



Diagnosis and Management of Non-Healing Arterial and Venous Wounds

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VENOUS ULCERATION

- Due to sustained venous hypertension from CVI
- Normally venous leg pressure decreases with exercise and rest
- If valves in perforating veins are incompetent, venous leg pressure will increase which can create a VLU



DIRECT RISK FACTORS FOR VLUs

- Varicose veins
- Deep vein thrombosis
- Chronic venous insufficiency
- Poor calf muscle function
- Arterio-venous fistulae
- Obesity
- History of leg fracture



VLU EXAMINATION

- 95% OF VLUs are found around the malleoli
- Ulcer bed is usually covered with stringy slough, granulation and usually with copious of amounts of drainage (weeping legs)



VLU's

- Pitting edema usually worse at the end of the day
- Hemosiderin staining



VLUs

- Lipodermatosclerosis with areas of atrophie blanche (inverted champagne bottle)



MANAGEMENT OF VLUs – COMPRESSION!

- Compression is the mainstay of venous ulcer management
- Compress only if ABIs are >0.7 in non-diabetics
- Graded compression, with greatest pressure (about 40 mm Hg) at the ankle, tapering to 18 mm Hg below the knee
- Remove compression at the first sign of trouble!



MANAGEMENT OF VLUs – SHARP DEBRIDEMENT!



BIOFILM AND INFECTED VLU_s

- Often becomes infected with S.aureus, P.aeruginosa and Group A beta-hemolytic Strep (S.pyogenes)
- Treat empirically until cultures are in (2 weeks)
- Avoid topical ABTs
- ADDRESS THE UNDERLYING BIOFILM IN THE WOUND WITH YOUR HYPERSPECTRAL IMAGING DEVICE!!



INFECTED VLUs – Ouch!



ARTERIAL ULCERATION

- Due to reduced arterial oxygenated blood to the lower limb
- MCC is atherosclerotic disease of med. and large arteries
- DM is a big culprit in the US and directly causes calcification of the arterial vasculature. High risk of limb loss.
- Smoking, HTN, HLP, obesity are all risk factors for PVD
- Very painful due to ischemia



ARTERIAL ULCERS

- Usually over toes, heels and bony prominences of the foot
- “Punched out” appearance
- Dusky erythema, cool to touch, hairless
- Gangrene
- Decreased or absent pedal pulses



Arterial Ulcers (if you do nothing!)



ABI Index

Index	Signs and symptoms	Severity of disease	Action
≥0.7-1	Mild intermittent claudication, or no symptoms	Mild arterial disease	Reduce risk factors and change lifestyle: stop smoking, maintain weight, exercise regularly, consider antiplatelet agent
0.7-0.5	Varying degrees of intermittent claudication	Mild to moderate arterial disease	As for index ≥0.7-1, plus referral to outpatient vascular specialist and possible arterial imaging (duplex scan and/or angiogram)
0.5-0.3	Severe intermittent claudication and rest pain	Severe arterial disease	As for index ≥0.7-1, plus urgent referral to vascular specialist and possible arterial imaging (duplex scan and/or angiogram)
≤ 0.3 or ankle systolic pressure <50 mm Hg	Critical ischaemia (rest pain >2 weeks) with or without tissue loss (ulcer, gangrene)	Severe arterial disease; risk of losing limb	Urgent referral to vascular emergency on-call team and possible surgical or radiological intervention



MANAGEMENT OF ARTERIAL ULCERS

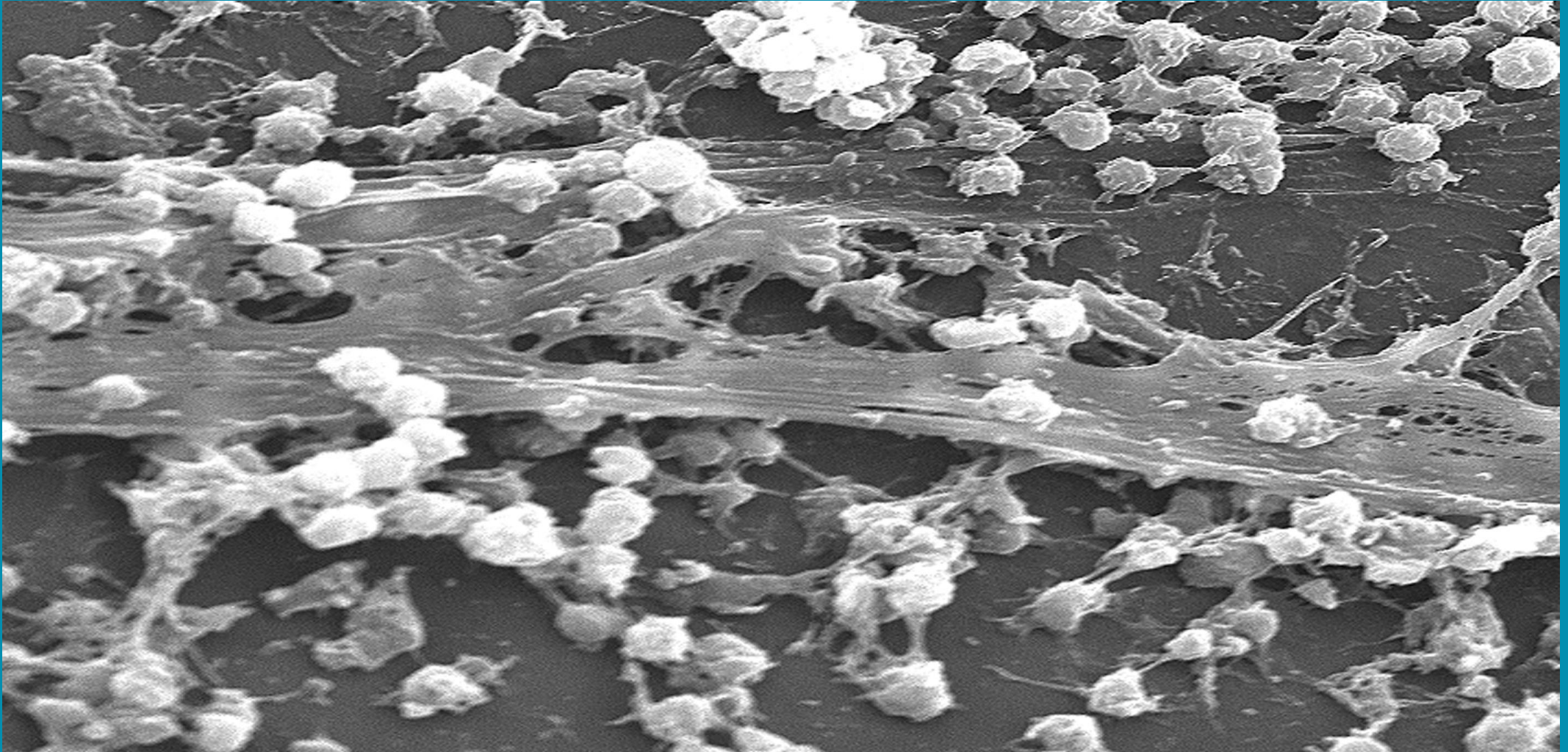
- Increase peripheral blood flow (bypass vs angioplasty)
- Cut out the “lifestyle” risk factors
- Address the ischemic pain with opioids if necessary
- Infection is also a concern here. **BIOFILM!!**
- Debride wound as needed. This may lead to worsening of the wound if done before reperfusion intervention
- Be aware that a mixed etiology is common: combo of venous and arterial disease will limit degree of compression



BIOFILM IN NON-HEALING WOUNDS

- Biofilm is a collection of microbes, consisting of bacteria and fungi.
- A polysaccharide matrix, a false ECM!
- **ONLY VISIBLE BY ELECTRON MICROSCOPE** until recently!!
- This film forms a protective barrier for the microorganisms and prevent antibodies, and antibiotics, from reaching them.
- What can we do????
- A good option is **CADEXOMER IODINE**, also known as **IODOSORB GEL!!**

 **DANGER!! BIOFILM AHEAD!** 



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MolecuLight i:X Device

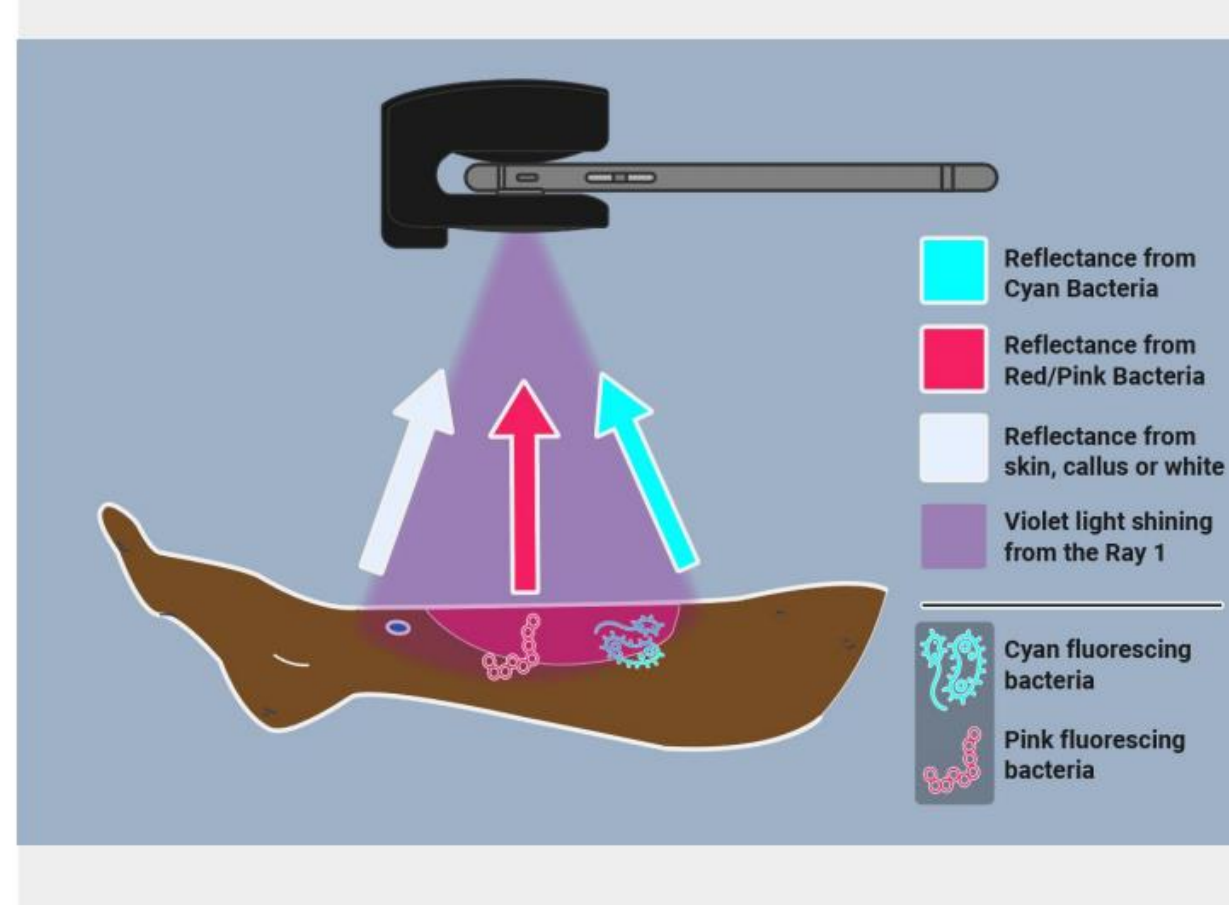


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HOW DOES IT WORK?

Fluoro imaging shines a **violet light** onto an area which will then reflect off of some specific surfaces based on the color.

Specific bacteria types and loads will reflect different **colors and brightness** levels which can then be seen using a **fluorogram**.

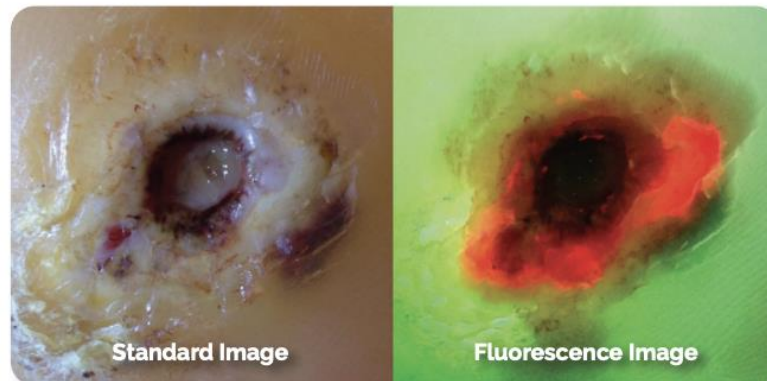


HARNESSING THE POWER OF VIOLET LIGHT!

- Shining a safe violet excitation light (405 nm) causes wound components (skin, slough, blood, bacteria, etc.) to fluoresce in different colors.
- Green fluorescence from the skin depicts anatomical context.
- Red and cyan fluorescence are associated with regions of bacterial load $\geq 10^4$ CFU/g which is typically moderately-heavy growth.

Red Fluorescence

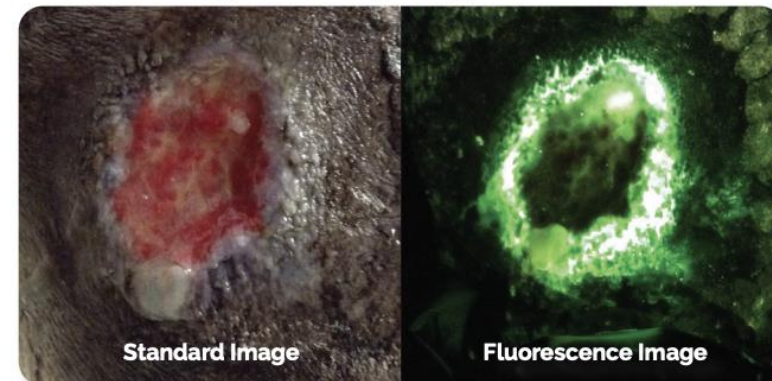
The majority of bacteria fluoresce **red** under violet light²⁻⁵.



Microbiology: 2.3×10^8 CFU/g, including *Staphylococcus hominis*, *Campylobacter ureolyticus* etc.

Cyan Fluorescence

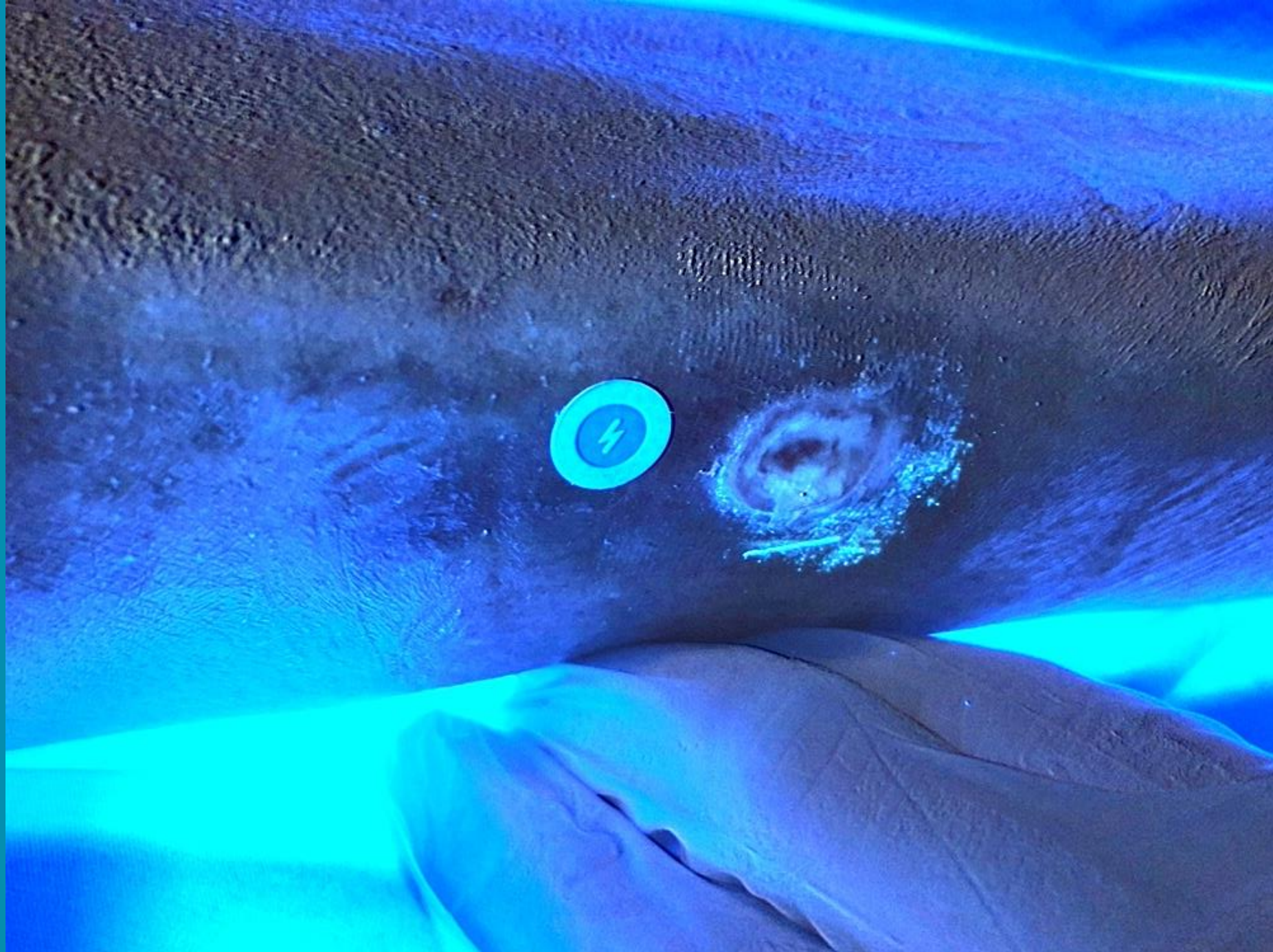
Pseudomonas aeruginosa fluoresces **cyan** (blue/green with a glowing white center)^{2,4}.



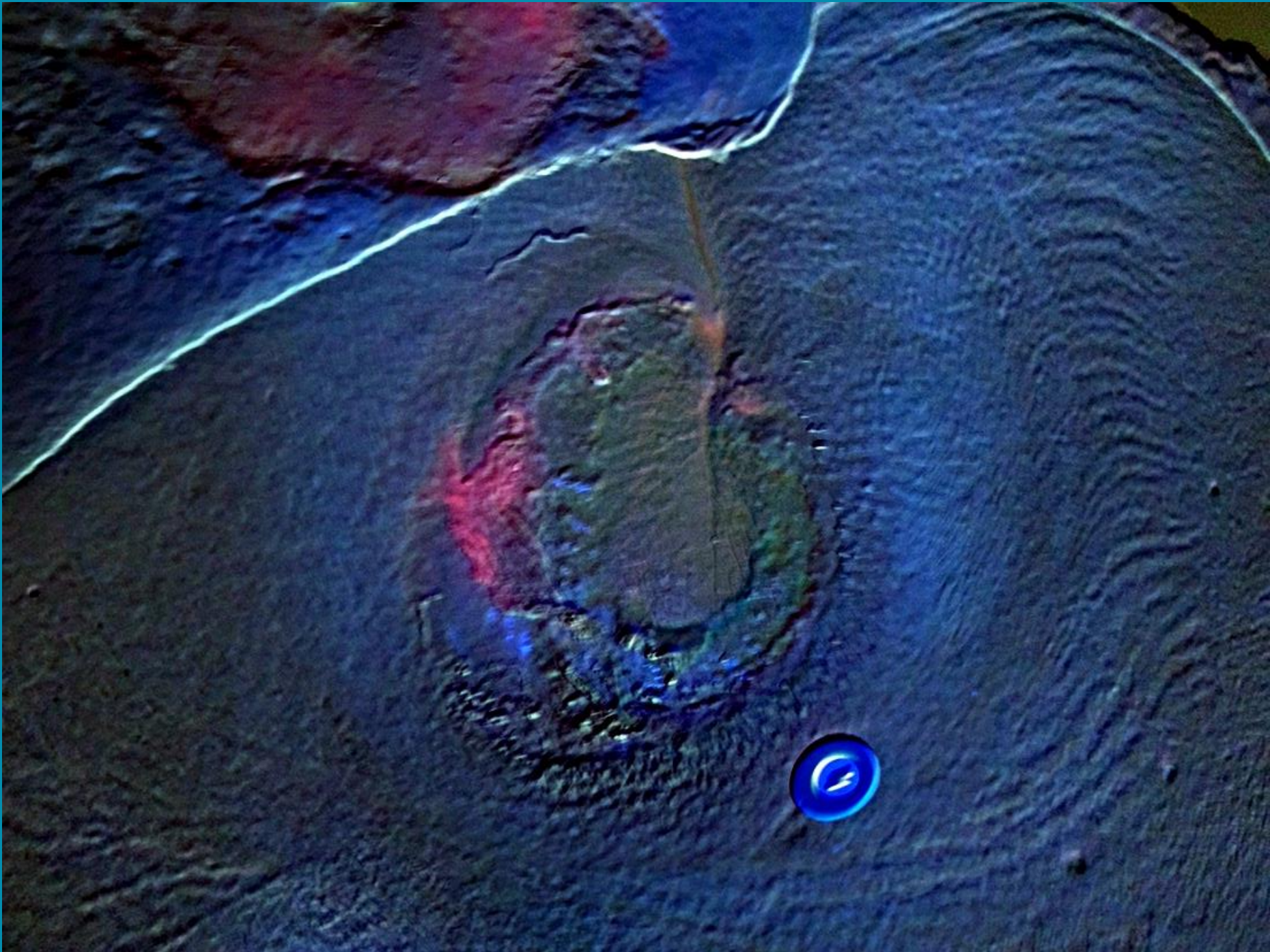
Microbiology: 1.5×10^7 CFU/g, primarily *Pseudomonas aeruginosa*.



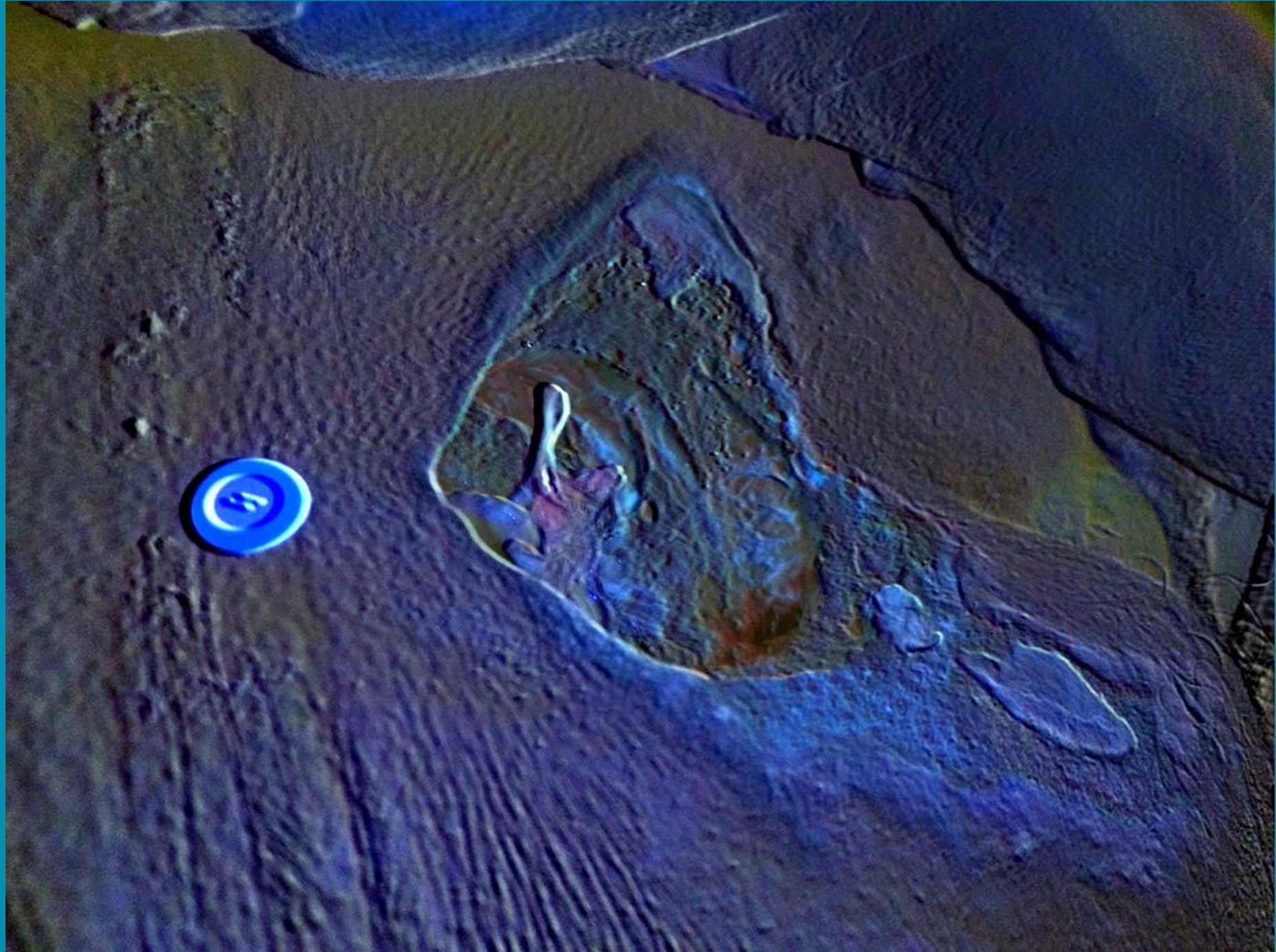
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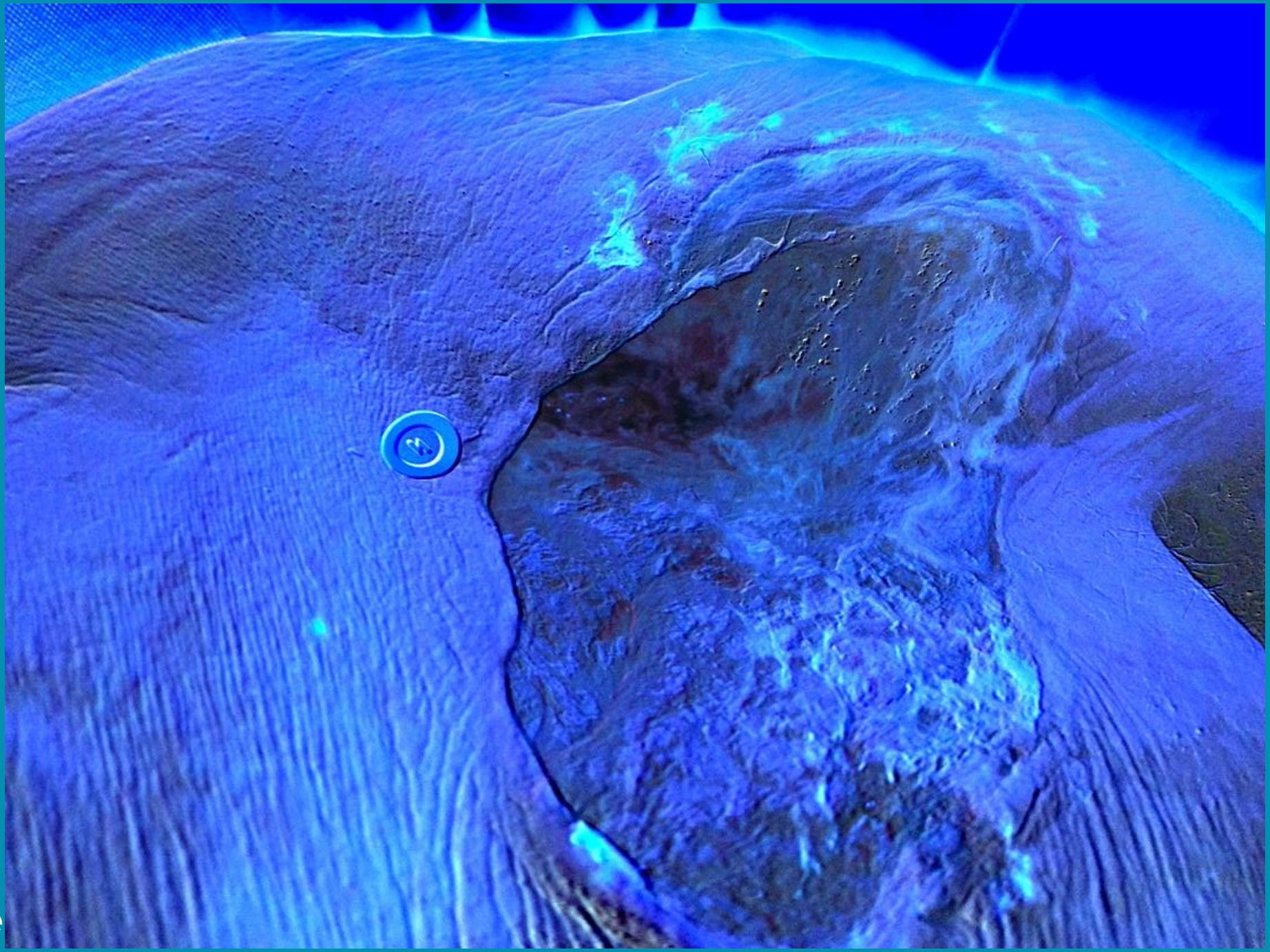
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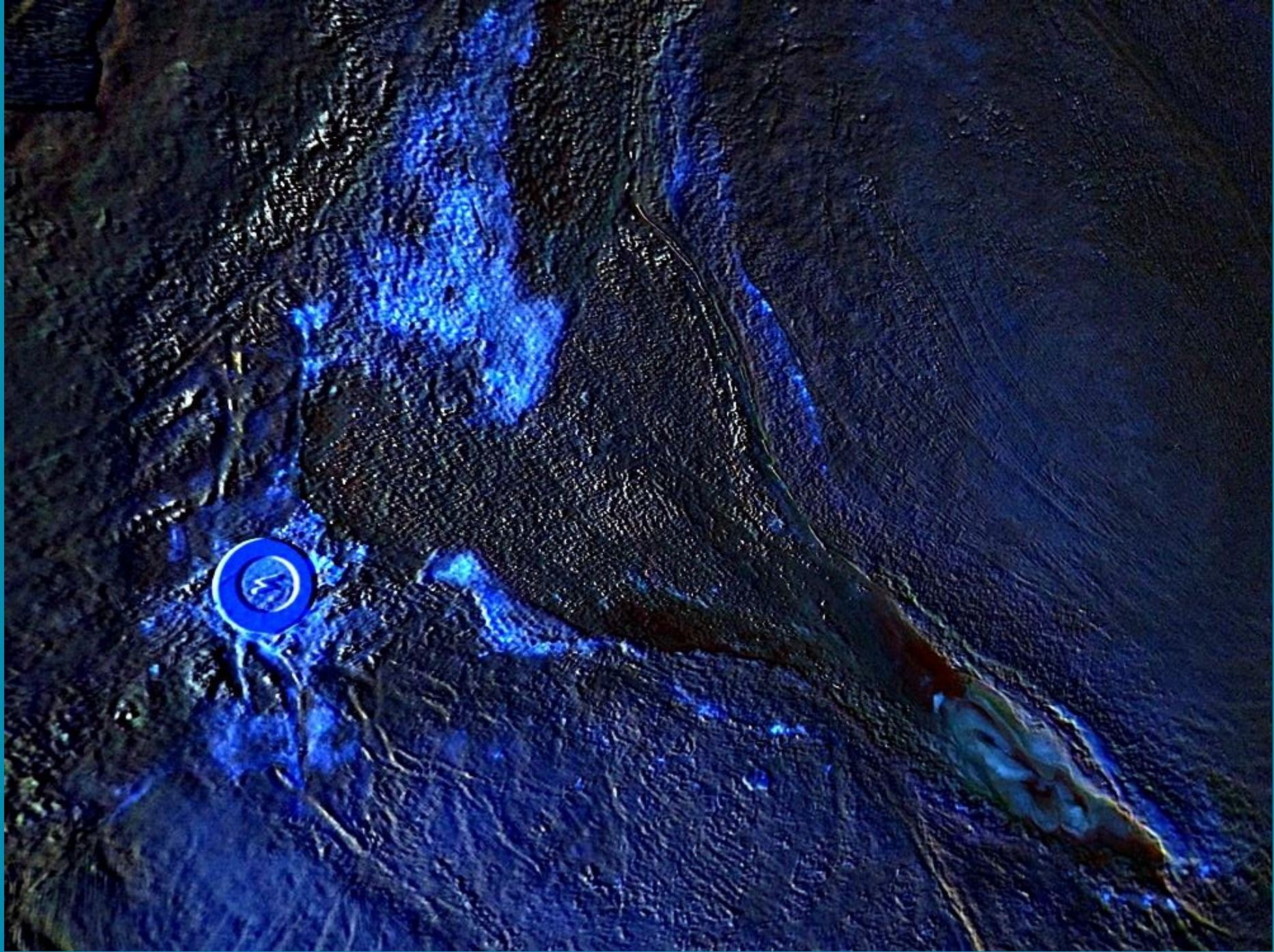
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