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RESEARCH ARTICLE

FOCALIZED PRESSURE ON THE WOUND BED PREVENTS INFECTION DOUBLE FOCAL COMPRESSION BANDAGING

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ABSTRACT

Bacterial contamination does not necessarily mean infection", was presented at the 17th World Meeting of the International Union of Phlebology American College of Phlebology, in 2013, Boston (USA). A common mistake made by physicians, when there is an unfavourable clinical course of the wound, is to confuse contamination with wound infection, then, they ask for a bacteriological swab and there are three possibilities: A/ Prescribe antibiotics before knowing the test result. B/ Prescribe antibiotics after knowing the test result, C/ Prescribe no antibiotics and healing the ulcer. This is a clinical experience in more of 150 patients with vascular leg ulcers, which were healed by means of using a compression technique, called "Double focal compression bandaging", without using any kind of antimicrobial agent, This technique is based on physiological concepts such as arteriogenesis and angiogenesis.

INTRODUCTION

There are objective criteria to identify wound infection and an international consensus of clinical indicators of wound infection (1), but it is important to consider that bacterial contamination does not mean infection (2). According to the result obtained in this clinical experience, with this technique:*"Local compression on the wound bed prevents infection and the use of antimicrobial agents"*. This statement will probably be the subject of controversy because differs from current guidelines about the treatment of wounds This clinical experience, in more than 150 patients with vascular leg ulcers, shows that we did use any antibiotic for healing the vascular ulcers, applying only compression therapy. A modality of compressive therapy that I named "Double focal compression bandaging"(Fig.1).This technique consists of using two bandages. The first bandage is used for the focal compression of the wound bed and another bandage covers the first, to achieve a gradual external compression from the toes to the knee, each turn of the band covers the preceding tour by 50-70%. The area of the ulcer receives the pressure of 3 layers (that of the pressure over the wound bed, and the double effect of the external gradual compression) (3). This hypothesis, based in the physiological effect of the compression in legs, could explain why no antimicrobial agent needs to be used:

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"Applying local pressure on the wound bed, there is an increase in tissue perfusion, which prevents infection and leads to the ulcer healing.This could be explained by two physiological concepts such as arteriogenesis and angiogenesis". Arteriogenesis is induced by physical forces, most importantly fluid shear stress and angiogenesis is induced by hypoxia and results in new capillaries (4). When there is an obstruction in myocardial vessels, there is a decrease in arterial pressure behind the stenosis, blood flow is redistributed via the pre-existent arterioles that now connect a high-pressure with a low-pressure region (5). This leads to an increased flow velocity and hence increased shear stress in the pre-existent collateral arteries. This causes a marked activation of the endothelium with increases in the expression of MCP-1 and of endothelial surface receptors involved in monocyte tethering, rolling and migration (6, 7, 8, 9). The upregulation of cell adhesion molecules in the proliferating collateral arterioles under conditions of elevated shear stress was confirmed (10). The subsequent increased adherence of monocytes and their transformation into macrophages are obligatory for the growth of these vessels since these cells produce numerous cytokines and growth factors involved in arteriogenesis (11. There is a relationship between negative-pressure, wound therapy and angiogenesis in ischaemic tissues (12). When we apply local pressure over the wound bed, we achieve a similar effect. It is admitted that there is a bacterial contribution in the chronicity of wounds (13)and, communities of organisms might indirectly impede wound healing by promoting a chronic inflammatory response (14).In my opinion, a vascular ulcer is the result of a pressure deficit in this area, leading to tissue perfusion deficit.

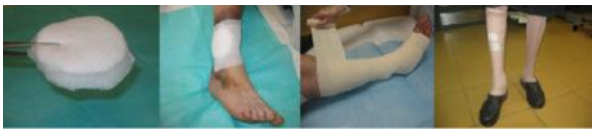

Fig. 1. Double focal compression bandaging technique.

It is necessary to improve tissue perfusion for healing the ulcer, and we achieve it, applying local pressure on the wound bed, in addition to preventing infection throughout of monocytes, which fight off bacteria, viruses, and fungi (Fig. 2).

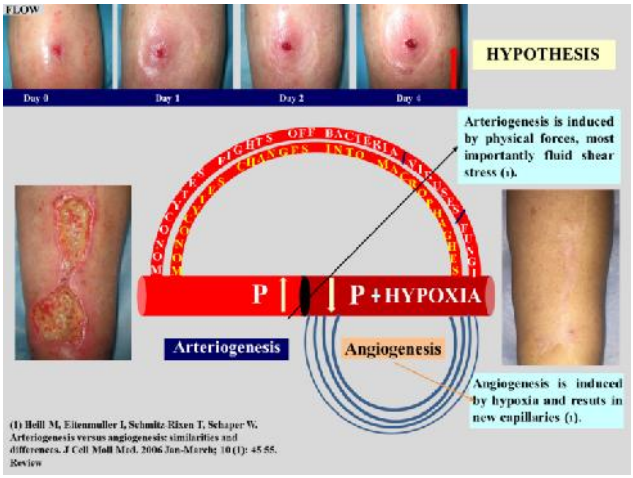

Fig 2. This picture shows how the physiological effect of compression on the wound bed, leads to the healing of the ulcer, through arteriogenesis and angiogenesis

We can find a positive bacteriological swab, but this does not mean necessarily infection. It may be only contamination. Infection is considered as a cause of the delayed wound healing, so doctors often ask for a bacteriological swab. So, there are three possibilities depending on the outcome:

A/ The doctor may prescribe an antibiotic,before knowing the test/antibiogram result.

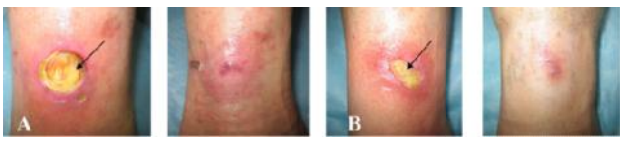

Fig. 3: Fibrin is not pus.Two clinical cases healed using "Double focal compression bandaging". Three months (A) and five months (B) after treatment

B/ The doctor may prescribe an antibiotic, after knowing the test/antibiogram result.

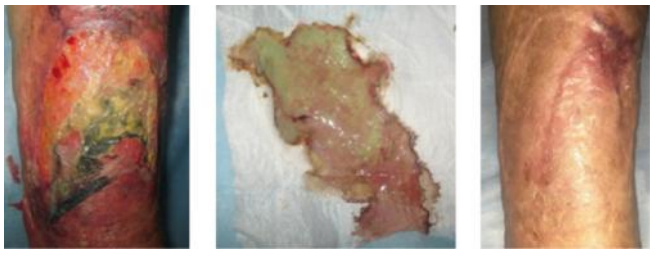

Fig. 4: A very odour ulcer healed by compression without antibiotics.

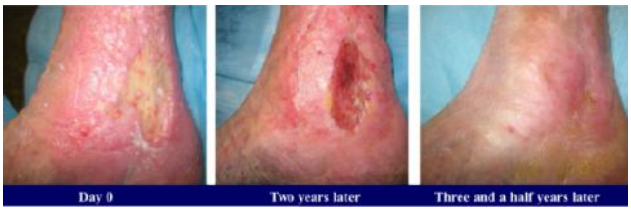

Fig. 5: A vascular ulcer healed by this technique, after 3,5 years of treatment

C/ The doctor do not prescribe an antibiotic, even with a positive test/antibiogram for bacteria and achieves to heal the ulcer. We chose this last possibility to demonstrate that, antibiotics are not necessary if we apply local pressure on the wound bed, with an external compression bandaging from toes until below the knee (Double focal compression bandaging technique). We have made a follow up of the clinical course of the ulcer from the beginning of treatment until its healing, in order to detect anyone sign or symptom of infection such as cellulitis, abscess, lymphangitis, and fever, in which case we would prescribe him/her antibiotics, but this did not happen. The classic signs and symptoms of wound infection include inflammation, new or increasing pain, local heat, swelling, advancing redness and purulent (15).There are international consensus relating to clinical indicators for a wound infection (16, 17,18). According to the results in this clinical experience, it is important to consider the following points:

A/ Fibrin should not be confused with pus (Fig.3).

Fibrin is a sign of tissue repair. A look yellow of the wound with a delay in the ulcer healing leads to think in infection, then, doctors order a bacteriological swab and prescribe antibiotics, if there is a positive result for bacteria.This is an important mistake that we should avoid. Bacterial contamination does not mean infection. There must be no antibiotic prescribed, if there are no signs and symptoms of infection.

B/ A foul-smelling wound is considered an infected wound. (Fig.4).

This is another important mistake that has to be avoided. It has to be accompanied by other signs and symptoms of infection.

C/ Painful ulceration

The ulcers can be painful. Painful ulcer, in itself, cannot be considered a symptom of infection. We only have to prescribe a soft analgesic, as paracetamol, and the pain should decrease in a few days, if this does not happen, it is necessary to re-evaluate the diagnosis.

D/ Delayed wound healing (Fig.5).

It can be considered as a risk of infection (**18**), as well as increasing the biofilm character of a wound infection corresponded with a greater delay in wound healing (**17**). Some vascular ulcers can take months or years to heal (Fig. 5).

E/ Increased volume of exudate cannot be considered a sign of infection (Fig. 6).

We can see an ulcer, as showing in the picture, with an increase of green colour exudate on the wound bed, that suggests an infection by pseudomonas aeuroginosa. We have to keep in mind that bacterial contamination is not infection. This ulcer was healed by compression therapy, without using any antibiotic.

F/ **Erythema and/or induration**(Fig. 7):These clinical signs alone are not indications of antibiotic prescription. This is often mistaken for erysipelas but, in this case, there is no fever or systemic signs of infection.

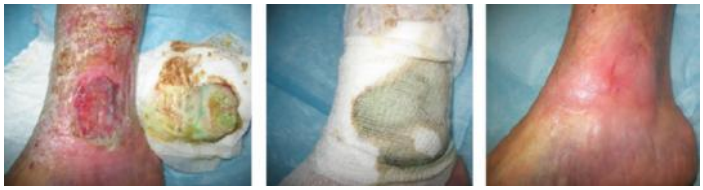

Fig. 6:Increased volume of green colour exudate and ulcer healed.

G/ **Formation of new leg ulcers in the tissue perilesional inflammation of pre-existing ulcers** (Fig.8,9):It can be considered as a sign of infection and to prescribe antibiotics. According to the results of this clinical experience, in my opinion, the formation of new leg ulcers is because compression causes a tissue perfussion deficit in areas with a poor vascular perfusion.

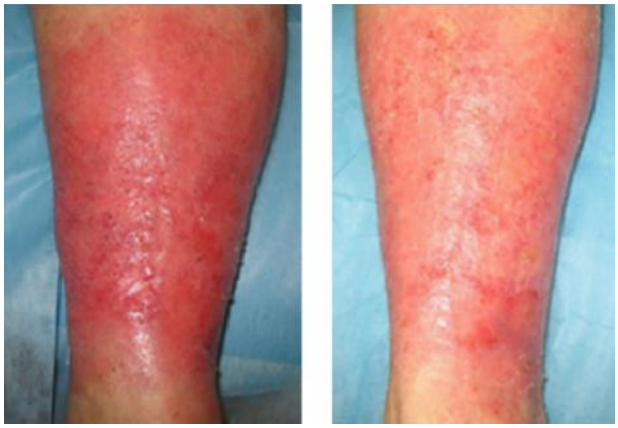

Fig.7. Five days after applying the compression, the redness of the erythema decreased

The picture (Fig.8) shows how one month later of applying compression therapy, a second ulcer appears close to the first one. This cannot be considered as a sign of infection. The compression detects badly perfused areas, that it can become to ulcers, after minor trauma on the leg. The picture (Fig. 9) shows two vascular ulcers on the right legandeighteen months later, new ulcers appear on the left leg.They were healed using only “double focal compression bandaging”. It is important to note that, an ulcer on one leg, some time later, may appear on the other leg.

H/ Sudden onset of black necrotic spots (Fig.10).

This fact, in itself, cannot be considered as sign of infection. It is the result of inadequate tissue perfusion,leading to hypoxia and later necrosis. We only use local pressure on the wound bed, and four months later, the black necrotic spots disappeared. We think it is due to the improvement of blood flow bythe effect of arteriogenesis/ angiogenesis.

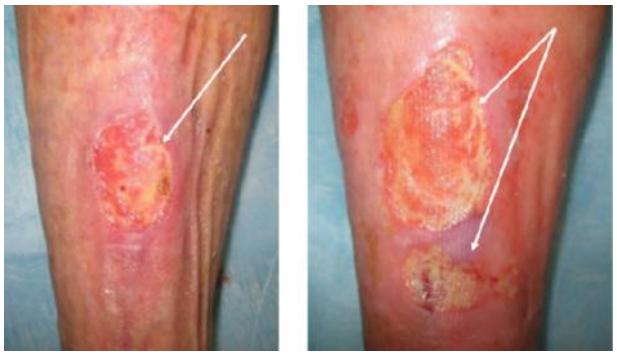

Fig. 8: An ulcer on the right leg. A month later a second ulcer appears

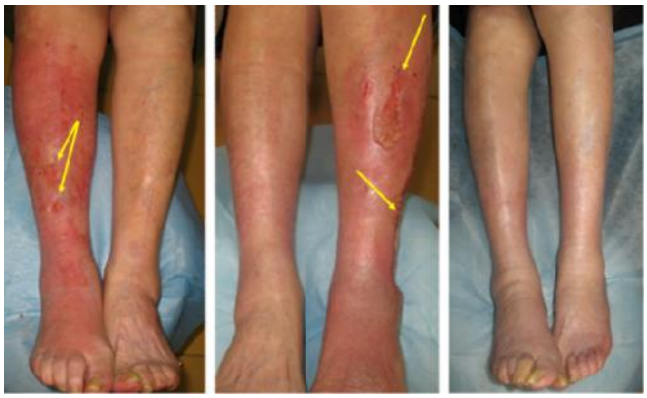

Fig. 9. Similar ulcers in both legs.

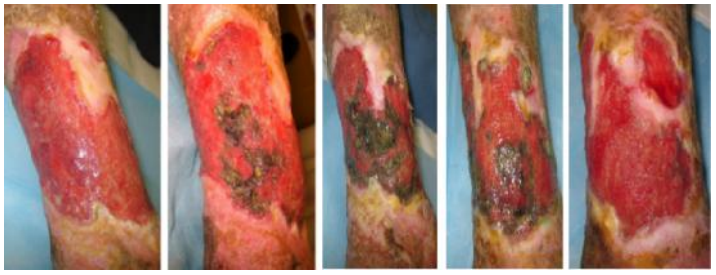

Fig. 10. The necrotic areas disappear after compression therapy.

I/ New necrosis or increase in size of necrotic tissue in the wound bed (Fig. 11).

It should not be considered as a sign of infection. Tissue perfusion deficit leads to hypoxia and subsequent necrosis. If there is an increase in blood flow to this area,the necrosis will be solved without using any kind of debridement,by arteriogenesis/angiogenesis. We can do it by increasing the pressure on the bed wound, with a padding placed on the bed wound with a gradual external bandage (Fig.11).

Fig. 11. One month later, necrosis disappears after compression therapy

J/ Change in colour into wound bed is associated to different bacteria (Fig.12 A/B/C).

The colour of the wound bed is associated with different bacteria (**19**).

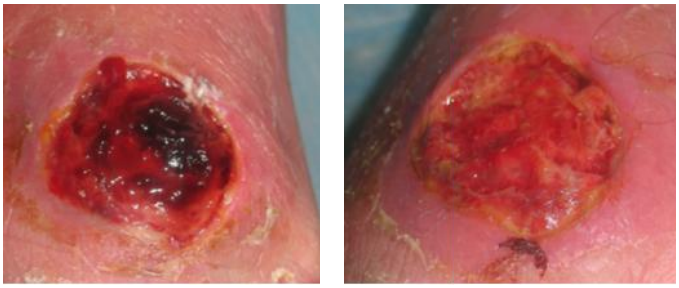

Fig. 12 : A/ The black colour is associated with anaerobic bacteria

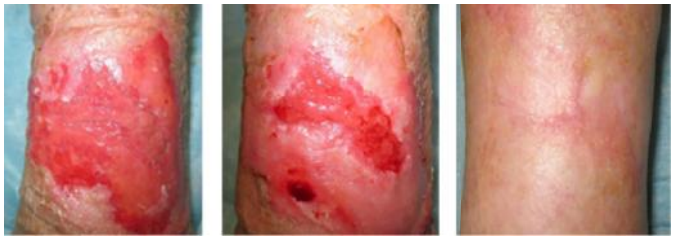

Fig. 12: B/ The brilliant red is associated with streptococcus

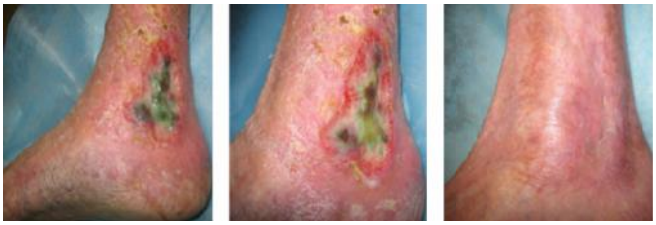

Fig.12: C/ The green colour is associated with pseudomonas

We cannot identify an infection according to the colour of the wound bed, if there are not others signs or symptoms, it is just contamination: We have not used any antimicrobial agent, to heal vascular ulcers in more than 150 patients. We only used compression therapy: "Double focal compression bandaging".Based on the results of this clinical experience, it is concluded that:

"Focalized pressure on the wound bed prevents infection"

The clinical course of the vascular ulcers and the results support this statement, which will certainly be controversial. These are some relevant cases, in patients with vascular leg ulcers, healed by this technique. No antibiotic was prescribed.
We have followed a simple methodology: 1st/In the first week we did a daily follow-up of the patient, to detect any signs or symptoms of infection such as, cellulitis, lymphangitis or fever, in which case, we should prescribe antibiotics, but this did not happen. After, we made a weekly follow up until healing ulcer.
2nd/ We ordered a bacteriological swab and if there were no signs or symptoms of infection, we did not prescribe antibiotics, despite having tested positive for bacteria. Many of these patients had been treated by their doctors, with different therapies and antibiotics, without positive result. We show the most significant cases treated by this technique.

We did detect contamination in the wound bed, but no infection. In some cases,we achieve to eradicate bacteria from the wound bed but not in others.

Case report 1(Fig. 11): A 70-years-old male diagnosed with arterial hypertension and type 2 diabetes mellitus. He was sent to our office because of suffering from a right leg ulcer.He had been treated with different therapies and antibiotics, without positive result. We measure his ankle-brachial-index(ABI:0.90) to apply compression therapy, and we startedtreating him(02/04/2014): "Local pressure on the wound bed with a gradual external compression, from the toes to the knee". We ordered four bacteriological swabs throughout the clinical course of the ulcer until healing. We found bacteria in three of them.

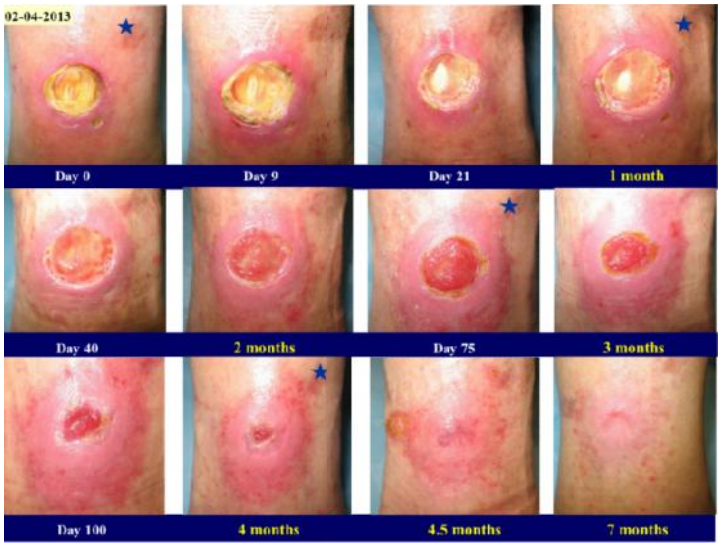

Fig.11. Clinical course of the ulcer healing. We can see how fibrin turns into granulation tissue, and the ulcer heals four months later. Blue stars indicate we ordered bacteriological swabs.

Proteus Mirabilis and Staphylococcus aureus, however, in the last one, bacteria disappeared. The ulcer healed in four an a half months, applying only this technique, and eradicating bacteria in wound bed. No antibiotic was prescribed. A yellowish appearance of the wound along with a late healing, led to being diagnosed as an infection and treated with antibiotic. Fibrin should not be confused with pus.When we order a bacteriological swab, we will probably find bacteria and prescribe antibiotics.

This is a mistake that is necessary not to commit, because it can indicate bacterial contamination. Bacterial contamination does not mean infection.

Case report 2 (Fig. 12): A 54-years-old femalediagnosed with type 2 diabetes mellitus, diabetic retinopathy treated with laser photocoagulation, arterial hypertension, obesity grade II and dyslipidaemia. Three years ago, she underwent surgery for removing varicose veins in her left leg. On that date (07/14/2014), she was referred to hospital admissionby suffering from severe pain in her left leg. She was diagnosed with a perforating plantar ulcer with an ulcer on her left leg, and treated with antibiotics for having a positive bacteriological swab on the wound bed. The patient was released from the hospital, two weeks later.

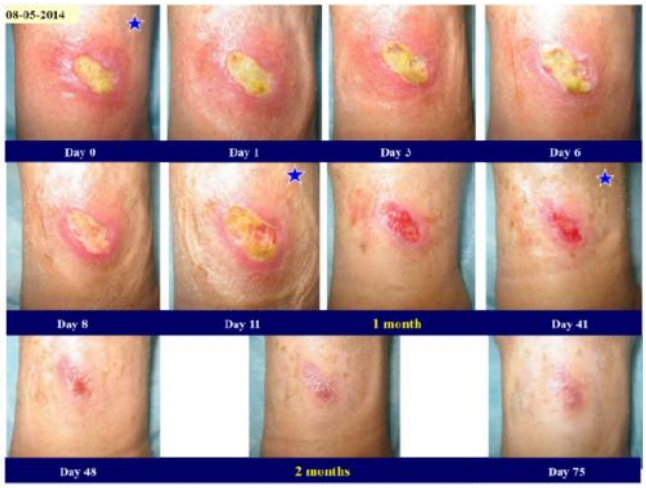

Fig. 12. Clinical course of ulcer healing.Fibrin turns into granulation tissue.Blue stars indicate we ordered bacteriological swabs

She came to our office because the ulcer did not heal, even though different treatments had been prescribed by her doctor. We ruled out peripheral arterial disease by measuring the ankle-brachial index (ABI: 0.95), and we treated her with compression therapy (double focal compression bandaging). We ordered three bacteriological swabs and found *Pseudomonas aeruginosa* in two of them. The third was negative for bacteria. The image (Fig. 12) shows how fibrin turns into granulation tissue. It is very important to remember that *fibrin should not be confused with pus.*

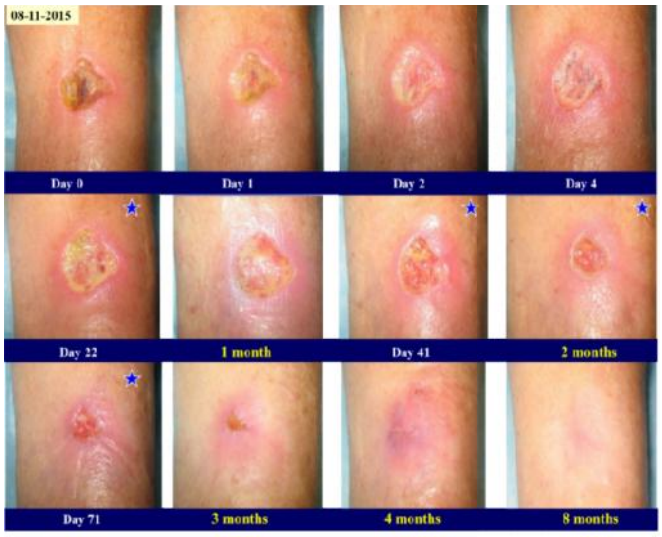

Fig. 13. Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs

Case report 3 (Fig. 13): A 72-years-old female diagnosed with arterial hypertension, type2 diabetes mellitus, asthma, ataxia, and coxarthrosis. Hospital admission (05/19/2015) for acute right leg ischaemia. She was diagnosed with severe peripheral arteriopathy, treated with intravenous antibiotics and operated (endovascular treatment) On this date (05/30/2015), the patient was discharged from the hospital. A month later, the patient came back to the hospital because of suffering severe pain in her left leg. She was admitted to the hospital for 2 weeks, and released (07/14/2015) with an ulcer on her left leg.

She was treated with antibiotics, by her doctor, without positive result. The patient is referred to our office to assess whether she could benefit from the use of compression therapy. The patient had surgery for suffering from severe peripheral arteriopathy. The ankle brachial index was 0.55, when she was discharged from hospital.We monitor the clinical course daily for 5 months, to detect any complications induced by compression therapy. After that, we followed up weekly. We achieve to heal the ulcer without using any antibiotic, and to increase the ankle brachial index, which is normal five years later.The patient continues to do compression bandages on both legs, removing them at bedtime. In expert hands, and according to the signs and symptoms that the patient suffers, we can improve the arterial flow in patients diagnosed with peripheral arteriolopathy, using compression therapy with daily clinical follow up. We ordered four bacteriological swabs and we found *Pseudomonas aeruginosa*, in the first ones and negative for bacteria in the last ones. The ulcer healed using only this technique, four months later. In these three cases we have achieved to eradicate bacteria in the wound bed without prescribing antibiotics, but this does not always happen, as we will see in the following cases, but the result is the same: Healing the ulcer.

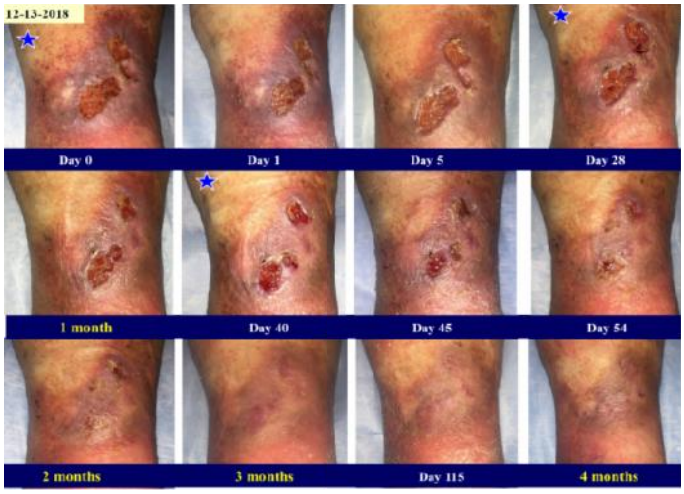

Fig. 14 . Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs.

Case report 4 (Fig. 14): A 64-years-old femalediagnosed with arterial hypertension, type 2 diabetes mellitus insulin-dependent, dyslipidaemia and obesity (metabolic syndrome). She underwent surgery for varicose veins, ten years ago. The patient has an ulcer in the left leg, so she was treated with antibiotics for having bacteriological swabs positive to bacteria. On this date (12/13/2018), she came to our office due to the bad evolution of the ulcer.Wemade sure that herankle-brachial index was normal (ABI: 0.92), and started treating the ulcer using only compression therapy. We ordered three bacteriological swabs and found *methicillin-resistant Staphylococcus aureus* in the first, and *Staphylococcus aureus* in the last two. After two months of treatment, we achieved to heal the ulcer but not eradicate bacteria.

Case report 5 (Fig. 15): A 53-years-old female diagnosed with antiphospholipid syndrome, recurrent episodes of deep vein thrombosis and anticoagulant treatment. She had an ulcer in her right leg with a long time of poor evolution (more than one year).

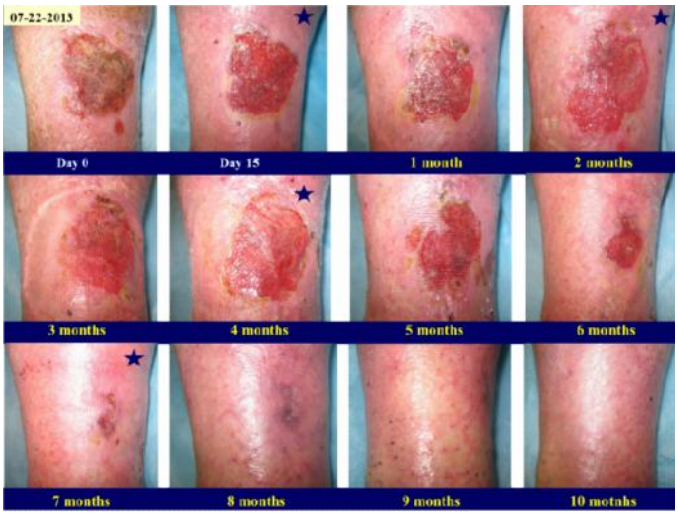

Fig. 15. Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs

She came to our office because she had been treated with different therapies and antibiotics, without positive result. We measured the ankle-brachial index (ABI: 0.90), and started treating her, on this date (07/23/2013). We ordered four bacteriological swabs, finding the following positive results for bacteria: *Staphylococcus aureus and Pseudomonas aeruginosa*, but we failed to eradicate bacteria in the wound bed. Using this technique, the ulcer healed 8 months later.

Case report 6 (Fig. 16): A 82-years-old female with arterial hypertension, depression, atrial fibrillation and anticoagulant treatment. She had an ulcer on her left leg that had been treated with antibiotics and other therapy, without positive result. She came to our office (10/22/ 2014), due to months of bad evolution.We verified by measuring the ankle-brachial index (ABI: 0.96), that the patient could apply compression, sothe treatment of the ulcer was started, using only compression therapy. We ordered three bacteriological swabs with a positive result to *Staphylococcus aureus* and *Group A Streptococcus pyogenes.* After three months, we achieved to heal the ulcer but not eradicate bacteria.

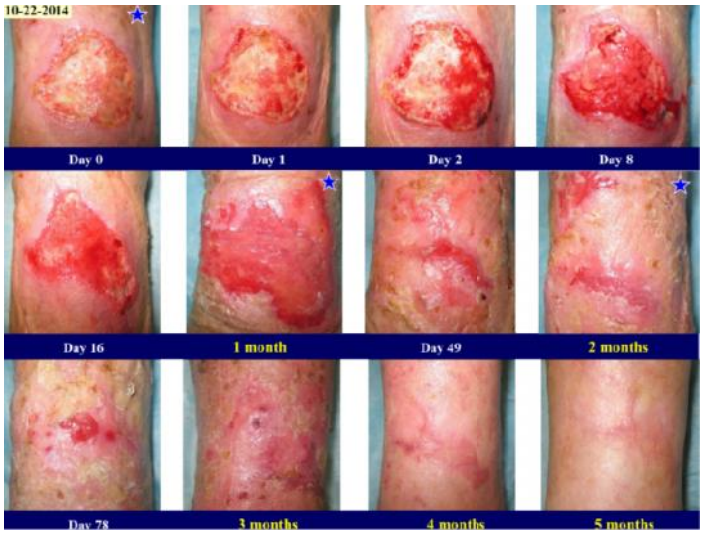

Fig. 16. Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs.

Case report 7 (Fig.17): A 80-years-old female with arterial hypertension, dyslipidaemia and venous insufficiency chronic. She has had an ulcer after a trauma in her right leg. We measured her ankle-brachial-index (ABI: 1.05) and started treating her on this date (04/07/2016), achieving to heal her, one month later. We orderedthree bacteriological swabs with positive result for the following bacteria:

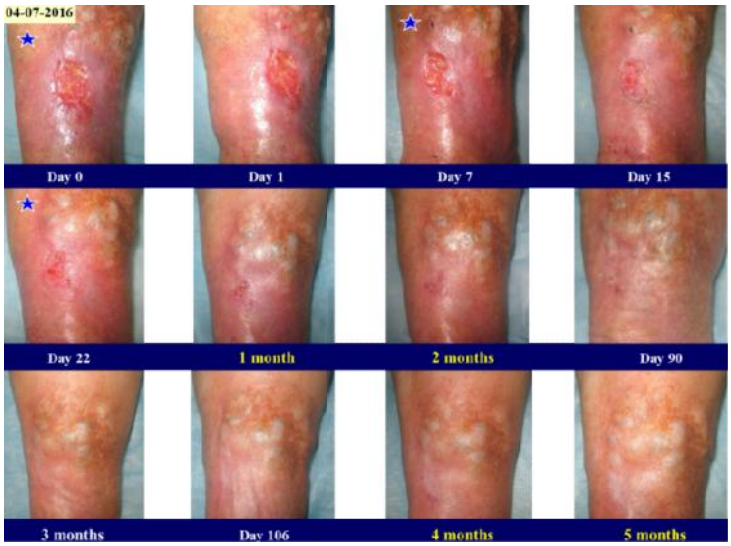

Fig. 17. Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs

A/ The first one was positive to *Morganella morganii* and *Staphylococcus aureus*.

B/The second one was positive to *Enterobacteria ceae*and *Enterococcus faecalis.*

C/ The third one was positive to *Proteus vulgaris* and *Staphylococcus aureus*.

We did achieve to heal the ulcer, but not eradicate the bacteria on the wound bed.

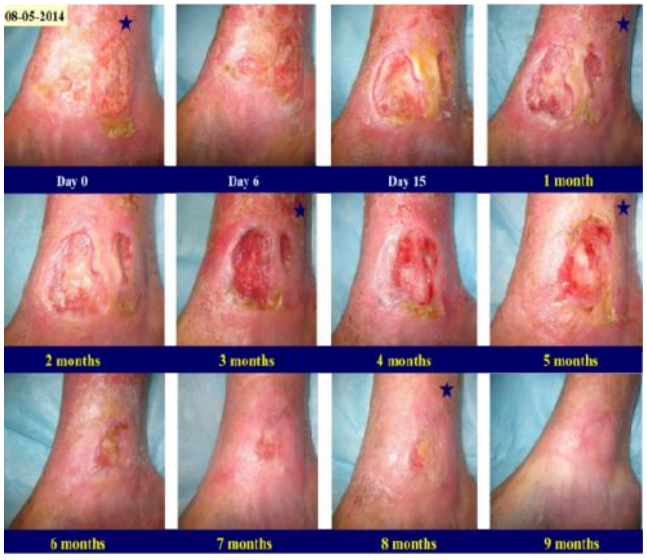

Fig. 18.

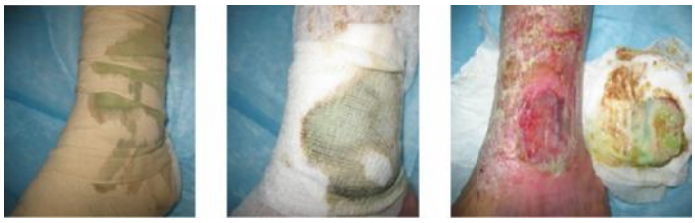

Fig. 19. Clinical course of the ulcer healing.Blue stars indicate we ordered bacteriological swabs.

Case report 8 (Fig.18, 19): A 68-years-old female with arterial hypertension, dyslipidaemia, and depression. She suffered trauma in her left leg that led to an ulcer with bad evolution. We measured her ankle-brachial-index (ABI: 0.89) before applying compression therapy, and started treating heron this date (05/08/2014), achieving to heal her nine months later. We ordered six bacteriological swabs, with positive result to these bacteria: *Staphylococcus aureus, Pseudomonas aeruginosa, Group AStreptococcus pyogenes.* We did achieve to heal the ulcer, but not eradicate bacteria in the wound bed. It is possible to see dirty bandages by exudates green colour, suggesting contamination by Pseudomonas aeruginosa. This does not mean infection; it is contamination if there are not any signs or symptoms of infection. In this case, the ulcer did heal by only compression "Double focal compression bandaging".

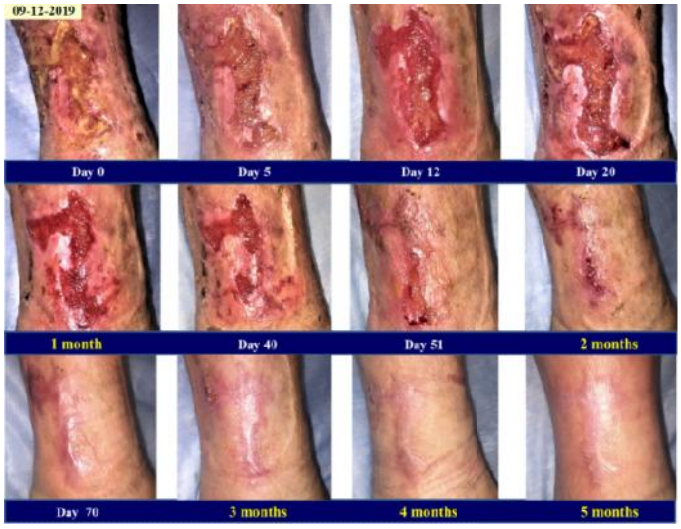

Fig. 20. Clinical course of the ulcer healing.

Case report 9 (Fig. 20): A 61-years-old female with arterial hypertension, obesity, type 2 diabetes mellitus. She underwent two operations for suffering from varices in legs. The first one was fifteen years ago. She was operated for recurrence of varicose veins on this date (06-14-2018). Months later, she suffered vascular leg ulcers that they were treated with antibiotics, according to antibiogram test result. Different bacteria were found in bacteriological swabs, being the commonest *Staphylococcus aureus.* The vascular surgeon's report indicated that no other surgery was possible. She came to our office (09-12-2019) to be evaluated to check the possibility of applying compression therapy. The ankle-brachial-index was measured (ABI: 0.85) and the patientwas treated with double focal compression bandaging. Three months later, we achieved to heal the ulcer, without using any antibiotic, but not eradicate bacteria in the wound bed.

According to the results in this clinical experience, when we apply local pressure on the wound bed, with a gradual external compression bandaging from toes to below the knee, we generate pressure gradients in the wound bed, that stimulate the arteriogenesis/angiogenesis in that area.

I think the reason there is no infection, could bein the arteriogenesisstimulated around the wound bed, with blood rich in monocytes (monocytes fightoff bacteria, viruses and fungi).

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