Exaggerated arterial curves due to endovascular interventions: Novel concept that describes how curves in the arterial wall affect the formation of neointimal hyperplasia, and explains why the long-term results of endovascular interventions are inferior to the long-term results of open surgical procedures

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ABSTRACT

Despite the vast advancement made in endovascular practice, it is still lacking the long-term success of open surgery in treating critical limb ischemia. In Bypass surgery excess length of the graft is cut to straighten it between the arterial anastomoses to avoid any excess length, in order to make sure that there will be no curves, but in endovascular approach this option is not available. In endovascular procedures, there are factors affecting negatively the long-term results. These factors contribute to excess length of the arterial wall, which is exaggerated after balloon dilatation. With deployment of stent, the stent straightens the arterial segment, so any extra length shifts proximal and distal to this segment leading to exaggeration of curves in arterial wall which affect hemodynamics of blood flow leading to neointimal hyperplasia and restenosis even at the edges of stent. This explains neointimal hyperplasia and restenosis at the edges of stent graft although its edges are landing on a normal artery. New solution for this durability problem of endovascular procedures is to avoid excess length. It can be achieved through a kind of stent that abolishes curves from the arterial tree.

Introduction

Peripheral vascular disease is a very frequent disease caused mainly by atherosclerosis and much less frequently by other causes. In the process of progression of Atherosclerosis the loss of arterial wall elasticity is a very important factor in developing peripheral vascular disease [1].

Open surgical bypass was the gold standard in the treatment of patients suffering from peripheral vascular disease in critical limb ischemia patients. Then the Endovascular procedures proved efficiency in treating such conditions without the postoperative morbidity caused by surgery [2]. Surgical bypass either using Saphenous vein graft or synthetic graft have a very good long term results of patency of the lumen of the artery compared to endovascular procedures. This problem of durability of the results of endovascular interventions lead to an evolution of endovascular practice from using only balloons to dilate the stenosed or occluded segments of the artery to a variety of procedures many of which include deployment of stents to avoid the recoil of the artery or the flow limiting dissection or the application of stent grafts to avoid the effect of neointimal hyperplasia which causes instent stenosis [3,4].

This shows that endovascular procedures had a long way of advancing the tools and techniques in order to increase their results durability in relation to open surgery. But despite the vast advancement in endovascular practice and the investments made to optimize their long term results, these long term results could not match that of the surgical procedures [3,4]. Even with the use of stent graft which avoids the development of instent stenosis due to neointimal hyperplasia because of the covering of stent shaft which isolates the lumen from the inflammatory process in the arterial wall, but still it was found that there is neointimal hyperplasia at the proximal and distal edges of the stent graft resulting in stent edge stenosis, although that the edges of the stent graft are landing on a relatively normal segment of the artery [5,6].

Which should raise our attention to the fact that there is still something important missing. We need to question the understanding of the reason why endovascular procedures are still behind the surgical bypass in terms of long term patency.

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Novel concept explaining the reason of inferior long-term results of endoasular interventions in relation to surgery

In the beginning we should know that in bypass surgery either with reversed saphenous vein graft or synthetic graft for treatment of femoropopliteal segment stenosis or occlusion, we cut any excess length of the graft to straighten it between the 2 arterial anastomoses to avoid any extra length, in order to make sure that there will be no curves which will affect the success of the bypass, but in endovascular approach, we do not have this option to cut the excess length [7].

Proof of extra length added to the arterial wall caused by atherosclerosis

As mentioned before, in saphenous vein or synthetic graft bypass surgery, it is mandatory to cut any excess length, so the graft will be without any curves to avoid disturbance of the blood flow [7].

During the procedure of carotid endarterectomy using the eversion technique, after the removal of atheroma from the internal carotid artery, there is excess length and redundancy in the arterial wall, so we remove the excess length from the transected internal carotid artery to shorten the artery before making the anastomosis [8].

In a magnetic resonance angiography study of the femoropopliteal segment, during the knee flexion, it was found that in older participants of the study there was elongation in the arterial wall which was manifested in the form of curves and some of these curves did not disappear during knee extension in people aged 60 years or older and that elongation and tortuosity in the vessel wall is age dependent [9].

In subintimal angioplasty the artery does not rupture from the balloon inflation although the balloon is inflated under high pressure and the outer wall is already weak because it is only a part of the wall, but yet it does not rupture, because of the excess length and redundancy of the wall that expands with the balloon inflation is preventing the rupture of arterial wall [10].

Explanation of extra length of the arterial wall due to atherosclerosis that leads to formation of curves which are exaggerated after endovascular procedures

The normal arterial wall is elastic, but with the progress of the pathogenesis of atherosclerosis the arterial wall loses its elasticity. Then further progression leads to formation of atheromatous plaque. As a compensatory mechanism there is a dilatation of the artery to compensate hypoxia till maximum vasodilatation. This dilatation adds to extra length, because the vessel permanently dilates circumferentially with the same length of arterial tree [11]. There is no space to accommodate this extra length, so the wall of the artery form curves.

In case of normal arteries the pulse wave dilates the vessel which means it presses it outwards, but in an atherosclerosed artery which has already lost its elasticity the pulse wave presses the vessel wall out and distal, because part of the energy of the pulse wave is lost in the friction with the more stiff wall which causes it to move with the direction of the pulse wave. So when the arterial wall becomes redundant from lost elasticity, and dilatation of the artery due to hypoxia, it elongates which will form curves in the arterial wall. These curves will be more manifest and exaggerated after endovascular procedure, because after dilatation of the stenosis or occlusion of the artery with a balloon, we do not have the option to cut any extra length in the arterial wall but we only can shift this extra length to be accumulated proximal and distal, causing the curves to become more manifest and exaggerated proximal and distal to the site of the dilated lesion which affect the hemodynamics of blood flow leading to neointimal hyperplasia which leads to vessel restenosis.

Effect of curves on hemodynamics of blood flow, and their effect on formation of neointimal hyperplasia and restenosis

The extra length which will be manifested as curves will be on different levels of the artery especially where the artery is relatively has less ability for mobility such as at the sites of collaterals or bifurcations which explains the famous sites of atheroma formation at the branching sites.

The sites of branching in the arterial tree are sites where there is change of diameter and sites for relative limitation of movement of arteries with the pulse wave, so when the pulsatile wave hits the arterial segment at the site of the branches it moves a little bit slower than the rest of the artery which results in more ability to form curves or make the already existing curves more manifest which affect the shear stress forces at this segment. Curves disturb the laminar flow of the blood in the artery leading to low wall shear stress at the sites of the curves which leads to smooth muscle cell migration from media to intima leading to intimal hyperplasia [12].

Stent fracture can be caused due to curves. With the movement of the leg, the superficial femoral artery moves and the curves exert pressure on the stent, or instead the curves with the movement of the artery it can make a kind of friction with stent leading to neointimal hyperplasia, and vessel wall trauma especially at the proximal and distal ends of the stent [13].

Exaggerated curves after endovascular interventions

After treating the arterial lesion with endovascular balloon dilatation, this shifts the extra length in the form of curves partially into another segment proximal and distal to the treated segment of the artery, and part of this extra length will be manifested as recoil after balloon dilatation. In case of stent deployment, the stent straightens the vessel wall, shifting all the extra length proximal and distal, making the curves more manifest and exaggerated proximal and distal to the stent, which results in low wall shear stress leading to neointimal hyperplasia and restenosis at the sites of the exaggerated curves. Even with the deployment of a stent graft, which avoids the instant stenosis by isolating the lumen from the arterial wall, this will not solve the problem of exaggerated curves proximal and distal to the stent graft. This explains why there is neointimal hyperplasia and restenosis at the edges of stent graft although the edges of the stent graft are landing on a relatively normal part of the artery.

These exaggerated curves affect negatively the long-term results of endovascular interventions even with the vast advancement in the endovascular practice.

New proposed solution for the problem of curves to avoid neointimal hyperplasia, in order to optimize the long-term results of endovascular interventions

We should not think about treating only the lesion but the artery, we should think about the diseased artery as a whole.

The success of abolishing curves must not be done by trying to straighten the artery at one segment, because this will push the excess length proximal and distal leading to exaggeration of curves proximal and distal to the treated segment. We need to abolish the excess length by another way.

How to do that?! It is a matter of new ideas about how to prevent the curves from becoming exaggerated after endovascular procedures, in order to improve the long-term results of endovascular interventions.

For me, I believe it can be done through a kind of stent with an external part at certain level of the stent that pushes out the arterial wall. It should exert a permanent dilatation of the wall at the level of the stent to consume this extra length by pushing it to the outside, in order to prevent the extra length from being shifted proximal and distal by the endovascular intervention.
This external part of the stent can be like a space covered by semipermeable membrane that can be fully expanded by the blood, and presses to the outside of the artery for example a covered stent with external circumferential or spiral parts. Or the external part can be a metal part designed in a special way allowing it to hold the extra length to the outside. There are many ideas that can be discussed later in an extensive way.

**Conclusion**

Curves have very important effect on hemodynamics in the arteries. In open surgery we have the option to straighten the graft and avoid any curves which is not available in Endovascular procedures.

There are factors that affect negatively the long-term results of endovascular interventions. These factors which result in excess length of the arterial wall include redundant arterial wall from the loss of elasticity, and maximal dilatation of the arterial wall. This excess length results in formation of curves in the arterial wall and then these curves are exaggerated by the dilatation of the affected arterial segment with the balloon. When we deploy a stent, this results in straightening of this arterial segment, and the excess length which form curves will be shifted proximal and distal to this segment which means that the excess length accumulates proximally and distally, which results in making the curves more manifest and exaggerated proximal and distal to the stented segment of the artery, which affects the hemodynamics of blood flow leading to low wall shear stress at the sites of exaggerated curves which results in neointimal hyperplasia and restenosis. So the problem will not be solved even if we use a stent graft where neointimal hyperplasia is found at its edges due to the exaggerated curves which are formed proximal and distal to the stent graft.

In order to improve the long-term results of endovascular procedures, we need to think differently. We can make the long-term results of endovascular interventions not less than those of open surgery, and this can be achieved by preventing this extra length from exaggerating the curves in the arterial wall. A new kind of stent that can abolish the effect of curves is a matter of discussion.

**Conflict of interest**

None to declare.

**References**