

Microvasculature of the Unconditioned Latissimus Dorsi Muscle after Vascular Delay and Postmobilization for Cardiomyoplasty

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Abstract

Vascular deafferentation of the Latissimus Dorsi Muscle (LDM) for dynamic cardiomyoplasty is an important cause of ischemia in the distal portion of the muscle with degeneration of muscle fibres and consequent reduction in the effectiveness of the surgery.

Experimental studies suggest that a vascular delay procedure and a 10-day delay adaptation significantly improve the distal perfusion and function of the LDM flap for cardiomyoplasty.

We here report a comparative histological and ultrastructural study of biopsies from unconditioned LDM flaps of two patients, taken 30 days after vascular deafferentation and dynamic cardiomyoplasty, and 10 days after a vascular delay procedure, respectively.

In the first case, in the distal part of LDM we found muscle atrophy and degeneration, necrosis of capillaries, with swelling of endothelial cells and disruption of cytoplasmic organelles, and capillary neoangiogenesis. In the second patient, the distal LDM after vascular delay procedure show minimal muscle fibre degeneration, dilatation of capillaries with endothelial cell preservation, and scarce capillary neoangiogenesis.

The present results seem to confirm that a vascular delay procedure on LDM for use with cardiomyoplasty may improve the LDM perfusion and function, inducing moderate morphological changes in muscle fibres and vasculature in comparison to the deafferented LDM.

Key words: vascular delay, dynamic cardiomyoplasty, Latissimus Dorsi flap, damage.

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Dynamic cardiomyoplasty (DC) is a surgical procedure in which a pedicled Latissimus Dorsi Muscle (LDM) flap is transposed into the chest and wrapped around the ventricles of a failing heart. The skeletal muscle is electrically stimulated to contract synchronously with the heart using a special training protocol for cardiac assist in patients with congestive heart failure [3, 13].

Intraoperative deafferentation of perforant arteries (derived from intercostal and lumbar vessels) partially deprive the LDM of the afferent lateral vasculature. It is evident that muscle fibres and microvasculature undergo changes and adaptation after surgery.

Ischemic damage of the distal portion of LDM occurring in muscle transfer for cardiomyoplasty has been described [2, 4, 9, 10, 12, 15, 16].

Evidence of inadequate revascularization and long term muscle damage, with interstitial fibrosis in the distal portion of the wrapped muscle has been also observed [13, 14]. Recently a vascular delay procedure has been performed to preserve the viability of the distal part of LDM for cardiomyoplasty [5, 10].

The present study designed to analyze morphological and electron microscopic changes of small blood vessels from the unconditioned LDM for cardiomyoplasty in 2 patients, in the early postoperative period (30 days after deafferentation) and after a 10-day vascular delay procedure, respectively.

Materials and Methods

The morphological study was performed on the LDM muscle from 2 patients.

In the first case (a male patient aged 39 years), muscle specimens were collected from the left deafferented LDM immediately transferred for cardiomyoplasty. The patient died suddenly 30 days after surgery. At autopsy the left LDM appeared well wrapped to epicardium and viable. A total of 10 specimens were collected from the proximal and distal part of the pedicled graft, taking care to include 4 specimens from the heart-muscle interface. Ten specimens were obtained from the contralateral normal LDM and were used as control for morphometric analyses.

LD vascularization in cardiomyoplasty

In the second case, a total of 6 biopsies were taken from the proximal and distal portion of the left unconditioned LDM of a male patient aged 48 years. The LDM was subjected to a vascular delay procedure [2, 5] and transposed around the heart for cardiomyoplasty 10 days later.

All muscle samples were immersed for 4 hr at 4°C in Karnovsky fixative solution and then postfixed in 1% OsO₄ for 1/2 hour. After dehydration in graded alcohols the specimens were embedded in Epon 812. Semithin sections were stained with toluidine blue and examined at the light microscope. Ultrathin sections contrasted with 3% uranyl acetate and lead citrate were examined at a Philips EM 910 electron microscope. Morphometric analysis of capillaries was carried out on semithin sections by measuring the patent capillaries as capillary/fibre ratio by counting in semithin sections under low magnification the number of capillaries around a muscle fibre in at least 30 fibres.

Results

a) Post-mortem specimens obtained from deafferented LDM 30 days after cardiomyoplasty revealed different patterns in the proximal and distal part of the muscle.

The proximal part of LDM was composed by small fascicles of round or polygonal muscle fibres, with moderate variation in muscle fibre size (mean diameter 36 ± 2.2). Interposed lobules of fat tissue were present as 16% of the total cross sectional area. Distribution of intramuscular capillaries and small blood vessels was normal. Capillaries were always patent and sometimes dilated and their morphology was normal.

Specimens obtained from the distal area of deafferented LDM of the same patient showed muscle fascicles composed by polygonal fibres with evident variation in size (mean fibre diameter: 32 ± 4.6). Fat tissue was present as 20% of the total cross sectional area. Some fibres were atrophic and numerous fibres showed internal nuclei. Semithin sections examined under light microscopy showed dilated intramuscular capillaries; some of them showed degeneration or necrosis of the endothelial cells. At the electron microscopic examination numerous capillaries showed necrosis of the wall, consisting in swelling of endothelial cell and disruption of cytoplasmic organelles. The endothelial cell nuclei show poor and loose chromatin, with dispersed areas (Fig 1a, b, c). Capillaries with ultrastructural patterns referred to neoangiogenesis were also observed.

b) Specimens from biopsies of the distal part of LDM subjected to a 10-day vascular delay procedure showed fascicles composed by polygonal fibres with moderate variation in size (mean fibre diameter 30 ± 4.0). Fat tissue was present as 18% of the total cross sectional area. Some atrophic fibres were seen at the periphery of the flap. Capillaries were frequently dilated but no degeneration or necrosis of the wall was observed. In scarce capillaries,

endothelial cells showed microvilli or cytoplasmic protrusions of luminal plasmalemma (fig. 1d). Isolated patterns of neoangiogenesis were seen.

Morphometry

In Table 1 the studied parameters of LDM capillarity from the patients are reported.

Measurements were obtained from normal contralateral LDM and from the distal part of deafferented LDM 30 days after cardiomyoplasty, and on biopsies from LDM subjected to a 10 day vascular delay procedure in the second patient. The comparison between capillary morphometry in the distal part of deafferented LDM and the LDM subjected to vascular delay showed preservation of the capillary/fibre ratio in the last condition.

Discussion

Since first experiments [3], about 800 patients had cardiomyoplasty and they demonstrated highly variability in the hemodynamic response after surgery [15].

In the past, some authors showed that intraoperative deafferentation of perforant arteries deprive the LDM of a part of the afferent vasculature and evidence of inadequate revascularisation and long term muscle damage in the distal portion of the wrapped muscle was demonstrated [13, 14, 16].

In these conditions ischemia of the distal portion of the LDM flap for cardiomyoplasty may reduce muscle contractility and electrical conditioning.

In the present study, necrosis of capillaries was seen in the distal portion of the unconditioned LDM in the early postoperative period. The vascular changes occurred as consequence of surgical deafferentation and reperfusion. It is possible that reperfusion may occur after opening of anastomoses between the thoraco-dorsal circulation and the resting intramuscular vasculature. Regeneration of some capillaries, mainly in form of neoangiogenesis was also observed in the areas of impaired vascularization.

Microvascular injury occurring after LDM deafferentation was accompanied with the occurrence of capillaries with ultrastructural changes indicative of regeneration or neoangiogenesis. In the present study, the occurrence of microvilli and protrusions of luminal plasmalemma of endothelial cells of preserved capillaries was evident; this pattern was considered to be a cellular response to altered hemodynamic conditions [17]; the functional significance

Table 1. Capillary/fibre ratio (C/F) on normal LDM (a), on the distal part of transposed LDM 30 days after cardiomyoplasty (b) and on the distal part of LDM subjected to a 10 day vascular delay period and then used for cardiomyoplasty (c).

a) Normal contralateral LDM	C/F: 1.2 ± 0.4
b) Distal portion of deafferented LDM	C/F: 0.8 ± 0.2
c) Distal portion of LDM after vascular delay	C/F: 1.0 ± 0.2

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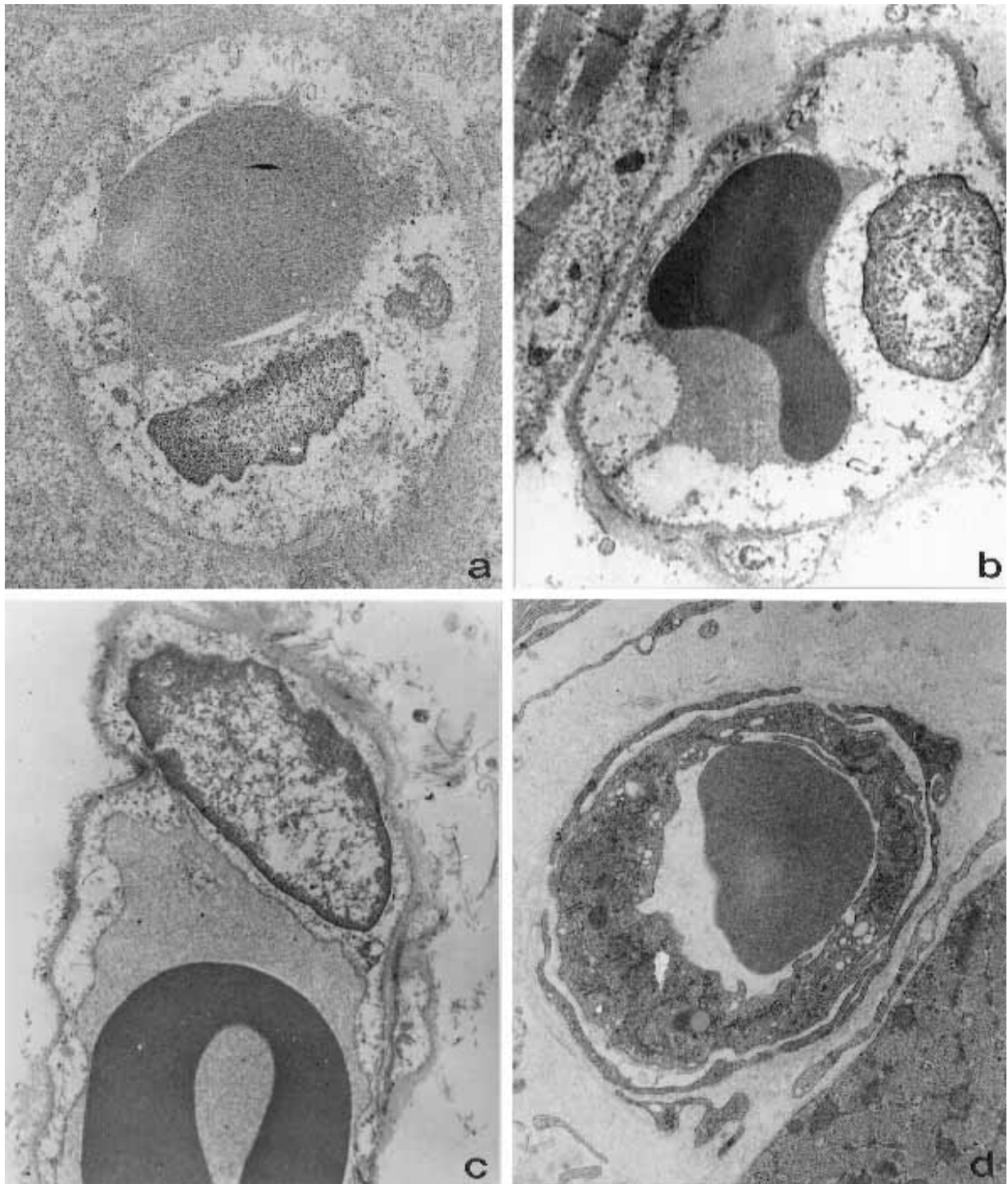


Figure 1. a-b. Swelling of endothelial cells, disruption of cytoplasmic organelles and nuclear chromatin changes are evident in a small intramuscular vessel from the distal portion of the deafferented LDM. Case 1. Electron microscopy. X 10,500. c. Necrosis of the capillary wall in the distal part of deafferented LDM. The nuclear chromatin is partially preserved. Case 1. Electron microscopy. X 10,500. d. Normal appearance of an intramuscular small vessel characterized by large endothelial cells showing numerous ribosomes. Rare villous-like endothelial cell processes are present at the luminal surface. Distal part of LDM subjected to vascular delay procedure. Case 2. Electron microscopy. X 10,500.

of endothelial microvilli and protrusions is not clear but it seems related to the tendency of cells to increase its surface area after hypoperfusion conditions [7].

The phenomenon of reperfusion is very important in the pathogenesis of muscle damage. In fact is well demonstrated that the resistance to reperfusion in skeletal

muscle after ischemia (so called the no-reflow phenomenon) is an important cause of cellular damage [8, 11]. After ischemia numerous capillaries show functional changes as increase in permeability, reduction of peak hyperemic reflow, and tissue edema. These alterations lead to an increase in the capillary vascular resistance reducing the post-ischemic reperfusion. Endothelial cells, platelets and leucocytes are activated under hypoxic conditions and contribute to the development of tissue injury. In experimental models of peripheral ischemia an increase in the vascular resistance was found even in the absence of morphological changes [1, 6].

Recently a vascular delay procedure was used to preserve the viability of LDM for cardiomyoplasty [2, 5, 10]. In this procedure the perforating vessels from the chest wall supporting the middle and distal part of the LDM were severed 10-15 days prior the standard cardiomyoplasty.

In the present study the vasculature of the LDM subjected to a 10-day vascular delay period showed less degeneration of muscle fibres and diffuse dilatation of capillaries in the distal part of the muscle. Necrosis of capillaries and neoangiogenesis was exceptional.

Though the analysis of LDM in the studied cases was performed at different times (30 days after CD and 10 days of vascular delay), the present study supports the results obtained by others [2, 5, 10] and suggests that vascular delay procedure can improve the perfusion of LDM for cardiomyoplasty.

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