

ONCOLOGY

RATIONALE FOR THE USE OF LOW-INTENSITY PHOTODYNAMIC THERAPY IN STAGE I BASAL CELL SKIN CARCINOMA

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ABSTRACT

Background. The paper describes a method of low intensity photodynamic therapy (LIPDT) for the treatment of superficial basal cell skin cancer (SBCSC) stage I using topical application of the photosensitizer Photoditazine gel[®] or intravenous Photoditazine[®] solution. LIPDT is the treatment of choice in medical organizations lacking laser equipment, specialized facilities, or for remote use (in distant areas). It can also be used as an adjunct to other treatment methods.

Aim. To provide a theoretical rationale for the use of a LIPDT device with Photoditazine[®] and Photoditazine gel[®] for the treatment of SBCSC.

Materials and methods. The developed device consists of the following elements: a battery, a control board, a DC-DC voltage converter, a microcontroller, a driver, and an LED matrix. The mechanism of the device is based on the classical PDT method but has several key differences: automatic mode (eliminating the need for manual adjustment), low output radiation power (which prevents oxygen depletion in tumor tissue), and the ability to function under conditions unsuitable for standard PDT.

Conclusion. The proposed LIPDT method for treating stage I SBCSC will reduce treatment costs while maintaining the effectiveness of the existing standard PDT method.

Key words: basal cell skin cancer (BCSC), low-intensity phototherapy therapy (LIPDT), photosensitizer (PS), Photoditazine[®], Photoditazine-gel[®]

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ОБОСНОВАНИЕ ПРИМЕНЕНИЯ СПОСОБА НИЗКОИНТЕНСИВНОЙ ФОТОДИНАМИЧЕСКОЙ ТЕРАПИИ ПРИ БАЗАЛЬНОКЛЕТОЧНОМ РАКЕ КОЖИ I СТАДИИ

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РЕЗЮМЕ

Обоснование. В работе представлено описание методики проведения низкоинтенсивной фотодинамической терапии (НФДТ). При лечении поверхностного базальноклеточного рака кожи (БКРК) I стадии по системе AJCC 8-го пересмотра (2017 г.) оценен результат наружного применения препарата «Фотодитазин-гель»[®] или раствора «Фотодитазин»[®] для внутривенного введения. НФДТ является выбором метода лечения в условиях медицинских организаций, не имеющих лазерного оснащения, специализированного помещения или возможности дистанционного контроля лечения (на отдаленных территориях). Может быть применен в дополнение к другим способам лечения.

Цель. Теоретически обосновать применение аппарата НФДТ с препаратами «Фотодитазин»[®] и «Фотодитазин-гель»[®] для лечения поверхностного БКРК.

Материалы и методы. Разработанный аппарат состоит из следующих элементов: аккумулятор, плата контроля, DC-DC преобразователь напряжения, микроконтроллер, драйвер и светодиодная матрица. Механизм работы аппарата базируется на классическом методе фотодинамической терапии (ФДТ), имея при этом ключевые отличия: автоматический режим (без необходимости ручной настройки), низкую выходную мощность излучения (благодаря чему не происходит истощение кислорода в опухолевой ткани), возможность работы в условиях, не приспособленных для стандартной ФДТ.

Заключение. Способ лечения НФДТ позволит снизить стоимость лечения поверхностного БКРК I стадии, сохранив при этом эффективность уже существующего стандартного метода ФДТ.

Ключевые слова: базальноклеточный рак кожи (БКРК), низкоинтенсивная фотодинамическая терапия (НФДТ), фотосенсибилизатор (ФС), препараты «Фотодитазин»[®], «Фотодитазин-гель»[®]

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INTRODUCTION

In 2023, a total of 76,081 patients with skin malignancies (excluding melanoma) were registered in the Russian Federation with a first-ever diagnosis of a malignant neoplasm, thereby ranking first in cancer detectability. Malignant neoplasms of the breast ranked second (74,008 patients). Between 2013 and 2023, the prevalence of skin malignancies increased by 18.3 % [1]. Photodynamic therapy (PDT) is successfully used to treat various precancerous conditions and malignant neoplasms [2–4].

However, research continues to identify the optimal treatment modality to achieve complete tumor eradication. Reducing treatment intensity is also of great importance, given that pain remains the most common adverse effect [5].

AIM

To provide a theoretical rationale for the use of a LIPDT device with the photosensitizer “Photoditazine”® and “Photoditazine-gel”® for the treatment of superficial basal cell skin carcinoma (SBCSC), which will reduce treatment costs, simplify the procedure, while maintaining the effectiveness of photodynamic therapy.

MATERIALS AND METHODS

The developed device consists of the following components: a battery, a control board, a DC-DC voltage converter, a microcontroller, a driver, and an LED matrix. The operating principle of the device is based on the classical PDT method but has several key differences: an automatic mode (eliminating the need for manual adjustment), low output radiation power (which prevents oxygen depletion in tumor tissue), and the ability to function under conditions unsuitable for standard PDT.

The difference between LIPDT and conventional laser PDT lies in the fact that LIPDT uses an LED emitter based on a single LED or an LED matrix consisting of several LEDs, powered autonomously by a lithium-ion battery (power bank).

The LIPDT method for patients with malignant neoplasms is performed by applying “Photoditazine-gel”® or by intravenous administration of “Photoditazine”®, followed by exposure of the tumor growth zone to light radiation at a wavelength within the photosensitizer absorption range, with an energy fluence of 300 to 600 J/cm². The irradiated field area is selected to match the tumor area, extending 0.5 mm beyond the clinically evident tumor margin.

In practice, “Photoditazine-gel”®, a light penetrator, was used at a concentration of 0.5 % and a volume of 1 mL. This preparation is intended to enhance the penetrating effect of the light energy source used in phototherapy, including photodynamic therapy, under various light exposure conditions. The gel was applied topically to the skin

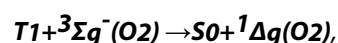
topically 30 minutes prior to light exposure, using 1 mL of gel for every 3–5 cm² of the treated surface. After 30 minutes, the applied gel was removed with a cotton swab moistened with distilled water, followed by light exposure.

The irradiation session is performed using LED emitters that provide a light flux in the 650 nm wavelength range.

LIPDT is performed using an LED emitter with a radiation wavelength of 650 nm, powered autonomously by a portable power bank. The size of the LED emitter is selected according to the irradiated field area, matching the tumor area circumferentially, extending 5 mm beyond the clinically evident tumor margin. The LED emitter (matrix) is fixed to the tumor surface using standard fixation methods (adhesive tape, securing bandage), allowing the patient to move freely and perform self-care during the session.

RESULTS AND DISCUSSION

The developed LIPDT method is based on the photodynamic effect on malignant cells following the topical application or systemic administration of photosensitizers (PS), which are substances that increase the sensitivity of tumor tissues to light. The LIPDT procedure can be divided into the following stages: administration of the PS into the body; preferential accumulation of the PS in the tumor area; excitation of the PS at the target area by LIPDT, resulting in the formation of reactive compounds; cell death; tissue destruction; elimination of the PS; and wound healing. The preferential accumulation of the PS in the tumor is a specific property of the photosensitizers used and is based on differences in oxidative processes between cancer cells and healthy tissues. Excitation of the PS occurs when the tumor area containing the accumulated PS is irradiated with light at a wavelength corresponding to the optical absorption band of the PS. The excitation energy of the PS is transferred to oxygen molecules present in the tumor cells, converting them from the ground triplet quantum state to the singlet state, resulting in the formation of reactive oxygen species (ROS). Singlet oxygen (¹O₂) is highly reactive and oxidizes cell molecules, leading to cell death and necrosis of tumor tissue. This constitutes the essence of both PDT and LIPDT. The photodynamic therapy process can be described by the following reaction:



where *T1* – PS in the excited triplet state, into which it transitions as a result of light exposure within the absorption band; ³Σ_g[−](O₂) – molecular oxygen in the ground triplet state in the cells of the tumor tissue; *S0* – PS in the ground singlet state, into which it transitions after interacting with oxygen; ¹Δ_g(O₂) – molecular oxygen in the excited active singlet state after interacting with the PS.

Singlet oxygen ¹Δ_g(O₂), having paired electrons on one orbital and a vacant second orbital, readily interacts

with electron-rich molecules [6]. The photosensitizer itself causes damage to specific parts of cells, inducing apoptosis, necroptosis, autophagy, and paraptosis, which can occur as a result of damage to mitochondria, lysosomes, and the endoplasmic reticulum [7]. An important factor for overall treatment effectiveness is the balance between these processes [8].

Under intense light irradiation, the primary PDT effect occurs at the onset of laser exposure, when the maximum number of oxygen molecules participates in the generation of singlet states. Subsequently, both the intensive consumption of oxygen in its active forms and the slow diffusion of free oxygen into the photoactive zone inevitably lead to a decrease in oxygen concentration around the photosensitizing molecules. This diminishes the photodynamic effect and, consequently, reduces treatment effectiveness. Studies conducted in several works, such as [4], have confirmed that singlet oxygen generation during PDT can deplete photochemical oxygen within the tumor, resulting in local hypoxia and reduced treatment effectiveness.

The rate of oxygen supply to the tissue after its consumption during the photodynamic reaction is relatively low. In practice, the time constant for tissue reoxygenation varies across different tissues, ranging approximately from two to eight seconds [9].

In this regard, solving the problem of tissue deoxygenation within the irradiated area is possible through reducing the power density (irradiance) of the light exposure, while simultaneously extending the exposure time to achieve the required total energy fluence. Addressing the issue of tissue deoxygenation during PDT in the irradiated area may be achieved at deliberately low light intensity levels, at which the replenishment of the tissue oxygen content is maintained at an adequate level due to the low rate of its conversion and the extended time required for its transition to the active singlet state.

The LIPDT method simplifies the PDT procedure, reduces the risk of thermal injury to the treated area, and eliminates the risk of damage to the optical fiber cable used for laser PDT. Ongoing physician monitoring of the current state of the treated area allows the therapy to be administered on an outpatient basis using a portable power supply, as well as remote monitoring. Unlike PDT using laser radiation sources, this method does not require specialized rooms (with special wall coverings to prevent light reflection) or eyewear with optical filters for protection.

Due to the low radiation intensity of LEDs compared to lasers, LIPDT requires a longer treatment interval following photosensitizer application. However, the use of LIPDT reduces the risk of thermal injury to surrounding tissues.

The rate and extent of preferential PS accumulation in tumors compared to surrounding tissues can be 3–10 times higher, depending on the tumor type and the PS employed.

Second-generation photosensitizers, commonly used in medicine, such as "Photoditazine"[®] for intravenous

administration and "Photoditazine-gel"[®] for topical application, developed by Russian companies, belong to the chlorin family. "Photoditazine"[®] is the di-N-methylglucamine salt of chlorin E6 and a derivative of chlorophyll *a* [10]. "Photoditazine"[®] is produced from the biomass of the microalgae *Spirulina platensis*, which is used to extract chlorin E6.

"Photoditazine"[®] exhibits high photodynamic activity for singlet oxygen generation and has several advantages compared to other second-generation photosensitizers. These advantages make it a preferred choice for PDT, as they help to minimize damaging effects on healthy tissues and organs, as well as skin damage caused by light exposure [11].

The LED emitter power and irradiation time are set to provide a total energy fluence of 300–600 J/cm², depending on the specific requirements. Figure 1 shows the spectral correlation between the LED emission and "Photoditazine"[®].

A key parameter for calculating the light dose is the energy fluence, which is the total light energy delivered to a unit area of irradiated pathological tissue in the tumor area.

The irradiation dose is calculated using the following formula:

$$E = P \cdot t, \quad (1)$$

where **E** – total irradiation energy (J), **P** – output power of the LED emitter (W), **t** – irradiation time (exposure), seconds.

To calculate the energy fluence (**Q**), the total irradiation energy must be divided by the area (**S**) of the light spot on the irradiated surface

$$Q = E / S = P \cdot t / S. \quad (2)$$

Accordingly, the following formula can be used to calculate the irradiation time (**t** in seconds):

$$t = Q \cdot S / P, \quad (3)$$

where **Q** – energy fluence (dose), J/cm², **S** – area of the light spot on the irradiated surface, cm², **P** – output power of the LED emitter, W.

Table 1 shows the correlation between the irradiation dose of light, the irradiation time, and the emitter power, assuming that the light spot area matches the tumor area. To determine the key parameter, the energy fluence, the total irradiation energy (**E** in J) from the table must be divided by the surface area of the irradiated tissue in the tumor area.

If the size of the light spot does not match the tumor size, two scenarios are possible: the tumor is smaller than the light spot size, or the tumor is larger than the light spot size.

When the tumor size is smaller than the light spot size, the irradiation dose delivered to the tumor must be calculated using the following formula:

$$E_{\text{tumor}} = E_{\text{total}} * S_{\text{light}} / S_{\text{tumor}} \quad (4)$$

where E_{tumor} – irradiation dose received by a particular area of the tumor, E_{total} – total irradiation dose from the table, S_{light} – light spot area, S_{tumor} – tumor area.

In this case, the energy fluence (dose density) Q can be calculated using the following formula:

$$Q = E_{\text{tumor}} / S_{\text{tumor}} \quad (5)$$

or:

$$Q = E_{\text{total}} / S_{\text{light}} \quad (6)$$

If the light spot size is smaller than the tumor size, the dose can only be calculated for the part of the tumor that has been irradiated, using Formula 6. The light spot can be moved across the tumor for subsequent irradiation

sessions, resulting in the summation of irradiation doses: $E_{\text{total}} = E_{\text{total1}} + E_{\text{total2}}$. In this case, the average dose per unit area can be calculated as $E_{\text{total}} / S_{\text{light}} = (E_{\text{total1}} + E_{\text{total2}}) / (S_{\text{light1}} + S_{\text{light2}})$.

Example 1. For a tumor size of up to 1 cm ($E = 200$ J/cm²) and an LED power of 0.05 W, the power density (P) is calculated as follows:

$$0.05 \text{ W} / 0.78 \text{ cm}^2 = 0.064 \text{ W/cm}^2$$

$$\text{W/cm}^2 = 3125 \text{ sec. or } 52 \text{ min.}$$

Calculation of the LIPDT procedure time (t) as a function of the diameter (D) and area (S) of the light spot at a given energy fluence (Q) and an LED power of 0.07 W (Table 2).

The active ingredient in "Photoditazine"[®] (chlorin E6 dimeglumine) has a selective accumulation in tumor cells. When the tumor area is subsequently irradiated with monochromatic light at a wavelength of 650–670 nm,

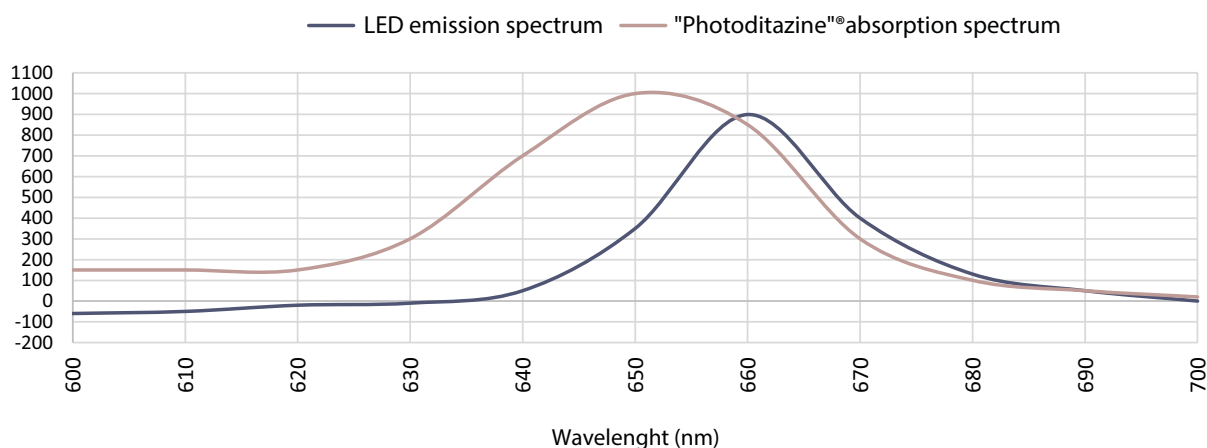


FIG. 1.
Spectral correlation between the LED emission spectrum and the "Photoditazine"[®] absorption spectrum

TABLE 1

DEPENDENCE OF THE IRRADIATION DOSE ON EXPOSURE TIME AND EMITTER POWER WHEN THE LIGHT SPOT AREA MATCHES THE TUMOR AREA

Power, mW →		5	10	20	30	40	50	60	70	80	90	100
t, sec. ↓	t, min.	Irradiation dose, J										
60	1	0.3	0.6	1.2	1.8	2.4	3	3.6	4.2	4.8	5.4	6
300	5	1.5	3	6	9	12	15	18	21	24	27	30
600	10	3	6	12	18	24	30	36	42	48	54	60
900	15	4.5	9	18	27	36	45	54	63	72	81	90
1200	20	6	12	24	36	48	60	72	84	96	108	120
1500	25	7.5	15	30	45	60	75	90	105	120	135	150
1800	30	9	18	36	54	72	90	108	126	144	162	180
2100	35	10.5	21	42	63	84	105	126	147	168	189	210
2400	40	12	24	48	72	96	120	144	168	192	216	240

a photosensitization effect occurs. This effect involves the generation of reactive oxygen species in the tumor tissue, including singlet oxygen and other free radicals. These species cause structural and functional changes to tumor cells, ultimately leading to their death.

The duration of the treatment after the application of the photosensitizer is determined by the calculated dose (Table 2).

The light-emitting device based on an LED matrix and power supply is schematically illustrated in Figure 2. A lithium-ion battery (1) is used to power the device. A battery charge/discharge controller (2) is used for charging and monitoring the battery. A DC-DC voltage converter (3) increases and maintains the voltage from the battery at a constant level. A microcontroller (4) generates a PWM signal, which is sent to a driver (5) to regulate the operation of the emitter (LED matrix) (8). The emitter operating mode is set using control buttons (7) and the status is displayed on the display (6).

The light-emitting matrix, comprising at least four LEDs, is mounted on a heat sink and enclosed within a plastic casing (Fig. 3). The casing has lugs for securing the emitter to the patient's body via elastic straps

or adhesive tape. On the external side of the casing, there are openings for heat dissipation from the heat sink and an opening for the LED power cable.

The light-emitting matrix is connected to a power supply and control unit through a flexible cable. The external view of the unit is shown in Figure 4. The driver stabilizes the current through the LEDs and, under control of the microcontroller, delivers current pulses to the LEDs at a specified frequency. The microcontroller controls the LED emission modes by sending control signals to the driver using pulse-width modulation (PWM), enabling the LEDs to emit in a pulsed mode with adjustable frequency. This allows treatment procedures to be performed in an optimal mode, reducing the thermal load on the irradiated area and allowing the selection of irradiation frequency and power for affected areas according to the most effective therapeutic protocol.

The power supply unit has control buttons for setting the emission mode, and parameters displayed on an integrated screen (Fig. 4).

Therefore, the proposed device allows for the treatment of skin neoplasms using the LIPDT method, resulting in good cosmetic outcomes.

TABLE 2
CALCULATION OF THE LIPDT PROCEDURE TIME (t)

Energy fluence, J/cm ²	100	200	300	400	500	600
Light spot diameter, cm	Irradiation time, min.					
0.5	7	13	20	26	33	39
0.6	9	19	28	38	47	57
0.7	13	26	38	51	64	77
0.8	17	33	50	67	84	100
0.9	21	42	64	85	106	127
1.0	26	52	79	105	131	157
1.1	32	63	95	127	158	190
1.2	38	75	113	151	188	226
1.3	44	88	133	177	221	265
1.4	51	103	154	205	256	308
1.5	59	118	177	236	294	353
1.6	67	134	201	268	335	402
1.7	76	151	227	302	378	454
1.8	85	170	254	339	424	509
1.9	94	189	283	378	472	567
2.0	105	209	314	419	523	628

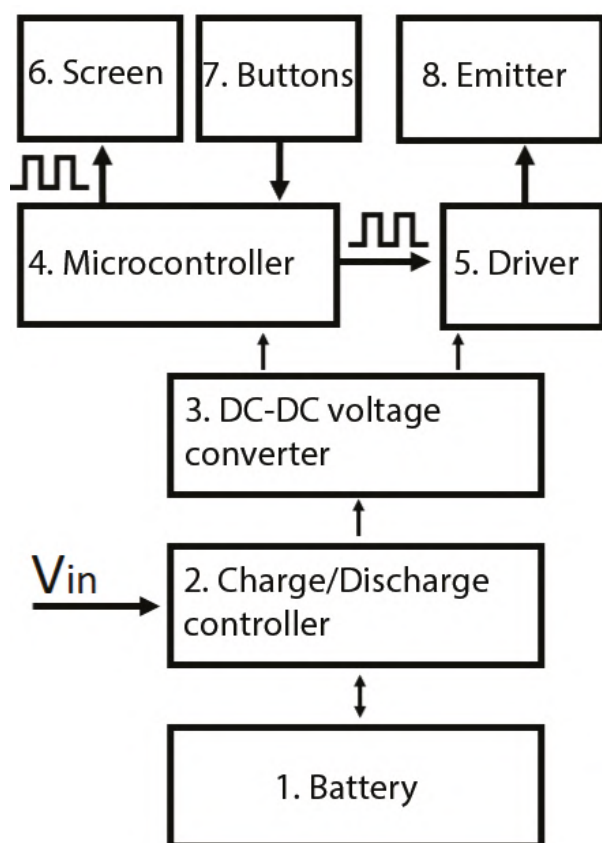


FIG. 2.
Block diagram of a light-emitting device based on an LED matrix and a power supply

CONCLUSION

The developed new treatment method (LIPDT) will reduce the cost of treatment for patients with stage I BCSC by simplifying the procedure while maintaining comparable effectiveness to conventional methods. This method does not require the use of specialized rooms or reflective-coated walls.

In medical organizations that lack laser equipment, LIPDT can serve as the treatment of choice. It can also be used in conjunction with PDT for cases of multiple primary tumors, as well as for remote monitoring in hospice settings. Simultaneous application of multiple LEDs can be utilized with this method.

Conflicts of interest

The authors declare no conflicts of interest.

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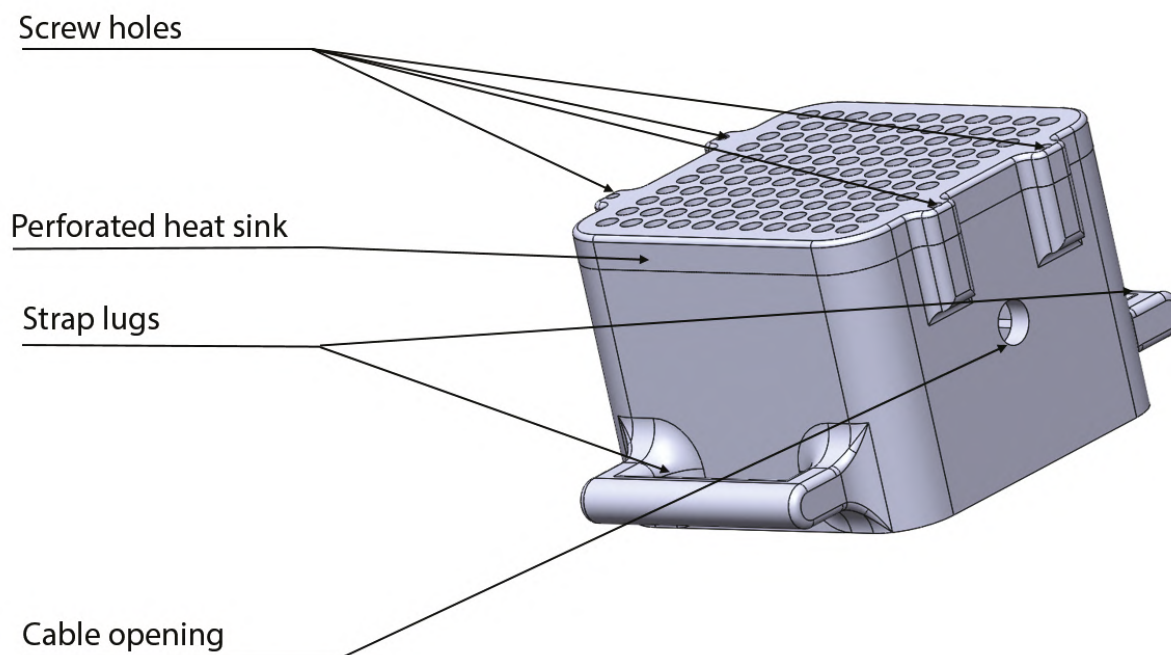


FIG. 3.
Light-emitting matrix based on LEDs for LIPDT

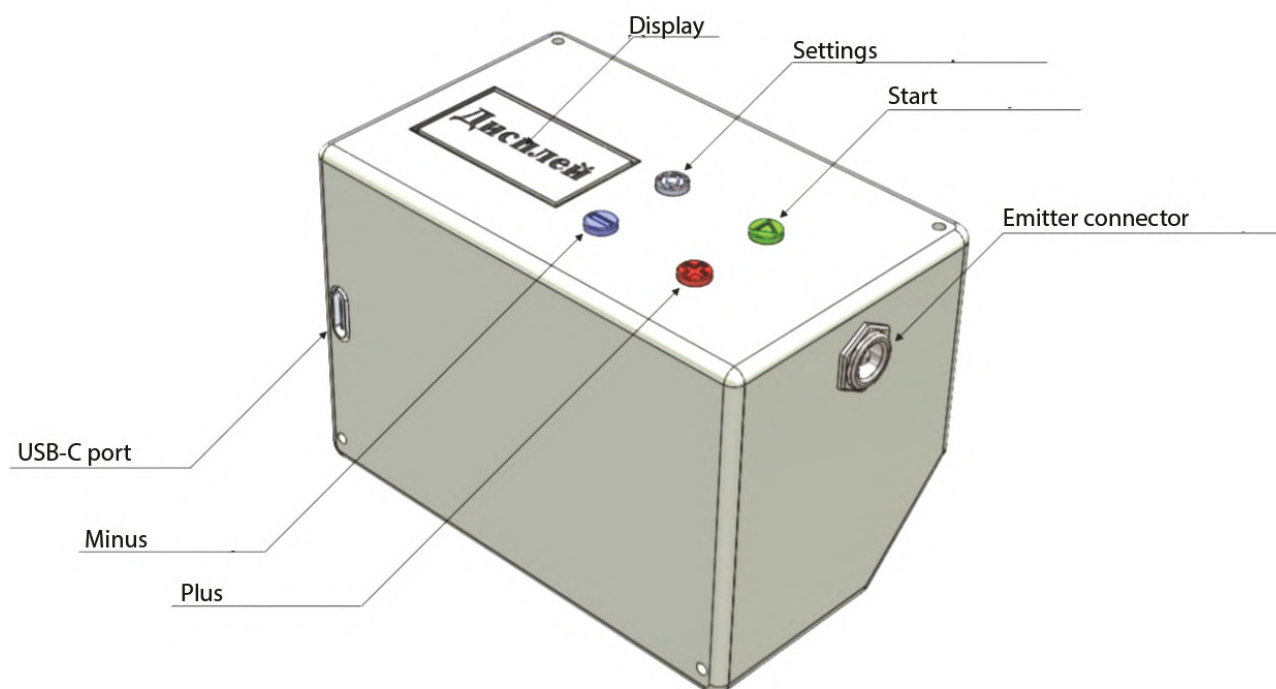


FIG. 4.
Power supply and control unit for the LED-based emitter for LIPDT

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