour* of the I.E.M. in the splenic arteries studied for a single area, was 404±122 μm, which was similar in all cases.

The relation between the contour and values of elastolysis, determines the so-called Elastolysis Index (E.I.). In the arteries with muscular columns this index was 5.61±2.61% and 5.87±0.16% in normal arteries. The E.I. of the arteries with generalized intimal thickening rose to 17.99±4.32%, determining significant differences ($p<0.01$) between this arterial group and the two previous ones (Fig. 3).

The mean thickness of the non-thickened intima was 36±17 μm, reaching levels of 279±70 μm, 8 times its value, in the arteries with generalized intimal thickening.

2.3. Special morphometry

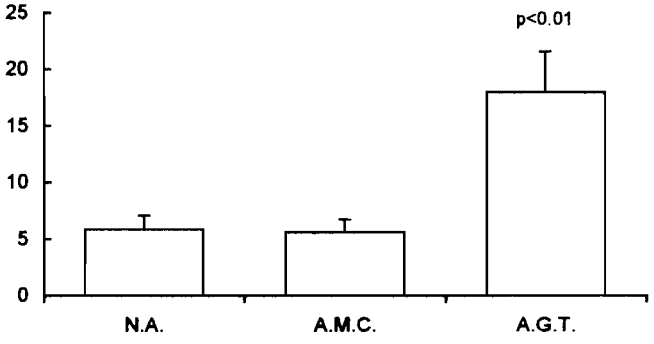
Quantification of color images: the elastin

We expand the data of table 1 with those referring to muscular columns.

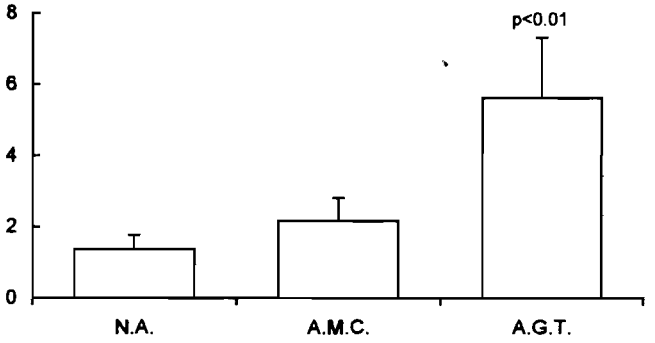
In the *splenic arteries with muscular columns in the media*, the elastin concentration in the medial layer was 15.34±5.97%. In the height of the muscular column it was 10.62±2.09% and in the non-thickened intimal layer (including the M.E.I.), 87.03±10.56%. In the localized intimal thickenings situated on the muscular column, the mean value of elastin was reduced to 30.76±6.57%.

In the *medial layer* (Graph 5) we obtained significant differences between the concentration of elastin present in the normal arteries compared to the elastin component of the muscular columns ($p<0.05$) observed in the thickness of the medial layer of the arteries with generalized intimal pathology ($p<0.01$).

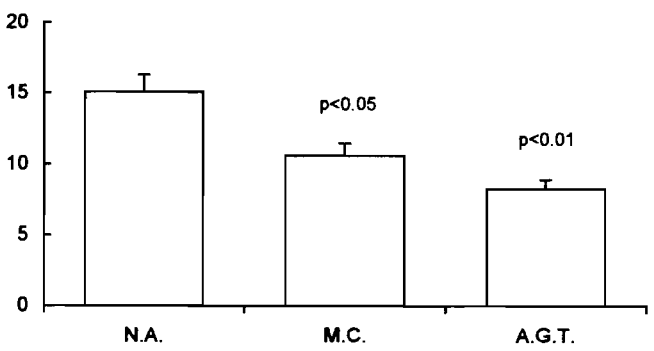
In the *normal intimal layer*, the concentration of elastin (including the M.E.I.) was significantly higher ($p<0.01$) than the one we found in the intima with



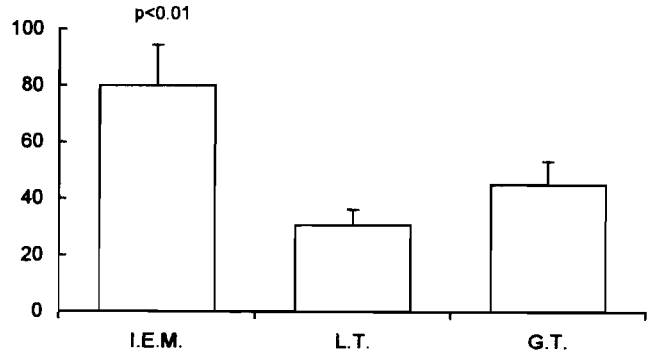
Graph 3. EI in the different arterial gropus. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.



Graph 4. PTI in the different arterial gropus. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.



Graph 5. Elastin of the medial layer in the different arterial groups. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.



Graph 6. Elastin of the intimal layer in the different arterial groups. N.A.: normal artery; A.M.C.: artery with muscular columns; A.G.T.: artery with generalized thickening.

localized or generalized thickenings (Graph 6).

Discussion

In arterial pathology, basic scientific studies aimed at considering the morphological-functional relation of its structural elements are limited in number and those carried out using human material are rarer still. The areas most commonly studied are limited to the coronary (Fuster et al., 1992), carotid (Guo et al., 1994), or cerebral arteries (Whiteand Bloor, 1992), without taking into account the important physiopathology of the arteries derived from the celiac trunk (Díaz et al., 1987).

Many of these studies are limited to the qualitative description of the elements which constitute the sound arterial wall in the selected area as well as the histopathologic alterations that appear as post-ischemia or desendothelization. Morphometry completes the previous studies through the quantitative statistics.

Morphometric studies of the arterial territory have been more or less sophisticated. The simplest are based on the measurement of areas with micrometers (Burrig, 1994; Malhotra et al., 1996) and studies in two dimensions with planimetry without a computer (Jellinek and Takács, 1995). Computers applied to arterial morphometry provide a notable advance by precisely calculating surfaces, diameters and contours. Programs are available for this purpose, as, for example, the MOP 3 image-analyze-system (Sisto and Isola, 1989), the Jandel Scientific's Computerized Morphometric image analyze system (Puglisi et al., 1995), the QUANTIMET 970 of Cambridge Instruments (Hellinger et al., 1995), the IKEGAMI VIP 21 CH of Kawasaki (Nishiyama et al., 1995). Other authors rely on very sophisticated mathematical models (Kassab et al., 1993) or morphometry through stereology (Avendaño et al., 1995).

One of the purposes of our study, in addition to the general histologic description of the human splenic artery and the measurement of surfaces and contours, was to ascertain the possible variations of elastin concentration in the different circunstances described. The QUANCOUL® system permits the morphometry of surfaces and the quantification of color images, as demonstrated in other studies (Daniel Lamazière et al., 1993) and clearly met our needs in this study for its reliability and user-friendly handling.

Our morphometric indices are based on the work carried out by Bürrig, Wilensky, Sisto and Puglisi who consider the media, the surface of the media, of the neo-intima and the arterial lumen, establishing quotients between the intima and media (Guo et al., 1994, 1995), similar to the I.T.I. presented here. The current studies of Nishiyama et al. (1995) and Malhotra et al. (1996) are also the most complete.

The most common disease reported in the human splenic artery is the aneurysm (Ohta et al., 1993; Billeter et al., 1994; Sidhu et al., 1995) where it is ranked third in frequency within abdominal arterial disease (Dogan et al., 1995). Alterations of the splenic arterial wall have also been described in diseases such as collagen, in mucoid degeneration of the medial layer or in cases of vascular fragility as with Cushing disease (Adami et al., 1993; Leu, 1993; Yoshitomi et al 1996).

In our study we have not considered the arteries with aneurysmatic illness as there are elements in their walls which could interfere with out results. Neither have we included anatomic variants as in those described by Sponza et al. (1993); Bertelli et al. (1995) and Higashi and Hirai (1995).

In surgery we have observed that the artery presented greater dilation and tortuousness in older patients, as described by Borley et al. (1995) and Sylvester (1995).

General morphometric data are slightly less than the actual ones as we have not used perfusion as a technique for setting the pieces.

The total mean area obtained corresponds to that of a main collateral artery of a primary trunk (4.2 mm^2), with a significant medial layer being a muscular-type artery.

The *theoretic arterial lumen* is wide but is reduced due to the presence of the muscular columns of the media which enter the lumen and by localized and generalized intimal thickenings, as the I.T.I. and L.R.I. demonstrate. These values are intermediate to those obtained in the facial (8.56% and 21.44%) and uterine artery (24.8% and 38.6% respectively) (Ortiz et al., 1996, 1997).

The *age factor* is always significant in arterial diseases as Sutton et al. (1994) and Hansen et al. (1994) have described. In our study, the average age was approximately 45 years. We have not been able to establish a statistical correlation between age and I.T.I. or L.R.I., although the tendency was to group the arteries of the older subjects with intimal thickenings, the wider and more tortuous arteries. We believe that the average age obtained in our case is less than what is necessary to obtain significant differences, usually beyond 55 or 60 years.

We have not found a significant correlation between the total area of the artery and the appearance of intimal thickening. Although we have observed that the generalized intimal thickenings are more common in the arteries of greater width and the localized in the areas with muscular columns protrusion.

The *L.R.I.* indicates that the muscular columns are as effective as the generalized intimal thickenings in reducing the width of the arterial lumen. We believe that the function of these muscular columns is to physiologically modify the blood flow of the artery, given the necessities of the organism. Its mechanism would be two-fold: on the one hand, the greater or lesser protrusion of the column toward the arterial lumen and, on the other hand, the shortening or lengthening of the column in lengthwise direction would also have a significant repercussion in the arterial width.

The generalized intimal thickenings significantly reduce the arterial lumen. But, unlike the muscular

columns, this reduction is fixed, disease-related and progressive, and difficult to reverse. The generalized thickening implies a state of serious structural alteration of the arterial wall and never a physiologic situation characteristic of the region.

The significant differences between the I.T.I. of the arteries with generalized thickening and the normal arteries, are attributable to definition of the index as this defines intimal disease. On the contrary, we highlight the presence of significant differences between the I.T.I. of the arteries with muscular columns and the normal arteries. If we consider that muscular columns are described as physiologic and normal elements of this territory, these differences should not exist. It is possible that muscular columns produce a setback in the integrity of the underlying intima, as if it were undergoing greater stress and reacting with a thickening.

The P.T.I. indicates that such an alteration is not as marked as in the cases of generalized intimal thickenings, as no significant differences were detected between the normal arteries and those of the muscular columns. These are established with the generalized arterial thickenings.

This does not rule out that, in addition to other destabilizing factors, the small localized thickenings may be the site of a progressive, generalized intimal degeneration over time.

It seems to confirm, as Porreca et al. (1994) and Lawrence et al. (1995) indicate, that in the pathogeny of the generalized intimal thickenings, the medial tunic, neointima and disorganized I.E.M are involved. The physiological pores or orifices in the I.E.M. interchange between the endothelium and the medial layer and habitually measure less than 25 μm. The lesion, rupture and fragmentation of the I.E.M. permits the passage and migration of the SMC in a process of cellular dedifferentiation (Ross, 1986; Thyberg, 1990) through its orifices or elastolysis as described by Fuster (1992) and Libby (1995).

Our study confirmed elastolysis values in the arteries with generalized intimal thickening are significantly higher (Table 1) than those in the other two arterial groups and consequently the E.I. This confirms the implication of the I.E.M. in the intimal processes and the classification of the muscular columns as physiological structural element of the human splenic artery.

The data obtained related to *elastin concentration* in this territory, confirms that it belongs to the group of muscular arteries where elastin is very localized at the level of its internal and external elastic membranes and limited in extension in its medial layer.

In the *medial layer*, the mean elastin concentration is 14%, a figure which could be considered slightly less than that obtained in the uterine artery. Both are also tortuous (Ortiz et al., 1997) where we obtained levels close to 20% and less if compared with arteries such as the aorta or the internal mammary, which have strong elastic content (Fuda et al., 1995).

We believe that the lowest concentration of elastin inside the muscular columns and in the medial layer of the arteries with intimal thickening compared with the elastin of the medial layer of normal arteries, has different origins:

In the *muscular columns* we believe there is a probable genetic determination favoring SMC which are contractile, very differentiated and low in the elastin production compared to the rest of the medial layer.

In the *medial layer of the arteries with intimal pathology*, the dedifferentiation of the SMC, the production of extracellular matrix and elastin degradation, implies its reduced concentration. The arterial wall therefore lacks a significant structural element in the maintenance of its integrity.

We consider the elastin's polymorphism as a protective element. The radial reticules, the undulated fibers or knots and fibril rolls described in the qualitative study, may favor a reduced rate of atherogenicity which would be raised if it were too dependent on its concentration (Díaz et al., 1988). The atheromatose zones are limited almost exclusively to the most tortuous zones. This reduction in atheromatosis allowed Cherqui et al. (1994) and Troppman et al. (1996) to use this artery in the revascularization of the transplants of pancreas and liver. Irgau et al. (1993) propose it as a useful artery in renal revascularization and Morgenstern and Mills (1993) in coronary bypass surgery, although with worse results than those obtained using those obtained using the internal mammary.

In addition, elastin is one of the *main components of I.E.M.* appearing in our study with a concentration of approximately 81%. This concentration is similar to the one described in other arterial regions.

In the *generalized and localized intimal thickenings,* the amount of elastin is reduced significantly, with values of 45%. This situation is easy to comprehend if we bear in mind that the elastin of the I.E.M. is fixed and the surface of the thickening progressively increases. Thus there is dispersion, fragmentation and fraying of the elastin as the neointima is established and the intimal thickening increases and becomes generalized.

In conclusion we may affirm that in the splenic artery we have observed a peculiar structure which are the muscular columns of the medial layer of longitudinal and spiral disposition. We believe that this morphologic element provides this artery with considerable stretching capacity in its two axis and variations in its internal width. This causes notable differences in its blood flow depending on the physiological necessities of the moment, where the spleen is a decisive organ.

These muscular column protrude their lumen as endoarterial pads o cushions, described in other arterial territories, such as the uterine artery (Whyte, 1996) where there are also great hemodynamic variations, cyclical in nature.

For the underlying intimal tunic, this means a situation of aggression which is more significant and is not suffered in other zones of the arterial intima, creating small localized intimal thickenings.

With age and other atherogenic elements, the splenic artery increases in width, dilates, increases it tortuousness and its hemodynamic character changes. In this case, the localized intimal thickenings gain ground over the normal intima and become more generalized, thus contributing to its atheromatose arterial destructurization and degeneration.

More in-depth study of the splenic artery may provide important data regarding atheromatose pathogeny and explain the cases of infarctions in the abdominal territory, which are not widely known.

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References

Adami P., Manzoni P. and Rohmer P. (1993). Evolutive aneurysms in type IV Ehlers-Danlos syndrome. Ann. Radiol. 36, 129-133.

American Heart Association SAC (1991). A definition of intima of human arteries and its atherosclerosis-Prone Regions. Arterioscler. Thromb. 12, 120-134.

Avendaño C., Roda J.M., Carceller F. and Díez-Tejedor E. (1995). Morphometric study of local cerebral ischemia in rats: a stereological evaluation. Brain Res. 673, 83-92.

Bertelli E., Di Gregorio F., Bertelli L. and Mosca S. (1995). The arterial blood supply of the pancreas: a review. The superior pancreaticoduodenal and the anterior superior pancreaticoduodenal arteries. An anatomical and radiological study. Surg. Radiol. Anat. 17, 97-106.

Billeter M., Franzeck U.K., von Segesser L. and Schoepke W. (1994). Inflammatory aneurysm of the splenic artery. Int Angiol. 13(2), 160-163

Borley N.R., McFarlane J.M. and Ellis H. (1995). A comparative study of the tortuosity of the splenic artery. Clin. Anat. 8, 219-221.

Bürrig K.F. (1994). The morphology of the carotid artery after uncomplicated endarterectomy. J. Cardiovascular Surg. 35, 413-418.

Cherqui D., Fiff Y., Julien M. and Fagniez P.L. (1994). The recipient splenic artery for arterialization in orthotopic liver transplantation. Am. J. Surg. 167, 327-330.

Daniel Lamazière J.M., Lavallée J., Zunino C. and Larrue J. (1993). Semiquantitative study of the distribution of two cellular antigens by computer-directed color analysis. Lab. Inv. 68, 248-252.

Díaz P., Torres A. and Sarrat R. (1987). Peculiaridades morfológicas de las arterias digestivas. Ann Anat. 33, 21-31.

Díaz P., Torres A. and Sarrat R. (1988). El tejido elástico como mecanismo protector frente a la arterioesclerosis. Anal. Anat. 34, 43-48.

Dogan R., Demircin M., Pasaoglu Y., Onat D.A. and Kutluay L. (1995). Surgical treatment of splanchnic artery aneurysms secondary to medial degeneration. Thorac. Cardiovasc. Surg. 43, 230-233.

Fawcett D.W. (1992). Sistemas vasculares sanguíneo y linfático. In: Tratado de histología. Bloom-Fawcett. 11 ed. Interamericana-McGraw Hill. Madrid. pp 369-409.

Fuda P., Pigatto A., De Gasperis C., Maselli D. and Cannas M. (1995). Structural and morphometric analysis of the internal mammary artery used in coronary bypass surgery. Minerva Cardioangiol. 43, 21-27.

Fuster V., Badimón L., Badimón J. and Chesebro J.H. (1992). The pathogenesis of coronary artery disease and the acute coronary syndromes. New Engl. J. Med. 326, 242-250.

Glagov S. and Zarins C.K. (1989). In intimal hyperplasia an adaptative reponse or a pathologic process? Observations on the nature of nonatherosclerotic intimal thickening. J. Vasc. Surg. 10, 571-573.

Guo J., Milhoan K.A., Tuan R. and Lefer A.M. (1994). Beneficial effect of SPM-5185, a Cysteine-containing NO donor, in rat carotid artery intimal injury. Circ. Res. 75, 77-84.

Guo J., Panday M.N., Consigny M. and Lefer M.A. (1995). Mechanisms of vascular preservation by a novel NO donor following rat carotid artery intimal injury. Am. J. Physiol. 269, 1122-1131

Hansen B.F., Mortensen A., Hansen J.F., Ibsen P., Frandsen H. and Nordestgaard B.G. (1994). Atherosclerosis in Watanabe heritable hyperlipidaemic rabbits. Evaluation by macroscopic, microscopic and biochemical methods and comparison of atherosclerosis variables. APMIS 102, 177-190.

Hellinger A., Konerding M.A., Obertacke U., Redl H., Bruch J. and Schlag G. (1995). Does lung contusion affect both the traumatized and the noninjured lung parenchyma? A morphological and morphometric study in the pig. J. Trauma. 39, 712-719.

Higashi N. and Hirai K. (1995). A case of the three branches of the celiac trunk arising directly from the abdominal aorta. Kaibogaku Zasshi. 70, 349-352.

Irgau Y., Fellows B.A. and Rosenbloom M.S. (1993). Splenorenal bypass for right renal artery revascularization. Ann. Vasc. Surg. 7, 359-362.

Jellinek H. and Takács E. (1995). Morphological aspects of the effects of orotic acid and magnesium orotate on hypercholesterolaemia in rabbits. Arzneim.-Forsch./Drug Res. 45 II, 836-842.

Kassab G.S., Rider C.A., Tang N.J. and Fung Y. (1993). Morphometry of pig coronary arterial trees. Am. J. Physiol. 265, H350-365.

Lawrence M., Watson L., Pettijohn T., Doyle T., Tasca S.I. and Stoica G. (1995). Immunohistochemical localization of proliferating cells, basic fibroblast growth factor and Fos protein following iliac artery balloon angioplasty in hypercholesterolemic rabbits. In Vivo 9, 27-34.

Leu H.J. (1993). Spontaneously dissecting aneurysms of arteries of muscular composition due to mucoid media degeneration. Pathologe 14, 325-329.

Libby P. (1995). Molecular bases of the acute coronary syndromes. Circulation 91, 2844-2850.

Malhotra R., Bedi H.S., Bazaz S., Jain S. and Trehan N. (1996). Morphometric analysis of the right gastroepiploic artery and the internal mammary artery. Ann. Thorac. Surg. 61, 124-127.

Morgenstern D.A. and Mills N.L. (1993). News conduits for coronary artery bypass. Coronary Artery Dis. 4, 677-681.

Nishiyama Y., Manabe N., Ooshima A., Miyatake S., Adachi T., Takahashi K., Kanda Y., Hiai H. and Fukumoto M. (1995). A sporadic case of Ehlers-Danlos syndrome type IV: Diagnosed by a morphometric study of collagen content. Pathol. Int. 45, 524-529.

Oguzkurt L., Balkanci F., Ariyurek M. and Demirkazik F.B. (1996). Traumatic aneurysm and arteriovenous fistula of the splenic artery. Pediatr. Radiol. 26, 195-197.

Ohta M., Hashizume M., Sugimachi K. and Yasumori K. (1993). Splenic hyperkinetic state and splenic arterial aneurysms in portal hypertension. Hepatogastroenterology 39, 529-532

Ortiz P.P., Daniel-Lamazière J.M., Lavallée J., Lavie J., Alconchel L.

Whyte J. and Sarrat R. (1996). Etude morpho-fonctionnelle de l'artère faciale humaine. Arq. Med. 10 (Supl. 7), 19.

Ortiz P.P., Whyte J., Díaz P., Tierz J.A., Torres A., Daniel-Lamazière J.M., Lavallée J. and Sarrat R. (1997). Estudio morfométrico de la arteria uterina humana. Acta Ginecol. (in press).

Ortiz P.P., Díaz P., Torres A., Whyte J., Daniel-Lamaziére J.M., Lavallée J., Bonnet J. and Sarrat R. (1997) Quantitative study of elastin in human arteries with high level of tortuosness. Eur. J. Anat. (in press).

Porreca E., Ucchino S., Di Febbo C., Di Bartolomeo N., Angelucci D., Napolitano A.M., Mezzetti A. and Cuccurullo F. (1994). Anti-proliferative effect of desferrioxamine on vascular smooth muscle cells in vitro and in vivo. Arterioscler. Thromb. 14, 299-304.

Puglisi R.N., Whalen T.V. and Doolin E.J. (1995). Computer analyzed histology of ischemic injury to the gut. J. Ped. Surg. 30, 839-844.

Ross R. (1986). The pathogenesis of atherosclerosis an update. New Engl. J. Med.14, 488-500.

Sidhu P.S., Khaw K.T. and Belli A.M. (1995). Anomalous splenic artery aneurysm: demonstration on CT scanning and angiography. Postgrad. Med. J. 71, 49-51.

Sisto T. and Isola J. (1989). Incidence of atherosclerosis in the internal mammary artery. Ann. Thorac. Surg. 47, 884-886.

Sponza M., Pozzi R. and Pozzi Mucelli F. (1993). Arterial anatomy of the celiac trunk and the superior mesenteric artery with computerized tomography. Radiol. Med. 86, 260-267.

Sutton J.M., Ellis S.G., Roubin G.S., Pinkerton C.A., King S.B., Raizner A.E., Holmes D.R., Kereiakes D.J. and Topol E.J. (1994). Major clinical events after coronary stenting. The multicenter registry of acute and elective Gianturco-Roubin stent placement. The Gianturco-Roubin Intracoronary Stent Investigator Group. Circulation 89, 1126-1137.

Sylvester P.A., Stewart R. and Ellis H. (1995). Tortuosity of the human splenic artery. Clin. Anat. 8, 214-218.

Thyberg J., Hedin U., Sjölund M., Palmberg L. and Bottger B.A. (1990). Regulation of differenciated properties and proliferation of arterial smooth muscle cells. Arterioesclerosis 10, 966-90.

Troppman C., Gruessner A.C., Benedetti E., Papalois B.E., Dunn D.L. and Gruessner R.W. (1996). Vascular graft thrombosis after pancreatic transplantation: univariate and multivariate operative and nonoperative risk factor analysis. J. Am. Coll. Surg. 182, 285-316.

Velican C. and Velican D. (1977). Studies on human coronary arteries. Branch pads or cushions. Acta Anat. 99, 377-385.

White F.C. and Bloor C.M. (1992). Coronary vascular remodeling and coronary resistance during chronic ischemia. Am. J. Cardiovasc. Pathol. 4, 193-202.

Whyte J., Díaz P., Tierz J.A, Pellejero S., Lostalé F., Ortiz P.P., Torres A. and Sarrat R. (1996). Engrosamientos intimales en la arteria uterina humana. Acta Ginecol. 53, 13-20.

Yoshitomi Y., Yoshimi H. and Yutani C. (1996). A case of splenic artery aneurysm with Cushing's syndrome. Int. J. Cardiol. 54, 263.

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