Lipid peroxidation of cellular membranes following photodynamic therapy with photosensitizers: sulfoxaporphyrin, dithiaporphyrin and hematoporphyrin derivative.

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Background. Photodynamic therapy (PDT) is known method of anticancer treatment involving application of photosensitizer and subsequent irradiation with light of appropriate wavelength. Mechanisms of PDT action are not completely elucidated, especially when new photosensitizers are applied. Objectives. Aim of study was checking whether new compounds in terms of PDT impair cellular membranes, thus facilitating cell death. Material and methods. Sulfoxaporphyrin (OXA) and dithiaporphyrin (DTP) were injected intraperitoneally to BALB/c mice transplanted with fibrosarcoma BFSI1 in doses 2.5, 5.0, 7.5 mg/kg in 0.9% NaCl. After 24 hours tumors were irradiated at total doses 50 or 100 J/sq cm and wavelength 670 nm (OXA), 695 nm (DTP) and 627 nm (HpD). Following 30 minutes, 6 and 48 hours observation mice were sacrified, tumors excised, and processed for lipid peroxidation (LP). LP was spectrophotometrically measured using kits for malonyldialdehyde (MDA) and 4-hydroxyalkenes (4HNA). LP was given in μM MDA+4 HNA/g of tissue. Results. Level of LP for particular photosensitizer did not very significantly. LP showed to be photodynamic dose-dependent (photosensitizer × light doses). The highest levels of LP occurred after 6 hours from light application at 100 J/sq cm (compound dose was 7.5 mg/kg). In such conditions, OXA-PDT resulted in 998.15, DTP-PDT in 899.04 and HpD-PDT in 1137.36 μM/g, whereas in controls it did not exceed 73.0 μM/g. At latest time point (48 hrs) these values were 10 times lower. Conclusions. All chemicals impaired cellular membranes via lipid peroxidation. We presume that mechanism of action of new photosensitizers on cellular membranes in tumor is similar to that of HpD and increase in LP depends mainly on photodynamic doses used in study.