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Charcot foot (neuropathic arthropathy) in diabetes as a "special needs foot". Case report of an efficient negative pressure wound therapy use.

Bartosz Cybułka

CASE REPORT

***Abstract*—Diabetes is the most common endocrine disorder of carbohydrate metabolism. If left untreated, or improperly treated, diabetes leads to multiple organ complications. One of the serious consequences of the disease is damage to the peripheral and autonomic nerves known as diabetic neuropathy. The most advanced form of neuropathy, leading to damage to the structures of the forefoot, midfoot and hindfoot, is the so-called Charcot foot, or neuropathic osteoarthropathy. Irreversible damage to the structures of the foot affects between 0,1% and 7.5% of patients with diabetes.**

The optimal care for that form of foot damage is still a subject to debate. Available methods of caring for Charcot foot include invasive orthopedic treatment and conservative treatment. The use of negative pressure wound therapy may be an effective, as well as transitional, way of managing Charcot foot.

***Keywords*—Charcot neuro - osteoarthropathy, Charcot foot, diabetic foot, diabetic neuropathy, negative pressure wound therapy (NPWT)**

I. Introduction

DIABETES mellitus is the most common endocrine disorder. Impaired metabolism of carbohydrates leads to numerous organ complications. Epidemiologically, in 2011 there were 285 million patients with diagnosed diabetes. This number constituted 6.6% of the population aged 20–79. It is estimated that about one-third of cases still remain undiagnosed. Among them, 2.5% will develop one of the most dangerous complications of diabetic foot, which is the Charcot arthropathy.[1]

Osteoarticular lesions that result from disturbances of innervation, referred to as neuropathic arthropathy, were described in 1868 by Jean-Martin Charcot.[2]

Complicated form of diabetes is the most common cause of neuropathic arthropathy in developed countries. Any peripheral neuropathy may lead to articular lesions. Other causes that may, consequently, lead to lesions in the joint structure are: syringomyelia, poliomyelitis, injuries of the spinal cord, leprosy, alcohol abuse, multiple sclerosis, heavy metal poisoning and rheumatoid arthritis.[3, 4]

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The clinical classification of Charcot foot distinguishes the acute form of the disease, characterized by edema, redness, increased blood circulation, and increased foot temperature. Charcot arthropathy should be suspected in patients with these symptoms, without characteristic ulceration. The skin temperature of a diseased foot may be increased even by 2-6^{o}C. Pain intensity depends on the stage of diabetic neuropathy.[5]

Charcot arthropathy is often diagnosed during the inactive, chronic stage when inflammatory symptoms are not present. In this phase, bones, joints and ligamentous apparatus of the foot are being continuously damaged. The use of imaging diagnostic (plain x-ray of the foot or magnetic resonance) confirms the clinical diagnosis. It is worth mentioning that the initial stage of metatarsal deformation will not be visible on a typical X-ray image in two projections. At the initial stage of the disease, magnetic resonance is the most sensitive imaging method.

In 1966, Sidney N. Eichenholtz described in his monograph cases of various osteoarticular lesions on the basis of 68 available radiograms, and introduced the term "Charcot joint".[6] (Tab. I)

II. Case report

A 58-year-old patient was admitted to a surgical ward for the treatment of a massive wound located between the second and third toe of the right foot. Medical history interview revealed a long-term insulin-dependent diabetes with numerous organ complications. Previously, the patient required amputation of the hallux of the right foot and the second toe of the left foot. Before the admission, the patient was provided with ambulatory care due to a non-healing clavus located on the plantar surface. Repeated resections of the clavus did not allow to eliminate this hyperkartotic lesion and during one of the visits, after a surgical resection of the clavus, an intervention to stop bleeding from blood vessels of the plantar surface was necessary.

Advanced destructive changes of the right foot were visible on an X-ray image (Fig. 1). The radiograms revealed the loss of the foot arch, fixed, spontaneous dislocation of the metatarsophalangeal joint, and foci of decreased mineralization in the tarsal and metatarsal bones (Fig. 2). No changes were observed at the level of the ankle joint and calcaneus.

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Table I
CLASSIFICATION OF THE CHARCOT NEURO-ARTHROPATHY (EICHENHOLTZ 1966)

Stage	Clinical characteristics	X-ray features
Stage I (development or fragmentation) Stage II (coalescence)	Edema of diabetic foot. Inflammatory stage: - odematous, - erythematous, - hot and hyperemic foot. Gradual remission of the inflamatory skin signs. Reduction of edema.	Luxation, subluxation, dislocation of joint. Peri-articular fractures. Foot deformity. Decrease of diabetic foot stability. Osteoporosis, resorption, bone debris. Increase of diabetic foot stability.
Stage III (consolidation or reparation)	Absence of inflamatory signs, Absence edema of diabetic foot.	Consolidated remodeling and deformation of the foot bones and joints.

Figure 1. Displacement of the matatarsophalangeal joint (MTP). Sanders and Frykberg's classification - pattern I

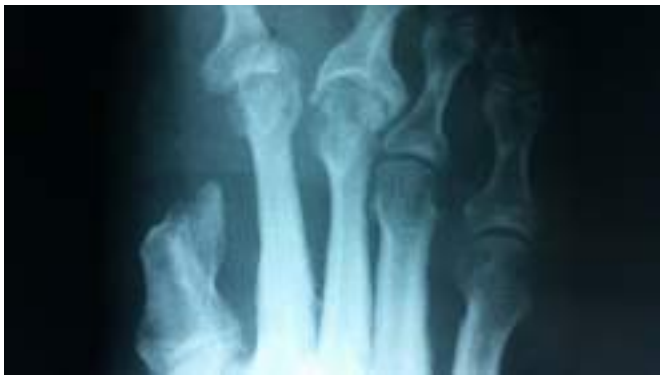

Figure 2. Previous amputation of the hallux

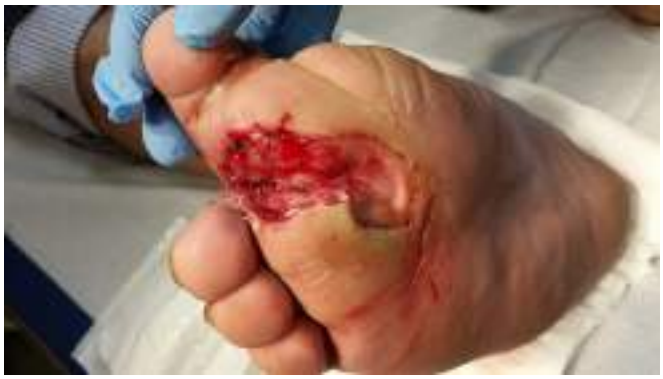

Figure 3. Management of the bleeding after initial debridement

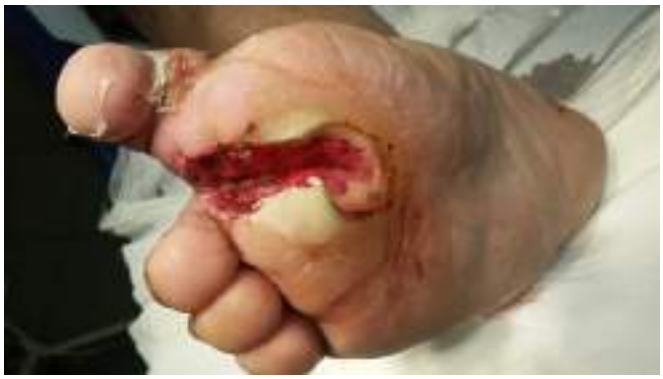

Figure 4. 1st wound dressing change after 48 hours of NPWT.

On the day of admission, after initial preparation of the bed and edge of the wound, a negative pressure dressing Vivano Tec produced by Hartmann was used. A 10 cm x 7.5 cm x 3.3 cm sponge had been modeled to the elliptic shape of the wound. Due to the fact that the wound was located between toes, it required special attention to maintain the seal of the dressing (Fig. 3).

Continuous mode of negative pressure (-125 mmHg) was applied. For better protection, the skin near the wound was covered with a stripe of silicon drape. The first dressing remained on the wound for 48 hours. During the first change of dressing, normal formation of granulation tissue and early signs of epithelialization of the wound edges were observed (Fig. 4). Negative pressure therapy was continued. The second negative pressure dressing remained intact for 72 hours. The third dressing set remained on the wound for 9 days due to full impermeability (Fig. 5). After the last 216-hours course the negative pressure therapy was finished and a reduction of the total wound surface was observed, as well as a proliferation of the vital, healthy granulation tissue. The tendency of spontaneous wound edges approximation was also observed (Fig. 6).

Finally, the upper pole of the wound, between the second and third toe, was approximated with the use of an interrupted dermal suture (Dafilon 2.0). The defect, dressed in such a way, healed properly by further approximation of the wound edges (Fig. 7). No foci of necrosis, abnormal exudate, or clinical symptoms of infection were observed during the treatment of the wound. In this case, the course of healing of the wound in a deformed diabetic foot was finished successfully.

Figure 5. Granulation tissue and maceration of the epidermis

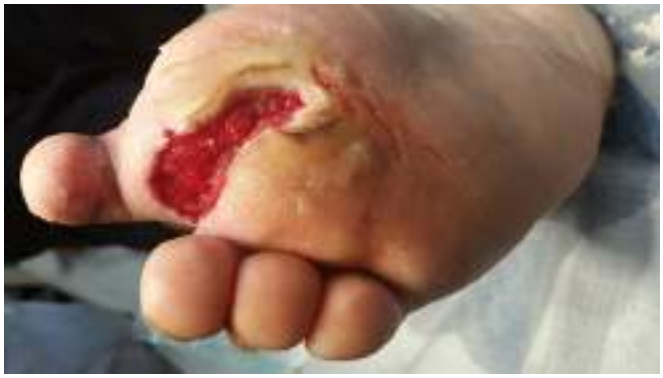

Figure 6. Evident reduction of tissue loss after 216 hours of continuous NPWT

III. Discussion

Peripheral neuropathy relates to 29% of patients with diabetes mellitus.[7] Charcot foot is a specific form of peripheral neuropathy in diabetes. Continuous destruction of nerve fibers leads to an autonomic neuropathy. Apart from hyposesthesia, blood circulation disorders and intensification of bone destruction are common this situation. The latter are due to the prevalence of osteolytic activity of osteoclasts on osteoblasts. The pathogenesis of Charcot foot is chronic, multifactorial, and progressive.

The chronic form of the disease is characterized by the reduction of edema, reduction of redness, reduction of

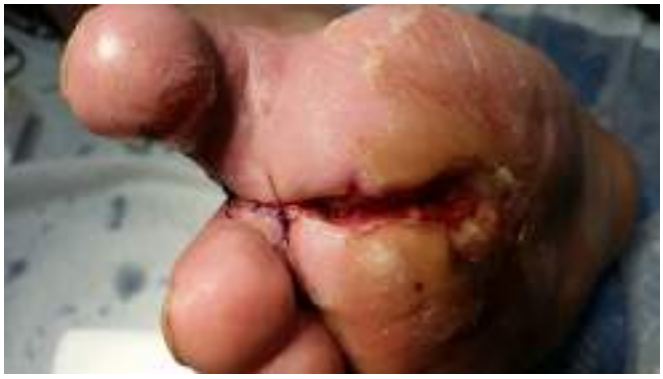

Figure 7. Postponed skin sutures in the diabetic foot

increased temperature and leads to permanent anatomical changes at the level metatarsal level. A diabetic foot deformed in this process becomes more prone to repetitive minor unnoticed injuries that can further ulcerate. Disruption of the skin continuity opens the door for infection. Infectious complications in cases of diabetic foot are the most common cause of amputations in diabetes mellitus.

Neuropathy can relate to peripheral nerves that are responsible for transferring pain stimuli, as well as autonomic nerves determining the cellular equilibrium of the bones. In the advanced form of the diabetic foot, there is a prevalence of destructive activity of osteoclasts at the expense of osteogenic activity of the osteoblasts. Autonomic neuropathy leads to an increased arterial blood inflow by impairing arteriovenous capillary connections.[8, 9] Clinically, increased temperature, redness of foot skin and hypervolemia occur.[10–12] Increased blood flow also influences the bone tissue, leading to its increased resorption with bone mineral density loss.[13, 14]

Peripheral neuropathy, referred to as the distal sensorimotor polyneuropathy is responsible for the loss of the protective pain, temperature, and touch sensation. A foot that is devoid of innervation is exposed to various kinds of damage that also disturb proper anatomy of the foot.

Another cause of foot damage is an increase of non-enzymatic glycation of collagen. Impaired collagen metabolism leads to the weakening of tendons, ligaments, therefore, leading to the change of foot biomechanics.[15]

In the case of Charcot foot, there is an increased pressure impacting the plantar surface.

Characteristic clinical symptoms of Charcot neuroarthropathy are described using the 5D acronym according to Rajbhandari:[16]

- joint distension
- dislocation
- debris
- disorganization
- increased density

If observed, any symptoms from foot fully oblige to prepare X-ray image of parts of the metatarsus. Visible changes characteristic for osteoarthropathy are dislocations and subluxations of foot joints, periarticular osteoporosis, bone tissue resorption, the presence of debris, loss of the foot arch, traces of previous surgical interventions.

Anatomical classification by Sanders and Frykberg differentiates five areas of foot damage:[17]

I (15%) lesions at the level of the forefoot, affecting the metatarsophalangeal (MTP) and interphalangeal (IP) joints.

II (40%) affecting the tarsometatarsal joint (TMT). Lisfranc joint.

III (30%) affecting the cuneonavicular, talonavicular, and calcaneocuboid joints. Chopart joint.

IV 10%) affecting the ankle joint.

V (5%) affecting th ecalcaneus.

According to the American Orthopedic Foot and Ankle Society, an optimal manner of treating various lesions referred to as Charcot neuroarthropathy still evokes therapeutic

controversies. It is one of the two most often discussed problems in this profession.[18]

The most common cause of Charcot osteoarthropathy is diabetes mellitus. The most common location of lesions is the foot. Unfortunately, in most cases, accurate diagnosis is significantly delayed. In Pakarinen's observation, the time to accurate diagnosis was 29 weeks after emergence of first symptoms [19]. In another study, the delay in diagnosis was 10 weeks.[19]

Appropriate treatment in advanced Charcot neuropathic osteoarthropathy is still a great therapeutic challenge. One of the available ways of Charcot foot treatment is an orthopedic operation. During an open surgery, any possible dislocation or subluxation of foot joints can be corrected. After repositioning of the joints, internal or external stabilization is used. Inserting an implant into foot bones is considered by many to be an unjustified practice.

In most cases, diabetic foot is treated conservatively. After initial termination of the inflammatory process, necrotic and ischaemic tissues are removed. Unfortunately, most cases of complicated diabetic foot require more radical procedures. In many patients, limbs need to be amputated below the level of talocrural joint. Such surgeries are referred to as minor amputations. Radical procedures at the level of the thigh or shin are called major amputations.

An optimal management in cases of Charcot neuropathic osteoarthropathy still evokes therapeutic controversies. Some patients require an orthopedic intervention with the use of bone connecting materials. A significant group of patients is treated conservatively due to an increased risk of infectious complications and coexistence of systemic complications of diabetes. Any form of diabetic foot is a real threat to lower limb amputation. A special case of diabetic neuropathy is Charcot arthropathy. Deformation of the anatomy of the foot and impairment of its functioning increase the risk of various complications. Therefore, it is important to take early and appropriate multidisciplinary care of a foot with 'special needs'. In the above example, the immediate use of negative pressure treatment protected the structure of the foot from infection in Charcot arthropathy, as well as allowed for an effective healing of the wound.

IV. Conclusions

As shown in the example above, the use of efficient methods of negative pressure therapy can be a recommended compromise between radical surgical treatment and conservative, expectant management. In many cases, negative pressure therapy inhibits further foot damage. Negative pressure therapy does not, in any way, supersede other available therapeutic possibilities but it remains the only effective method of treatment for many patients.

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Negative pressure wound therapy in a chronic radiation dermatitis of the scalp.

Tomasz Banasiewicz, Wojciech Francuzik

CASE REPORT

***Abstract*— The adverse reactions of late tissue damage in irradiated patients may range from bothersome symptoms that negatively affect their quality of life to severe life-threatening complications. We describe the case of a female patient, EM, 54 years old who presented with chronic radioation dermatitis on her scalp following radiotherapy (60 Gy in 30 fractions over 6 weeks) for a meningioma of the right frontal region.**

We decided to introduce the negative pressure wound therapy (NPWT) with a portable Avelle^{TM} system (Convatec; UK) in a continuous therapy mode with -80 mmHg vacuum. The dressing was subsequently applied directly on the skin of the skull and braced with extra adhesive strips and changed every 3 days (3 changes in total). During the therapy we didn't observed any side-effects or complications, system was well tolerated by the patient and lead to the improvement of the wound promoting the granulation and decreasing the exudate from the wound.

***Keywords*—venous ulcers, negative pressure wound therapy, chronic wound, chronic venous insufficiency, skin graft, silver, pain**

I. Introduction

SEVERE skin, soft tissue and bone infections are delayed complications of irradiation of the skull in the course skull and brain tumor radiotherapy.[1] The adverse reactions of late tissue damage in irradiated patients may range from bothersome symptoms that negatively affect their quality of life to severe life-threatening complications.[2] In patients with advanced necrosis it may lead to severe tissue and bone defects, with vast exposure of the dura mater surface and severe infections.[3] Up to 95% of patients who undergo irradiation develop adverse skin lesions.[4] Treatment of the radiotherapy induced skin, soft tissue, and bone adverse lesions is complicated, therefore prolonging the healing process and negatively impacting the patient's quality of life.[5] Topical interventions to prevent acute radiation dermatitis in head and neck cancer patients are still not effective and not routinely used.[6] This report follows the recommendations from the CARE Statement for writing case reports.[7]

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Table I
Characteristics of the patients

Date	Event
10/2016	The diagnosis of a meningioma of the right frontal region
11/2016	The neurosurgical local tumor resection
12/2016	Beginning of the Radiotherapy
01/2017	Radiotherapy concluded (60 Gy in total) Patient observed redness of the scar and pruritus
10/2017	Patient observed redness of the scar and pruritus
11/2017	First ulceration formed on the scalp
11/2017	Subsequent ulceration formed in the next weeks
11/2017	Topical therapy sine effectu
12/2017	Hyperbaric therapy sine effectu
09/01/2018	Initiation of the negative pressure wound therapy
18/01/2018	Significant improvement of the clinical symptoms

II. Patient Information

We describe the case of a female patient, EM, 54 y.o. who presented with a chronic non-healing wound on her scalp. The patient was diagnosed with a brain tumor in 2016 (meningioma of the right frontal region) and subsequently treated with the neurosurgical local resection followed by the postoperative radiotherapy (60 Gy in 30 fractions over 6 weeks) with good results, no side effects. The oncological therapeutic effect was satisfactory and at the time of the ambulatory admission at our department (1/2018) the patient showed no signs of recurrence of the malignancy.

In October 2017, the patient observed an irritation of the skin in the postoperative wound (scar) with itching and redness. Symptoms occurred 10 months after the last irradiation session. A few weeks later, the lesions transformed into a single ulcer 2 cm x 4 cm, and in the subsequent weeks additional ulcerations occurred. Patient reported for a visit in our office in January 2018.

The patients presented no other pathologies and concomitant diseases, no familial history of neoplasia, nor signs of cachexia were present. The patient didn't smoke and use alcohol.

III. Clinical Findings

In January 2018 patient was admitted to the outpatient Department of the General, Endocrinological Surgery and Gastrointestinal Oncology. On examination, there were 3 distinct longitudinal ulcers in various size, ranging between 2

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cm and 6 cm in width. Ulcerations increased in depth and size in 2 preceding weeks exposing the bone with necrosis and soft tissue inflammation at the lesion borders (Fig. 1). The skin of the scalp appeared poikilodermic with telangiectases, hypo- and hyper-pigmentation, and atrophy. A yellowish hue indicating radiation elastosis was present. The wound bed was covered with a thick yellowish exudate secreting into the wound dressing (Fig. 2). The chronic radiation dermatitis was diagnosed based on a clinical examination and patient's medical history.

IV. Therapeutic Intervention

Before the patient presented in our clinic, she underwent lesion debridement and the dense secretion was evacuated from the wound bed. Subsequently she was treated with the standard wound dressings (0,1% octenidine dihydrochloride and 2% phenoxyethanol wash and alginate wound dressings), changed 2 times daily without improvement. In December 2017, 6 weeks of hyperbaric therapy (5 x 60 minute sessions per week with 1,4-1,8 ATA pressure chamber) was introduced, again, without significant improvement.

We decided to introduce the negative pressure wound therapy (NPWT) with a portable Avelle^{TM} system (Convatec; UK). The localization of the wounds proved problematic for the wound dressing application as it was difficult to provide a reliable seal for the negative pressure therapy system. We chose a Hydrofiber^{TM} wound dressing of 12x21 cm (Avelle^{TM}, Convatec, UK), cut in the middle to reduce its size and make the application easier. To provide a better seal for the system, a sealing "frame" of the stoma paste (Stomahesive, Convatec) was put on the surface of the Hydrofiber^{TM} (Fig. 3). The dressing was subsequently applied directly on the skin of the skull and braced with extra adhesive strips, especially at the side that was previously cut, to secure it in place (Fig. 4).

We set the device initially to provide continuous mode therapy with -80 mmHg vacuum. We changed the wound dressing every 3 days (3 changes in total). At the first wound dressing change, we could still observe the signs of inflammation with pus secretion, erythematous skin irritation and the oedema of the soft tissues (Fig. 5). After another 3 days the oedema decreased significantly, similarly to the exudate secretion and the skin erythema. After the last dressing removal (9 days after the initiation of the NPWT) we observed no secretion, no soft tissue oedema, and minimal skin erythema (Fig. 6). Patient did not report pain or itching (which were moderately severe before). Patient was satisfied with the outcome and we decided together with the patient to repeat the course of the hyperbaric therapy where we referred her. The patient used the standard topical skin care with hypoallergenic, 10% urea containing emollients thereafter.

V. Discussion

Skin lesions in the course of radiotherapy are a common side effect, observed in various localizations and regions.[8] Every year, 1.2 million cancer patients receive radiation therapy in the United States only. Late radiation-tissue-injury

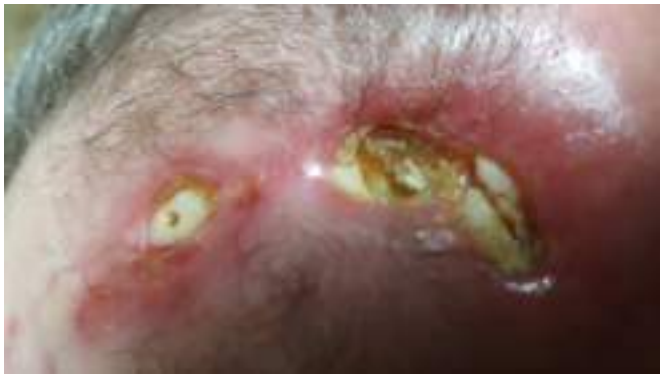

Figure 1. The scalp on admission the our department. Radiodermatitis with multiple skin lesions visible.

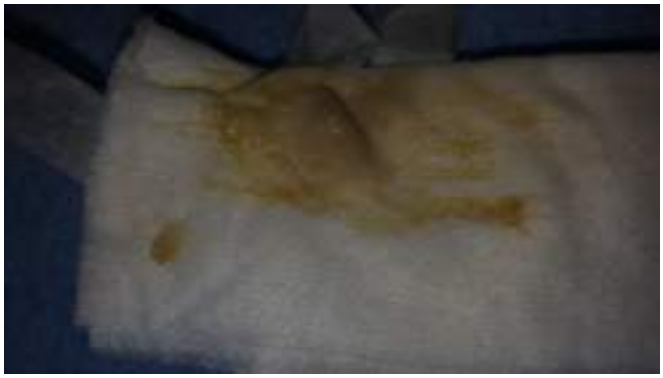

Figure 2. The standard gauze wound dresing after dressing change. Notice the significant amount of exudate.

occurs in an estimated 5-15% of these patients. Tissue injury can include skin necrosis, which can lead to chronic nonhealing wounds.[9] In severe cases, depending on localization, soft tissue necrosis, inflammation, and infection can also occur. Craniofacial radiation can lead to severe complicated wounds and, if used in childhood, to various abnormalities, from soft-tissue deficiency to osseous deformities.[10]

One of the effective method of treatment of complicated wounds is negative pressure wound therapy (NPWT). NPWT leads to the fast elimination of the septic conditions, improving the tissue vascularization, stimulating the fibroblast proliferation and migration, managing the wound exudation, and accelerating wound healing.[11] The use of this method has also been proved to improve the treatment results of severe surgical infections in neurosurgery.[12] One of the earliest reports describing treatment with vacuum assisted closure (VAC) therapy on complex cranial wounds was published by Andrews et al., where two patients with traumatic scalp injuries were successfully treated without any complications.[13] There are reports in the literature that describe the use of NPWT in scalp defects with exposed periosteum, and fewer reports describing defects in periosteum with exposed dura mater.[3, 14]

The NPWT is a more effective therapy for bone-exposed wounds than conventional gauze dressing therapy.[15] It can promote bone-exposed-wounds to heal by increasing collagen contents and angiogenesis while reducing inflammatory cells

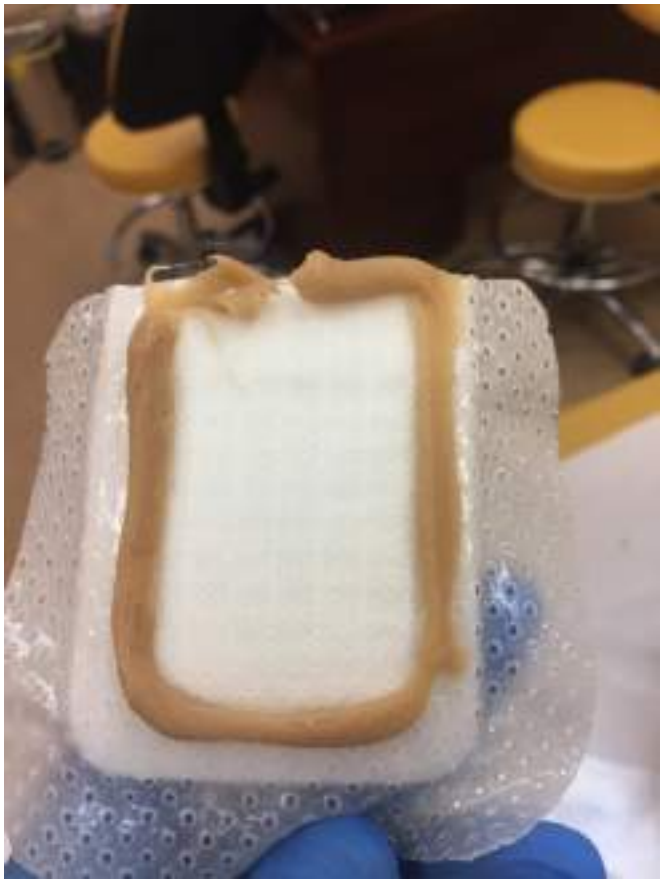

Figure 3. The Hydrofiber™ negative pressure wound dressing with a sealing stoma paste "frame".

Figure 4. The scalp after introducing the portable negative pressure wound therapy system

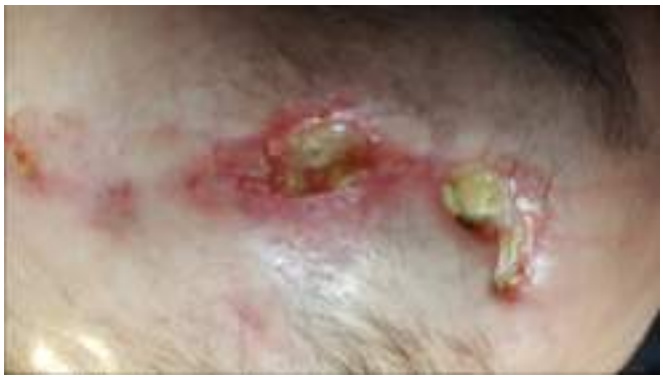

Figure 5. Therapeutic outcome after 2 wound dressing changes and 6 days of portable NPWT in a continuous mode of -80 mmHg.

infiltration, reducing wound infection rates, and inducing an ordered collagen arrangement.[15] In the head region NPWT can also be effectively used in patients with osteoradionecrosis.[16]

Described NPWT therapies in head and neck wounds mainly used the standard vacuum devices with canisters. Many studies in the last years confirmed the effectiveness of the disposable, canister-less negative pressure wound therapy system.[17] The results of the literature reviews show, that the portable, canisterless NPWT system meets or exceeds healing rates previously reported in the literature for canister-based systems.[17] This simple device enhancement offers a more portable NPWT option, particularly effective in the treatment of shallow wounds[18] and increases patient compliance.

In our case, we used the Avelle™ (Convatec; UK) system, recently introduced into the medical market.[19, 20] Avelle™ uses Hydrofiber™ dressings and the pressure used in therapy is a continuous -80 mmHg. The very common technical problem with application of the dressings, also NPWT dressing, is the curvature of the body region. Location of the NPWT system on the skull was uncertain, therefore, to avoid decompression, we decided to use the stoma paste. This modification, improved the seal and prolonged the time between wound dressing changes as described previously.[21, 22] It is most commonly combined with standard "big wound dressings" based on the polyurethane sponge and canister-based systems. There portable dressing systems may also be easily cut to obtain the optimal form and size for the wound bed. It is necessary to remember, that in such case the exudate collection volume will be lower, and the surgeon must account for that.

During the therapy we didn't observed any side-effects or complications, system was well tolerated by the patient and lead to the improvement of the wound promoting the granulation and decreasing the exudate from the wound.

To conclude, the portable NPWT can be successfully used in cases of complicated skull wounds with soft tissue and

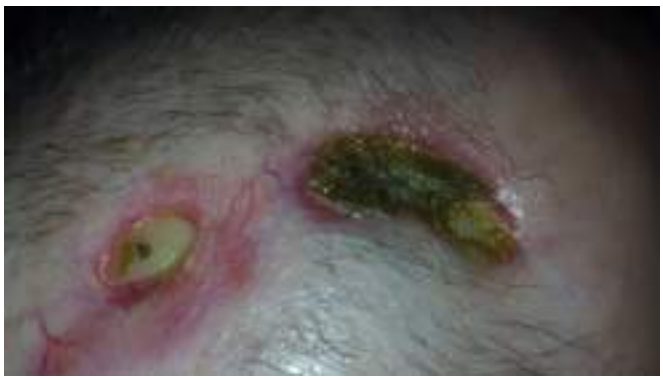

Figure 6. Therapeutic outcome after 3 wound dressing changes and 9 days of portable NPWT in a continuous mode of -80 mmHg.

bone defects. However, there are no data to determine the efficacy of this method relative to other wound healing methods. It should be remembered, however, that the NPWT allows to combine various therapeutic methods and poses a new option for the treatment of difficult no-healing skull wounds.

VI. Informed Consent

The patient provided an informed consent for the publication of this case along with unidentifiable photographic material.

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A simple and low-cost technique of creating a Negative Pressure Wound Therapy (NPWT) machine on the example of a severe phlegmon of lower limb in lower socio-economic area.

Marcin Kiszka, Filip Kazubski, Magdalena Maj, Tomasz Banasiewicz

CASE REPORT

***Abstract*—The Negative Pressure Wound Therapy (NPWT) is an approved method of healing lower extremity ulcers of various origin, accelerating the wound closure process, thus decreasing the hospital-stay time and lowering the cost of the treatment. Although it is scarcely needed in developing countries such as Kenya, there is a lack of official supplier of the NPWT equipment. We present an improvised method of constructing a reliable and effective NPWT dressing from widely available tools in a case of treating a post-traumatic phlegmon in an HIV-positive patient with good outcomes. Improvised NPWT may provide an effective treatment of chronic wounds regardless of their origin and may be successfully used in low-income parts of the world.**

***Keywords*—improvised negative pressure wound therapy, negative pressure wound therapy, phlegmon**

I. Introduction

THE Negative Pressure Wound Therapy (NPWT) is an approved method of healing lower extremity ulcers of various origin, accelerating the wound closure process,[1] thus decreasing the hospital-stay time and lowering the cost of the treatment.[2] Unfortunately, many developing countries such as Kenya lack an official supplier or producer of commercial NPWT equipment. The import of such could be an option, but as the insurance or reimbursement is very limited and most of the time patients have to pay for medical care themselves,[3] it is very unlikely to become a standard of care. The goal of this case report is to demonstrate a working NPWT set using materials that are economically and commercially available in these countries.

II. Case Report

A 30-year-old man was admitted to Bishop Kioko Catholic Hospital in Machakos, Kenya with an ulceration of the left lower limb, which developed as a result of an injury that had happened a month earlier. He denied applying any dressing to the wound, nor disinfecting it. The patient complained of the itching sensation while the infection was spreading to the subcutaneous area. He decided to ask for medical care only after the ulceration started to impede his work.

On admission, the patient was in good general condition, he didn't demonstrate fever, general malaise, tachycardia nor tachypnoea. He was oriented to person, place and time. The examination of his leg revealed tenderness, swelling and increased warmth. There was an open wound in the lower third of the patient's crus with palpable subdermal abscesses and visible purulent leakage. He was diagnosed with cellulitis by the local physician. He was a known HIV-positive patient, although a CD4-count or the date of diagnosis was unknown. He denied having any other comorbidities. The patient had the subdermal abscesses drained and received an intravenous antibiotics treatment. He also had the dressings changed every 2 days.The worsening of the clinical symptoms progressed and the subdermal inflammation has spread. A week later a new diagnosis of phlegmon was proposed (Fig. 1) and a more aggressive line of treatment was introduced.

After a week of such treatment, we decided to apply a self-made NPWT dressing to accelerate the healing process. Our NPWT dressing was made of (Fig. 2):

- a typical dishwashing foam, sterilized in the steam autoclave with its upper, abrasive layer removed
- a breakfast foil, sterilized by bathing in apovidone solution for half an hour
- a surgical suction machine, with a sterilization tube.

After thorough wound bed preparation the foam was cut to fill in the biggest wound, then the suction tube was placed onto it. One person was stabilizing the tube and the other covered the whole crus with foil. A few more layers of the foil were used to ensure robustness between the foam and the suction tube. Finally, the suction tube was connected to the suction machine. Due to the foil transparency, we were able to check whether the negative pressure was properly applied. There was no evidence of leakage, thus we set the negative pressure to continuous -0.2 kPa, which is an equivalent

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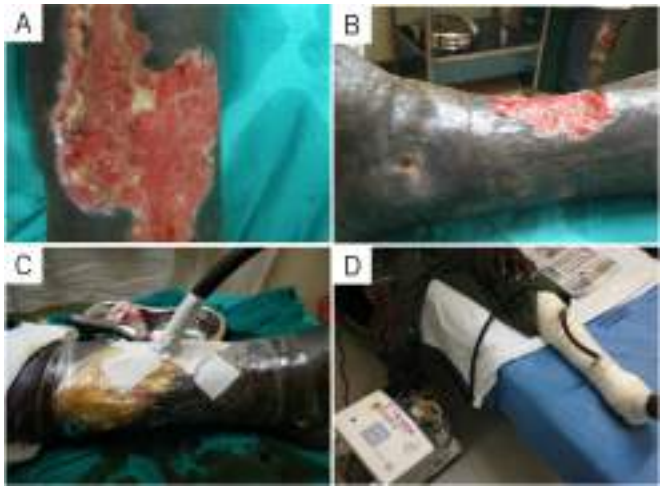

Figure 1. Components of the improvised NPWT, a dishwashing foam and a breakfast foil were used. B) Wound bed prepared for NPWT. c) A foam, breakfast foil and suction tube applied.D) A completed dressing

to -150 mmHg and observed the system for any signs of pressure drops. The patient did not report any discomfort during the treatment. He could move to the suction unit without interrupting the work of the device. 3 days later, we revised the wound and noticed a significant vascularization and granulation on over half of the wound's surface. There was less pus, the inflammation did not progress and the healing process after those 3 days was faster compared to the previous 1 week of moist dressing therapy. The exudate diminished and inflammation shrank.

The healing process was rapid and uncomplicated. The patient was discharged in a stable condition shortly afterward on his own demand. The reason was his economical status. Because of that, we could not continue our improvised NPWT. We could observe his recovery when he came every 2 days for the dressing change and debridement for the next 2 weeks until the time we had to leave Kenya (Fig. 3). Unfortunately, we could not witness his full recovery, nor had we any contact to confirm it.

III. Discussion

It has been reported that a low-cost negative pressure wound therapy can be beneficial to the patients with chronic wounds of the lower extremity in lower socioeconomic groups.[4] In comparison to our study, it was based on an Indian population and the negative pressure was achieved by connecting the suction tube to a vacuum in the wall, not to the suction machine. Despite its adequacy, it has to be stated that the study had to be withdrawn because of the lack of ethics committee approval. The lack of support from official producers is only one of many obstacles to implement NPWT in Sub- Saharan medical facilities successfully. Collection, preparation, and adjustment of the whole improvised NPWT machine lasted a whole day long. Covering the wound took almost an hour itself due to the technical difficulties. The local medical staff definitely needs a thorough training in NPWT and chronic wound therapy. However, we still believe

Figure 2. A) Anterior view of the wound. B) A view of the wound laterally. Arrow points to an abscess

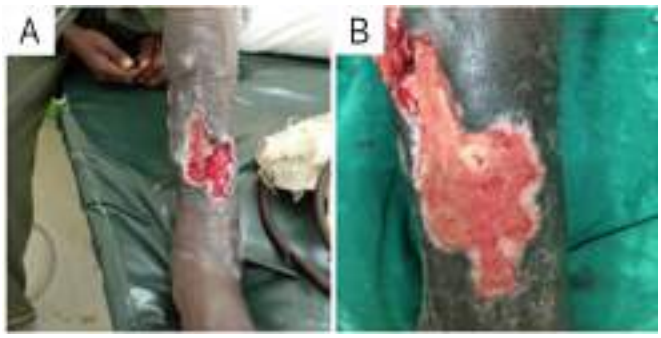

Figure 3. A) The wound after 3 days of continuous NPWT. C) The wound a week later, after debridement and dressing change.

that concerning the great percentage of diabetic foot and limb amputations,[5] pressure ulcers,[6] and a high percentage of HIV-positive individuals in Kenyan population[7] introducing the NPWT would be beneficial for patients and medical workers alike.

A limitation of this study was our inability to follow-up on the patient's recovery.

IV. Conclusions

Improvised NPWT may provide an effective treatment of chronic wounds regardless of their origin and may be successfully used in low-income parts of the world.

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U pacjentów z zaburzeniami czynności nerek: Dostosowywanie dawki nie jest konieczne u pacjentów z łagodnymi lub umiarkowanymi zaburzeniami czynności nerek (klirens kreatyniny 30 ml/min do 79 ml/min). Dostosowywanie dawki nie jest konieczne u pacjentów regularnie poddawanych hemodializie (3 razy w tygodniu); dalbawancyna może być podawana bez względu na czas hemodializy. U pacjentów z przewlekłymi zaburzeniami czynności nerek, u których klirens kreatyniny wynosi <30 ml/min i którzy nie są regularnie poddawani hemodializie, zalecany schemat dawkowania dalbawancyny raz w tygodniu należy zmniejszyć do dawki 750 mg, a następnie po tygodniu o 375 mg. U pacjentów z zaburzeniami czynności wątroby: Dostosowywanie dawki dalbawancyny nie jest zalecane u pacjentów z łagodnymi zaburzeniami czynności wątroby (stopień A w klasyfikacji Childa-Pugha). Należy zachować ostrożność, przepisując dalbawancynę pacjentom z umiarkowanymi lub ciężkimi zaburzeniami czynności wątroby (stopień B i C w klasyfikacji Childa-Pugha), ponieważ nie ma danych umożliwiających określenie właściwego dawkowania. Dzieci i młodzież: Nie określono jeszcze bezpieczeństwa ani skuteczności stosowania dalbawancyny u dzieci w wieku od urodzenia do poniżej 18 lat. **Sposób podania:** *Podanie dożylne.* Produkt leczniczy Xydalba musi być odtworzony, a następnie rozcieńczony przed podaniem w infuzji dożylnej przez 30 minut. Należy zapoznać się z instrukcją dotyczącą odtwarzania i rozcieńczania tego produktu leczniczego przed podaniem. **Przeciwwskazania:** Nadwrażliwość na substancję czynną lub na którąkolwiek substancję pomocniczą (mannitol (E421), laktoza jednowodna, kwas solny (do ustalenia pH), sodu wodorotlenek (do ustalenia pH)). **Specjalne ostrzeżenia i środki ostrożności dot. stosowania:** Reakcje nadwrażliwości: Produkt leczniczy Xydalba należy z ostrożnością podawać pacjentom, o których wiadomo, że są nadwrażliwi na inne glikopeptydy, ze względu na możliwość wystąpienia krzyżowej nadwrażliwości. Jeżeli wystąpi reakcja alergiczna na produkt leczniczy Xydalba, należy przerwać jego podawanie i zastosować właściwe leczenie reakcji alergicznej. Biegunka spowodowana przez *Clostridium difficile*: Podczas stosowania prawie wszystkich antybiotyków obserwowano związane z leczeniem przeciwbakteryjnym zapalenie okrężnicy i rzekomobłoniaste zapalenie okrężnicy, których przebieg może być od łagodnego do zagrażającego życiu. Z tego względu należy brać pod uwagę to rozpoznanie u pacjentów z biegunką występującą podczas lub po zakończeniu leczenia dalbawancyną. W takich przypadkach należy rozważyć przerwanie podawania dalbawancyny i zastosowanie leczenia wspomagającego oraz specyficznego dla zakażenia *Clostridium difficile*. U tych pacjentów nigdy nie należy stosować produktów leczniczych tłumiących perystaltykę. Reakcje związane z infuzją: Produkt leczniczy Xydalba podaje się w infuzji dożylnej, z wykorzystaniem całkowitego 30-minutowego czasu trwania infuzji, w celu zminimalizowania ryzyka reakcji związanych z infuzją. Szybkie infuzje dożylne przeciwbakteryjnego glikopeptydu mogą przyczynić się do wystąpienia reakcji przypominających „zespół czerwonego człowieka", który obejmuje nagłe zaczerwienienie górnych części ciała, półrzywę, świąd i (lub) wysypkę. Zaprzestanie podawania infuzji lub jej spowolnienie może spowodować ustąpienie tych reakcji. Zaburzenia czynności nerek: Informacje dotyczące skuteczności i bezpieczeństwa stosowania dalbawancyny u pacjentów, których klirens kreatyniny wynosi <30 ml/min są ograniczone. Na podstawie opinekcji, dostosowywanie dawki jest konieczne u pacjentów z przewlekłymi zaburzeniami czynności nerek, u których klirens kreatyniny wynosi <30 ml/min i którzy nie są regularnie poddawani hemodializie. Zakażenia mieszane: W przypadku zakażeń mieszanych, jeśli się podejrzewa obecność bakterii Gram-ujemnych, pacjentów należy leczyć odpowiednimi lekami przeciwbakteryjnymi działającymi na bakterie Gram-ujemne. Drobnoustroje niewrażliwe: Zastosowanie antybiotyków może promować namnażanie drobnoustrojów niewrażliwych. Jeżeli podczas terapii wystąpi nadkażenie, należy wdrożyć odpowiednie postępowanie. Ograniczenia danych klinicznych: Dane dotyczące bezpieczeństwa stosowania i skuteczności dalbawancyny w przypadku zastosowania więcej niż dwóch dawek (w odstępie jednego tygodnia) są ograniczone. W kluczowych badaniach w przypadku ABSSSI rodzaje leczonych infekcji były ograniczone jedynie do cellulitis/ropni, ropni i infekcji ran. Brak doświadczenia dotyczącego stosowania dalbawancyny w leczeniu pacjentów z silnie obniżoną odpornością. **Ciąża i laktacja:** Nie ma danych dotyczących stosowania dalbawancyny przez kobiety w ciąży. Badania na zwierzętach wykazały toksyczne działanie na reprodukcję. Xydalbarnie jest zalecana w okresie ciąży, o ile nie jest to bezwzględnie konieczne. Nie wiadomo, czy dalbawancyna przenika do mleka matki (mleka ludzkiego). Niemniej dalbawancyna przenika do mleka samic szczurów karmiących piersią i może również przenikać do mleka ludzkiego. Dalbawancyna nie wchłania się dobrze po podaniu doustnym, niemniej nie można wykluczyć wpływu dalbawancyny na florę żołądkowo-jelitową oraz florę jamy ustnej karmionego piersią niemowlęcia. Należy podjąć decyzję o kontynuacji/zaprzestaniu karmienia piersią lub kontynuacji/zaprzestaniu leczenia produktem Xydalba, biorąc pod uwagę korzyści z karmienia piersią dla niemowlęcia oraz korzyści z terapii dla kobiety. Płodność: Badania na zwierzętach wykazały obniżoną płodność. Potencjalne ryzyko, na które narażeni są ludzie jest nieznane. **Działania niepożądane:** Podsumowanie profilu bezpieczeństwa: W fazie 2/3 badań klinicznych dalbawancynę otrzymało 2473 pacjentów. Była ona podawana albo w infuzji jako dawka pojedyncza 1500 mg, albo w dawce 1000 mg, a następnie po tygodniu w dawce 500 mg. Najczęściej występującymi działaniami niepożądanymi występującymi u ≥1% pacjentów leczonych dalbawancyną były: nudności (2,4%), biegunka (1,9%) oraz ból głowy (1,3%), i zwykle miały lekkie lub umiarkowane nasilenie. Tabelaryczny wykaz działań niepożądanych: W fazie 2/3 badań klinicznych z zastosowaniem dalbawancyny zidentyfikowano poniższe działania niepożądane. Działania niepożądane podano zgodnie z klasyfikacją układów i narządów oraz według częstości występowania. Kategorie częstości występowania zostały opisane zgodnie z następującymi normami: bardzo często (1/10), często (1/100 do <1/10), rzadkie (1/1000 do <1/100), bardzo rzadkie (1/10 000 do <1/1000).

Klasyfikacja układów i narządów	Częste	Rzadkie	Bardzo rzadkie
Zakażenia i zarażenia pasożytnicze		zakażenia grzybicze pochwy i sromu, zakażenia dróg moczowych, infekcje grzybicze, zapalenie okrężnicy wywołane *Clostridium difficile*, kandydoza jamy ustnej	
Zaburzenia krwi i układu chłonnego		anemia, trombocytoza, eozynofilia, leukopenia, neutropenia	
Zaburzenia układu immunologicznego			reakcje anafilaktoidalne
Zaburzenia metabolizmu i odżywiania		zmniejszony apetyt	
Zaburzenia psychiczne	ból głowy	bezsenność	
Zaburzenia układu nerwowego		zaburzenia smaku, zawroty głowy	
Zaburzenia naczyniowe		nagłe zaczerwienienie, zapalenie żył	
Zaburzenia układu oddechowego, klatki piersiowej i śródpiersia		kaszel	skurcz oskrzeli
Zaburzenia żołądka i jelit	nudności, biegunka	zaparcie, ból brzucha, dyspepsja, uczucie dyskomfortu w jamie brzusznej	
Zaburzenia skóry i tkanki podskórnej		świąd, pokrzywka	
Zaburzenia układu rozrodczego i piersi		świąd sromu i pochwy	
Zaburzenia ogólne i stany w miejscu podania		*reakcje związane z infuzją*	
Badania		zwiększona aktywność dehydrogenazy mleczanowej we krwi, zwiększona aktywność aminotransferazy alaninowej, zwiększona aktywność aminotransferazy asparaginianowej, zwiększone stężenie kwasu moczowego we krwi, nieprawidłowe wyniki testu czynności wątroby, zwiększona aktywność aminotransferaz, zwiększona aktywność fosfatazy zasadowej we krwi, zwiększona liczba płytek krwi, zwiększona temperatura ciała, zwiększona aktywność enzymów wątrobowych, zwiększona aktywność gamma-glutamylotransferazy	

Opis wybranych działań niepożądanych: Ototoksyczność związane z klasą leków: Ototoksyczność jest związana ze stosowaniem glikopeptydu (wankomycyny i teikoplaniny); u pacjentów otrzymujących w skojarzeniu lek ototoksyczny, taki jak aminoglikozyd, ryzyka ototoksyczności może być zwiększone. Zgłaszanie podejrzewanych działań niepożądanych: Po dopuszczeniu produktu leczniczego do obrotu istotne jest zgłaszanie podejrzewanych działań niepożądanych. Umożliwia to ciągłe monitorowanie stosunku korzyści do ryzyka stosowania produktu leczniczego. Osoby należące do fachowego personelu medycznego powinny zgłaszać wszelkie podejrzewane działania niepożądane za pośrednictwem krajowego systemu zgłaszania. **Podmiot odpowiedzialny:** Durata Therapeutics International B.V. Spaces Zuidas II, Barbara Strozzilaan 101, 1083 HN Amsterdam, The Netherlands. **Przedstawiciel podmiotu odpowiedzialnego: Angelini Pharma Polska Sp. z o.o.**, ul. Podleśna 83, 05-552 Łazy, Polska, tel. +48 227 028 200, fax +48 227 028 202. **Pozwolenie na dopuszczenie do obrotu:** EU/1/14/986/001. **Kategoria dostępności:** Rpw.

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