



## RESEARCH ARTICLE

### WAS THE USE OF ANTIBIOTIC NECESSARY? A CLINICAL CASE REPORT DOUBLE FOCAL COMPRESSION BANDAGING

\*Carlos Sánchez

Carlos Sánchez Fernández de la Vega. Galician Health Service, Primary Care

#### ARTICLE INFO

##### Article History:

Received 26<sup>th</sup> October, 2021

Received in revised form

15<sup>th</sup> November, 2021

Accepted 19<sup>th</sup> December, 2021

Published online 30<sup>th</sup> January, 2022

##### Keywords:

Pressure, Wounds,  
Arteriogenesis,  
Antibiotics.

#### ABSTRACT

According to the outcomes of a clinical experience in more of two hundred patients, pressure focused on the wound bed prevents infection. We did a daily follow-up of the clinical course of the wounds, during the first few weeks, to detect any signs or symptoms of infection, in which case we would prescribe antibiotics, but this did not happen. We asked for cell cultures of the wound's exudate, finding bacterial contamination, but no infection. The explanation lies in the antibacterial effect of monocytes. due to increased blood flow from arteriogenesis, stimulated by focused pressure on the wound bed. We reported a clinical case of an 80-year-old woman with venous hypertension who suffered trauma to her right leg. The first cures were made in the emergency of primary care and, as the wound did not heal, he asked for consultation, eleven days later. We applied, only eight days, double focal compression bandaging technique, because the patient considered that the clinical course was not adequate so, she went to the emergency department of the hospital. She was admitted to the hospital for 19 days, treated with intravenous antibiotics for 8 days, and discharged from the hospital to be treated for home hospitalization. The ulcer healed 4 months later. This article highlights the anti-infective effect of the focused pressure on the wound bed, comparing this case with three similar ones, in which wound/ulcer healing was achieved using only the focused pressure on the wound bed.

Copyright © 2022, Carlos Sanchez. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original work is properly cited.

## INTRODUCTION

The usefulness of antibiotics to fight infection-cannot be doubted, however, antibiotic resistance is a global health problem. We need to use them when there are unmistakable signs and symptoms of infection. Antimicrobial resistance in bacterial pathogens is a challenge that is associated with high morbidity and mortality. Multidrug resistance patterns in Gram-positive and -negative bacteria are difficult to treat and may even be untreatable with conventional antibiotics (1). Microbial colonisation in chronic wounds is inevitable, and in most situations endogenous bacteria predominate, many of which are potentially pathogenic in the wound environment. The risk of wound infection increases as local conditions favour bacterial growth rather than host defence (2). The double-focal compression bandage technique is a type of compressive therapy, which consists of focusing pressure on the wound bed, to generate pressure that increases blood flow around the wound, stimulating arteriogenesis in the affected area, leading to revascularization, evidenced by an increase in granulation tissue, and healing of the ulcer. The pressure is obtained by applying a gauze pad on the wound bed, with an additional bandage for external compression, from the fingers to below the knee (Fig. 1).

\*Corresponding Author: Carlos Sanchez

Carlos Sánchez Fernández de la Vega. Galician Health Service, Primary Care.

According to the results of my clinical experience in the treatment of vascular ulcers, the pressure focused on the wound bed prevented infection (3). Over the years, in more than 200 patients treated with this technique, we have not detected any sign or symptom of infection, so it was not necessary to use antibiotics.

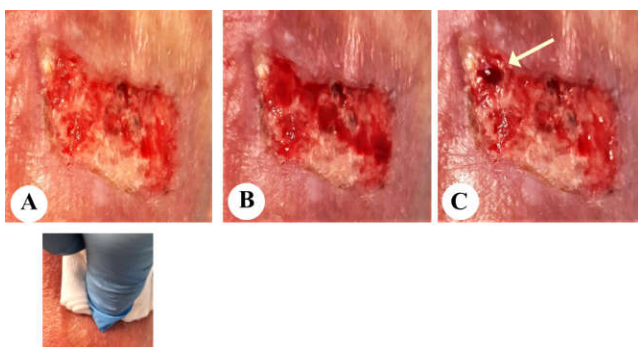
#### Why is there no infection when applying pressure to the wound bed?

This could be due to the antibacterial action of monocytes by increasing blood flow around the wound bed. Recruitment of monocytes is essential for effective control and clearance of viral, bacterial, fungal, and protozoal infections (4). Circulating blood monocytes supply peripheral tissues with macrophage and dendritic cell (DC) precursors and, in the setting of infection, also contribute directly to immune defence against microbial pathogens (5). This clinical fact can be explained by a similar effect on the obstruction of a myocardial artery. An obstruction in the myocardial vessels leads to a decrease in blood pressure, behind the stenosis. Blood flow is redistributed through the pre-existing arterioles, that now connect a high-pressure region (pre-obstruction) to a low-pressure region (post-obstruction) (6). Arteriogenesis is the rapid proliferation of the pre-existing collateral arteries and angiogenesis is a process by which new capillary blood vessels sprout from a pre-existing blood vessel (7).



**Fig. 1. Double focal compression bandaging technique**

Vascular ulcers, excluding neoplastic ones, are produced by a deficit of tissue perfusion, to solve it is a condition for its healing. This can be achieved by arteriogenesis, which leads to the formation of collateral vessels to compensate for that perfusion deficit. In the image (Fig. 2-A), we can see how a drop of blood flows, after pressing with a gauze on the wound bed, indicating an increase in blood flow (Fig. 2-B-C). Antibiotic usage has significantly reduced morbidity and mortality associated with bacterial infections. However, inappropriate use of antibiotics has led to emergence of antibiotic resistance at an alarming rate. Antibiotic resistance is regarded as a major health care challenge of this century. We insist on the importance of daily surveillance of the clinical course of the ulcer, in the first week, to detect any sign/symptom of infection, in which case I would prescribe him/her the antibiotic, but in my experience, this never happened. Early detection of wound complications is crucial to improving patients' quality of life.



**Fig. 2. Increased blood flow after applying focused pressure on the wound bed**

We reported three clinical cases with vascular ulcers, which healed by applying only the double focal compression bandage technique. They were, previously, treated with antibiotics. In the other case, the patient was initially treated with the same technique, but only for 7 days, since the patient decided to go to the emergency department of the hospital, where she was admitted to the angiology service, and treated with antibiotic and negative pressure therapy. In contrast to the above, the other clinical cases healed using just compression.

**Case report 1** (Fig. 3): An 80-year-old woman suffered trauma in the pretibial area of her left leg (16-07-2021). Ten days later, she comes to our office because the wound has a bad evolution. We can see what the wound looks like (Fig. 3-A). We started to treat her using the double focal compression technique. Photos 3 show the clinical course since she was treated with compressive therapy, only for one week (08-02-2021), because she decided to go to the hospital emergency room. If we take a closer look at the images, we can see the formation of a necrotic scab, in which drops of blood are observed when the bandage is removed, indicating an increase in blood flow in the ulcer area (Fig. 3-C). The patient had no fever, and the peripheral pulses were palpable.

Blood test from the emergency room showed a normal blood count with an ESR of 10.8. The patient was admitted to the vascular surgery room, where she was treated with intravenous antibiotics (Clindamycin), despite having a normal blood count. The following day, a culture of the wound exudate was requested with the result "Saprophytic skin flora"(Fig. 14). Despite this result, she was treated with intravenous clindamycin for eight days, followed by negative pressure therapy for two weeks. The patient was discharged from the hospital after 19 days with home hospital care, and negative pressure therapy for two more weeks. The ulcer healed after 4 months.



**Fig. 3. Clinical evolution until full healing, fourth month later. B/ The drop of blood that flows when the bandage is removed, indicates an increase in blood flow in the area**

What is relevant in this case is that the hemogram and wound culture showed no evidence of infection at the time of admission, however, the patient was treated with intravenous antibiotics, probably to prevent infection. I am convinced that, had compression therapy been maintained, the ulcer would heal within a few months, without the high cost of hospital admission and home hospitalization. To support this claim, we show three similar cases healed by "double focal compression bandaging", previously treated with antibiotics. The antibiotic was removed as soon as we started treatment.

**Case report 2 (Fig.4):** A 90-year-old woman with vascular dementia and vascular ulcer in her left leg, from years of evolution, in which she has been treated with antibiotics and different therapies, without positive result. She came at the doctor's office on this date 04/25/2021. We achieve a full healing five months later, by applying only "double focal compression bandaging" (Fig. 4). The inflammatory phase of the wound healing cascade gets activated during the coagulation phase and can roughly be divided into an early phase with neutrophil recruitment and a late phase with the appearance and transformation of monocytes. In the phase of proliferation, the focus of the healing process lies in the recovering of the wound surface, the formation of granulation tissue and the restoration of the vascular network. Therefore, next to the immigration of local fibroblasts along the fibrin network and the beginning of reepithelialisation from the wound edges, neovascularization and angiogenesis get activated by capillary sprouting (Fig. 5). The formation of granulation tissue stops through apoptosis of the cells, characterizing a mature wound as avascular as well as acellular.

During the maturation of the wound the components of the extracellular matrix undergo certain changes. The physiological endpoint of mammalian wound repair displays the formation of a scar, which is directly linked to the extent of the inflammatory process throughout wound healing (9) (Fig. 3).



Fig. 4. Clinical course of leg ulcer three months later applying compression therapy



Fig. 5. After compression therapy, healthy granulation tissue forms



Fig. 6. The ulcer healed after five months of compression



Fig. 7. After applying compression, the oedema decreases

**Case report 3:** A 38-year-old man who suffered a chainsaw cut on the big toe of his left foot. The patient had a cross wound from the anterior face to the posterior face of the distal phalange, with loss of soft tissue. He was sutured, (non-absorbable monofilament, Ethilon3/0), being discharged from the hospital with the following treatment Oral antibiotic for 7 days (amoxicillin/clavulanic acid); nonsteroidal anti-inflammatory drug (ibuprofen); proton pump inhibitor (omeprazole). In the following images (Fig. 8-9), we can see what the wound looks like, when the patient is discharged from the hospital and the subsequent clinical course to full healing. We stop antibiotic treatment, and only apply focused pressure on the wound bed, with a daily follow-up of the clinical course of the wound, in the first week, to detect any signs or symptoms of infection, but this did not happen, suggesting an anti-infective action of monocytes, due to increased blood flow in the area, by focused compression on the wound. The ulcer healed two and a half months later. The photographic sequence shows the clinical course, from the time it is treated by us to full healing (Fig 8.1-8.2).



Fig. 8.1. Appearance of the wound 5 days after removing the stitches and applying compression on the toe

We observed an increase in necrotic tissue, which disappears in the following days. The explanation can be in the action of monocytes transformed into macrophages. Months later, we get an optional result. We did not use any debriding or antimicrobial agent, only compression.



Fig. 8.2: 71 days after treatment



Fig. 9. Six months after surgery and focused compression on the surgical wound

**Case report 4 (Fig. 11):** A 58-year-old man suffered a traumatism to his right leg, with an abrasive wound on the knee. He attended the emergency department, where he was treated with antibiotics and anti-inflammatory drugs, with subsequent follow-up by his doctor and nurse in primary care. We see what the wound looks like, 12 hours after going to the emergency department. We can see a yellowish discharge with swelling of the knee, and how they disappear 24 hours later (Fig. 11-A-B).



Fig.11. A/Yellowish drainage and oedema, 12 hours after emergency treatment. B/ Granular tissue with oedema that disappears 24 hours after applying focused compression on the knee, and drug elimination

Antibiotics and anti-inflammatory drugs were eliminated by applying compression centred on the wound, fixing the gauze padding to the leg with adhesive tape in the shape of a baptismal cross (Fig. 12). We tell the patient to watch for the onset of fever, but this did not happen.



Fig. 12. Compression bandaging with padded gauze on the wound, fastened to the knee with adhesive tape placed in the shape of a baptismal cross.

The appearance of the wound has improved markedly, with the formation of granulation tissue leading to the healing of the ulcer, one months later (Fig. 13). A month and a half later, there is a full healing. Once again, focused pressure on the wound bed demonstrates that prevents infection.



Fig. 13. Wound healed, after applying focused pressure on the wound

**Are we questioning the prescription of antibiotics?:** Of course not. They are necessary when there are signs and/or symptoms of infection, but infection is not possible, with a normal blood count and a negative wound cell culture. In the first case described, as the poor clinical course was attributed to infection, it was decided to admit the patient to the hospital's angiology department. The patient was treated with intravenous antibiotic (Clindamycin), for 8 days, despite having a normal blood count and a wound exudate culture with saprophytic flora of the skin (Fig. 14). Following this, she was treated with negative pressure therapy until the ulcer healed. The wound was not infected, and there was no poor clinical progress. The following three clinical cases demonstrate that compression of the wound bed prevents infection, as they were healed without the use of antimicrobial agents. Certainly, a controversial statement but supported by the results. It is only necessary to follow, daily during the first week, the clinical course of the wound, to detect any/symptoms of infection, in which case it would be treated with antibiotics.

Asociación	Desc. Proba	Resultado	Unidades	Valores Ref.
HEMATITIA	Paragema			
Hemoglobina		4.39	g/dL	(11.1-15.1)
Hemoglobina		16.8	g/dL	(12.0-16.0)
Hematocrito		41.3	%	(38.8-48.8)
Velocidad sedimentación media		36.0	mm/h	(0.0-30.0)
Hemoglobina Corp. Media		31.8	pg	(1-1)
Val. ref. 27-31 µg				
Concentración hemoglobina Corp. media		35.4	g/dL	(1-1)
Val. ref. 32-36 g/dL				
Índice distribución hematocrito		122	%	(1-1)
Val. ref. 11.0-14.1				
Leucocitos		5.72	mil/m <sup>3</sup>	(0.0-10.0)
Neutrófilos (%)		75.5	%	(1-1)
Val. ref. 42-75 %				
Neutrófilos (F)		4.3	mil/m <sup>3</sup>	(0.5-6.0)
Val. ref. 20-40 %				
Linfocitos (F)		0.6	mil/m <sup>3</sup>	(0.0-5.0)
Val. ref. 1.0-4.0 %				
Neutrófilos (F)		1.7	mil/m <sup>3</sup>	(0.1-1.0)
Val. ref. 0.0-0.9 %				
Basófilos (F)		0.1	mil/m <sup>3</sup>	(0.0-0.4)
Val. ref. 0.0-0.9 %				
Basófilos (F)		0.7	mil/m <sup>3</sup>	(1-1)
Val. ref. 0.0-0.9 %				
Plaquetas		205.0	mil/m <sup>3</sup>	(100.0-400.0)
Plaquetas (F)		16.7	F	(1-1)
Val. ref. 7.4-10.4				
CD45/LA/CD45-Ro/normal exp/leucocitos				
Siempre en Presencia		12.0	Seg	(1-1)
Actividad TP		82.0	%	(70.0-120.0)
INR		1.05	(1-1)	(1-1)
Clotado TP		11.0	Seg	(1-1)
PTT		30.0	Seg	(1-1)
Ratio aPTT		1.0	(0.8-1.8)	(1-1)
Clotado aPTT		20.0	Seg	(1-1)
Fibrinógeno		323.0	mg/dL	(170.0-500.0)

**A/ Normal blood count**

**B**

Resultado: Cultivo ordinario

Observaciones: Crecimiento de flora saprofita de piel

**B/ Wound exudate Saprophytic flora**

Fig. 14. A/ Blood count (08-04-2021); B/ Wound exudate: Saprophytic flora (08-06-2021)

## REFERENCES

1. Marianne Frieri, Krishan Kumar, Anthony Boutin. Antibiotic resistance. *J Infect Public Health*. Jul-Aug 2017;10(4):369-378. doi: 10.1016/j.jiph.2016.08.007. Epub 2016 Sep 6.
2. Philip G Bowler. Wound pathophysiology, infection, and therapeutic options. *Ann Med*. 2002;34(6):419-27. doi: 10.1080/078538902321012360.
3. Sánchez, C. Focalized pressure on the wound bed prevents infection. Double focal compression bandaging. *International Journal of Information Research and Review*. Vol. 07, Issue,10, pp.7113-7120, October2020.
4. Chao Shi, Eric G Pamer. Monocyte recruitment during infection and inflammation. *Nat Rev Immunol*. 2011 Oct 10;11(11):762-74. doi: 10.1038/nri3070.
5. Natalya V Serbina, Ting Jia, Tobias M Hohl, Eric G Pamer. Monocyte-mediated defence against microbial pathogens. *Annu Rev Immunol*.2008; 26:421-52. doi: 10.1146/annurev.immunol.26.021607.090326.
6. Schaper W. Pasyk S. Influence of collateral flow on the ischemic tolerance of the heart following acute and subacute coronary occlusion *Circulation* 1976 53 I57 I62.
7. Buchmann I. and W.Schaper. Arteriogenesis Versus Angiogenesis: Two mechanisms of vessel growth. *News Physiol. Sci*. Volume 14, June 1999.
8. Dipannita Ghos, Balaji Veeraraghavan, Ravikrishnan Elongovan, Perumal Vivekanandan. Antibiotic Resistance and Epigenetics: More to it than Meets the Eye. *Antimicrob Agents Chemoter*, 2020 Jan 27; 64 (2):e02225-19. Doi: 10.1128/AAC.
9. Reinke, J M H Sorg. Wound pathophysiology, infection and therapeutic options. *Ann Med*. 2002;34(6):419-27. doi: 10.1080/078538902321012360.

\*\*\*\*\*