REVIEW ARTICLE

Radiation Cataractogenesis: The Progression of Our Understanding and Its Clinical Consequences

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ABSTRACT

In the high-volume and increasingly complex world of image-guided therapy and medical imaging, awareness of the potential risks secondary to occupational radiation exposure in medical professionals needs greater focus. One of these risks is radiation-induced cataracts, a recently recognized entity, which may impact the physician's professional proficiency, quality of life, and career span. This review article aims to explain the pathogenesis of radiation-induced cataracts, exploring emerging evidence regarding their development. It also explores the existing monitoring and protection measures available to protect against such radiation-induced pathologic conditions.

ABBREVIATIONS

ICRP = International Commission on Radiological Protection, O'CLOC = occupational cataracts and lens opacities in interventional cardiology, OR = odds ratio, PSCC = posterior subcapsular cataract

Radiation-induced cataracts secondary to occupational exposure represent a recently recognized entity. In the rapidly expanding world of medical imaging and image-guided therapy, awareness about the potential radiation risks to medical professionals has to be emphasized. Knowledge and constant reinforcement of the basic radiation protection principles are required to decrease unnecessary radiation exposure. Therefore, the present article explores our development of understanding of the pathogenesis of radiation-induced cataracts and its close link with radiation-induced oncogenesis. This is followed by discussion of existing monitoring and protection measures available to protect against such radiation-induced pathologic conditions.

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MATERIALS AND METHODS

We obtained anatomic pathology specimens of human lens with posterior chamber cataracts (Figs 1–4). A systematic literature search was performed by the authors using the PubMed database (US National Library of Medicine, National Institutes of Health) and the initial terms "radiation-induced cataracts" and "lens occupational radiation monitoring and protection." Any further searches were more specific to source information regarding radiation-induced oncologic pathologic conditions of relevance to the scope of this document. Inclusion criteria for initial literature searches were broad and as follows: full-text articles published in English between the years 1950 and 2016 with the subject matter of radiation-induced cataract pathogenesis, lens dose monitoring, or radiation protection strategies. The exclusion criteria were formed from the converse of the inclusion criteria. By reading the titles or abstracts, the same authors excluded studies not fulfilling the parameters set by the inclusion and exclusion criteria. Each remaining article was reviewed, and relevant information was extracted if in congruence with the scope of the paper. This information is presented as part of the paper's Results and Discussion. This search strategy is presented in Figure 5.

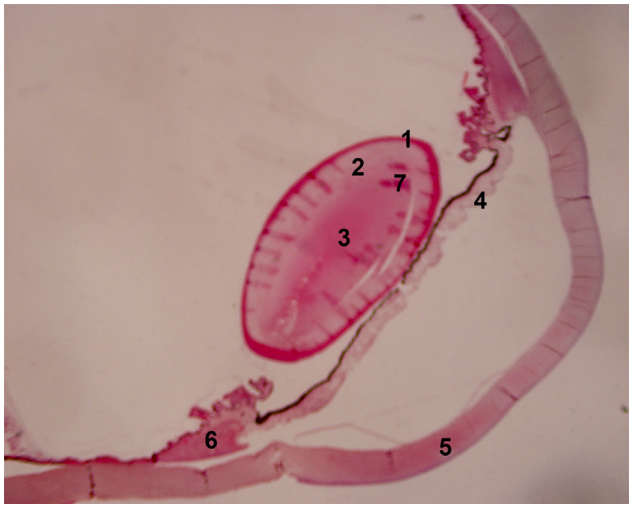

Figure 1. Normal lens showing (*1*) lens capsule, (*2*) peripheral lens fiber, (*3*) nucleus with higher concentration of lens fibers, (*4*) iris, (*5*) cornea, (*6*) ciliary body, and (*7*) artifact.

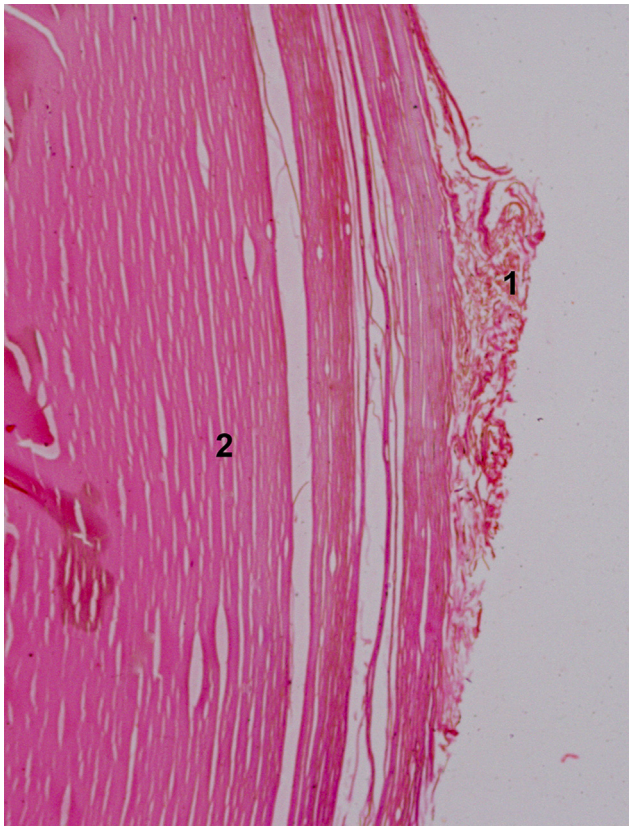

Figure 2. PSCC shows (*1*) the presence of a layer of epithelial cells under the posterior capsule, which have migrated from the equatorial cells. (*2*) The lens fibers still maintain some normal appearance.

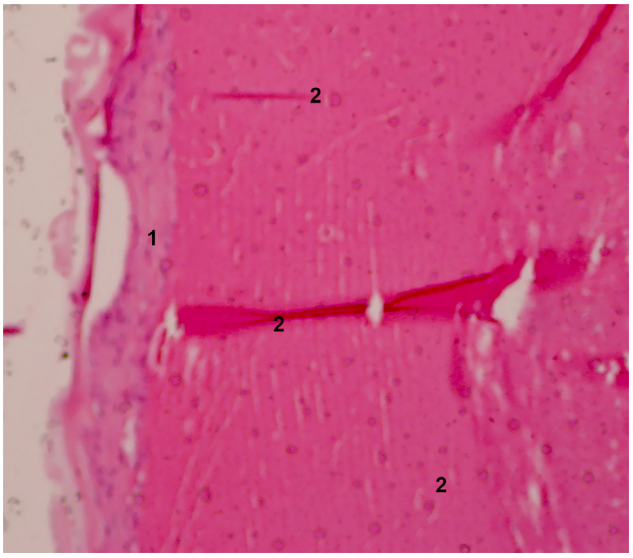

Figure 3. PSCC with (*1*) epithelial cells under the posterior lens capsule that have migrated from the equator or lens bow cells and (*2*) artifacts.

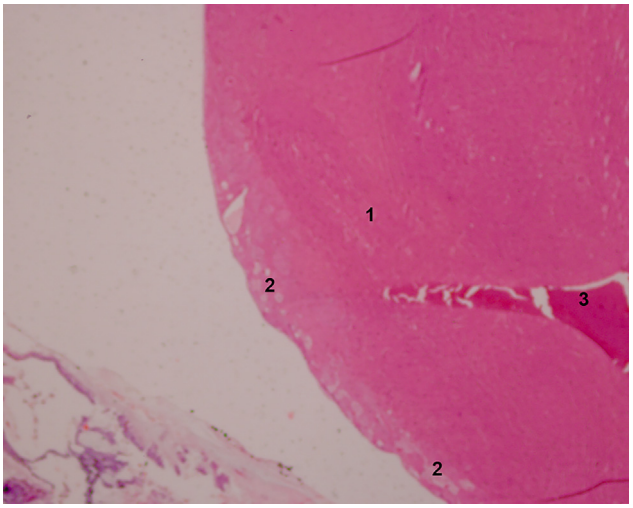

Figure 4. Nuclear cataract showing (*1*) lens fibers that have lost their concentric lamination, giving rise to homogenous eosinophilic appearance; (*2*) separation of lens fibers during slide processing; and (*3*) artifact.

RESULTS AND DISCUSSION

Pathogenesis of Radiation-Induced Cataracts

Modern cataract surgery uses an emulsification process to minimize incision size, so no anatomic specimens are obtained. After extensive searching, we found anatomic pathologic specimens of the human lens in a pathology museum. The unique structure of the lens is fundamental to its refraction capabilities, and is the basis for its high radiation sensitivity and the unique pathology of cataracts (1) (**Fig 1**). The development of cataracts results from the opacification of the lens (1). It is the main cause of blindness worldwide, and the second most common reason for visual impairment after uncorrected refractive errors (2). Age-related "senile" cataracts are the most common type of cataracts (3).

Cataracts are the classified anatomically into nuclear, cortical, and posterior subcapsular subtypes (1). Nuclear and cortical cataracts develop from pathologic changes

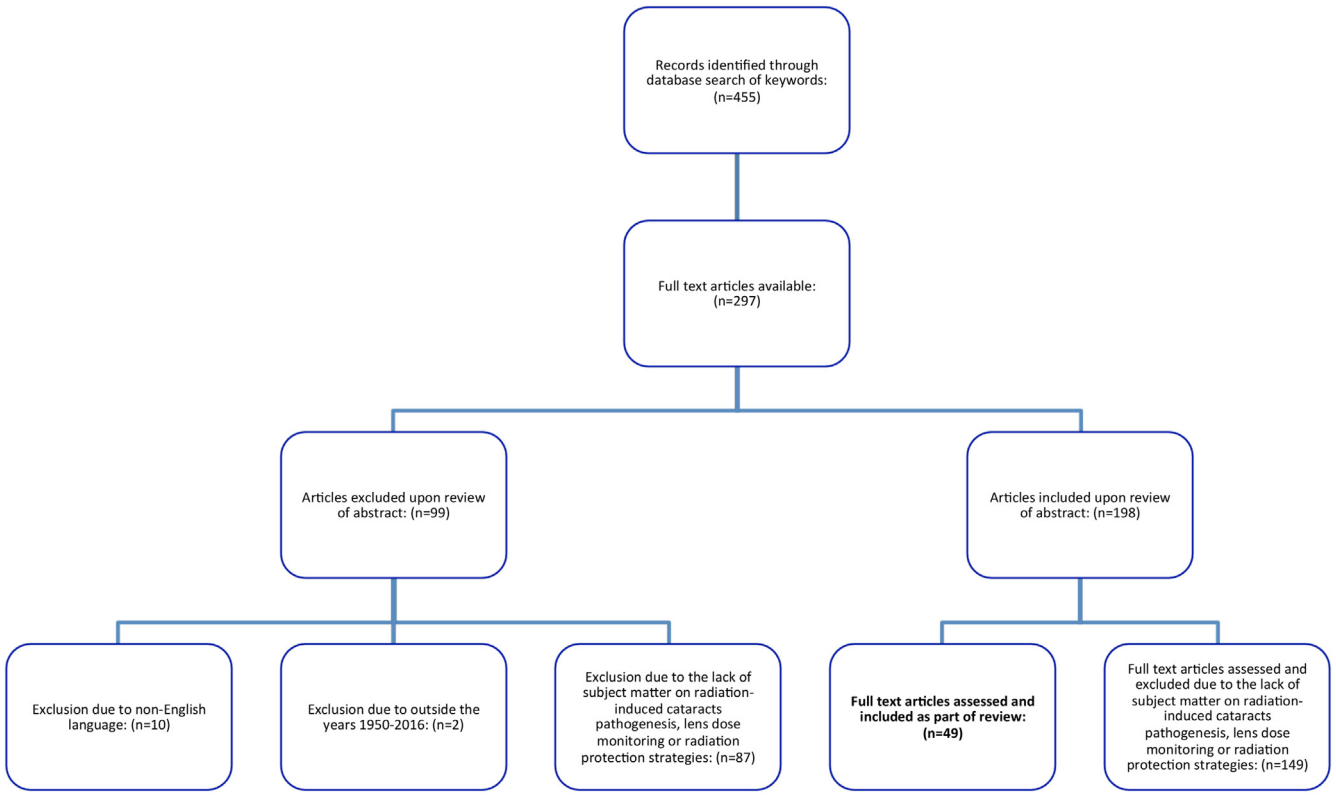

Figure 5. Search strategy.

within the lens fiber cells, whereas posterior subcapsular cataracts (PSCCs) are associated with abnormalities at the germinative zone of the lens (1,4) (**Figs 2–4**). PSCCs are the cataracts most commonly associated with ionizing radiation exposure (2), followed by cortical cataracts (5). However, other contributive factors to PSCC development include lack of endogenous antioxidants and steroid use (6,7).

Cataract pathogenesis is not fully understood and is most likely multifactorial, involving numerous genetic and environmental factors. The principal proposed mechanism of PSCCs is radiation damage to germinative-zone dividing cells (4), which induces a short period of mitotic inhibition at the basement membrane, followed by overcompensation with disorganized abnormal mitoses (8,9). This results in accumulation of aberrantly organized and shaped lens fiber cells with pyknotic nuclei that are theorized to produce the cloudy lentoid body at the posterior subcapsular region (6). This manifests histologically as small dots and vacuoles of opacification, which progressively coalesce over time to form larger conglomerates, eventually causing visual impairment if left untreated (10). Relatively minor opacities to the posterior pole of the lens's visual axis lead to significant visual impairment (10).

Interestingly, PSCCs have been viewed as a "cancer-like pathology" of the lens (4), as the ionizing radiation also causes damage to specific genes involved in DNA repair and cell cycle control that is usually attributed to the process of carcinogenesis (4).

Emerging Evidence and Its Consequences on the Understanding

Major influencing factors for radiation-induced cataractogenesis. Many factors have been shown to influence the development of radiation-induced cataractogenesis. Age is the single most important risk factor, with a 15% increased risk for each year of life (11,12). Other risk factors identified include female sex, marital status (single), low socioeconomic status, $>$ 15 pack-year smoking history, $>$ 25-kg/m^2 body mass index, increased alcohol consumption, diabetes, hypertension, hypercholesterolemia, and chronic use of systemic steroids (11,12). However, most of the previously stated risk factors also affect the incidence of non–radiation-induced cataracts, such as senile cataracts (3), which complicates the analysis of radiation-induced cataractogenesis prevalence.

Lately, there has been increased interest in identifying specific genetic variations that influence individual susceptibility to radiation-induced cataracts following radiation exposure (13). These potential genetic factors complicate the elaboration of safety guidelines, which currently assume relative homogeneity of radiation sensitivity within populations (7). In animals, earlier appearance of radiation-induced cataractogenesis was

found in mice nullizygous or heterozygous for the DNA repair genes *ATM*, *RAD9*, and *BRCA1* (14,15). Indeed, human *ATM* polymorphisms were found in atomic bomb survivors and seemed to modify the risk of undergoing cataract surgery (16). Interestingly, heterozygosity for the *ATM* gene has been estimated to occur in 1%–3% of the US population (17). *BRCA1* and *BRCA2* germline mutations are found in 2% of the Ashkenazi Jewish population. If some interventionalists are more radiation-sensitive than others, should consideration be given to genetic screening for those with diminished DNA repair ability?

Radiation-induced oncogenesis. In the past few years, it was hypothesized that radiation cataractogenesis may possibly function as a stochastic process (6,18,19). Indeed, Hamada et al (6) proposed that the lack of a dose-rate effect may suggest that only an initial accumulation of damage from radiation is needed within the lens tissue to trigger the stochastic process (6). This stochastic relationship with radiation exposure is also evident in radiation-induced oncogenesis, which is emerging as an important group of pathologic conditions to investigate when exploring radiation exposure to the head region.

A particularly concerning group of oncologic entities in close proximity to the eye are brain neoplasms. Current evidence of a direct causal relationship between brain tumors and chronic occupational radiation is suggestive but not conclusive as a result of small sample sizes and lack of follow-up (20). Nonetheless, when anecdotal reports of clustering are considered, the issues become quite concerning (20). For example, a 2013 case study (21) identified 31 cases of brain cancers in interventionalists, including glioblastoma multiforme (n = 17), meningioma (n = 5), and astrocytoma (n = 2). These specific tumors are known for their potential to be radiation-induced (22), with some case cohorts observing an 85% left-sided dominance thought possibly to be secondary to the more direct radiation exposure to this area during interventional procedures (21). In addition, a recent observational study (23) performed on technologists working with radiation showed a twofold increased risk of brain cancer mortality and mild elevations in the incidence of melanoma and breast cancer compared with technologists never exposed to radiation.

In addition, although specific genes have not yet been conclusively identified, epidemiologic studies, as mentioned earlier, have displayed variations in radiation sensitivity among particular subpopulations, which may have important implications regarding radiation-induced oncogenesis of the head and neck (24).

Dosing of radiation: Concepts and mechanisms understood. As mentioned earlier, there is still uncertainty about the exact pathogenesis of cataracts. The knowledge is also scarce regarding the relationship among cataract development, dose protraction and latency period, and stochastic versus deterministic nature of radiation-induced cataracts (4).

In April 2011, following emerging evidence from numerous studies, the International Commission on Radiological Protection (ICRP) reviewed its previous 2007 guidelines concerning lens dose thresholds for radiation cataractogenesis (25,26). The eye lens absorbed dose threshold was established at 500 mSv for lifetime exposure and was decreased from 150 mSv to 20 mSv per year for the annual occupational exposure limit (25,26). The annual occupational exposure limit now has to be averaged over 5 years, with no single year to exceed 50 mSv (25). The slim evidence of the risk-lowering effect of dose protraction has suggested that it did not significantly affect the threshold dose (18,19). Data on the latency period is also scarce and suggest an inverse relationship between dose and latency (27). To better understand the decision of the ICRP to significantly decrease the radiation thresholds for professionals, knowledge of the studies that led to those changes is essential.

Field Studies: Investigations of Dose Thresholds and Dose Effects

In 2007, Neriishi et al (28) analyzed the radiation dose exposure and dose response of 3,761 atomic bomb survivors, including 479 postoperative cataract cases. They found an increased cataract prevalence with a dose of 1 Gy at an odds ratio (OR) of 1.39. Within the 0–1-Gy range, a nonsignificant dose threshold of 0.1 Gy was seen. These results advocated for a far lower threshold than proposed in the 2007 ICRP recommendations, and also suggested the concept of a no-threshold dose–response relationship between radiation exposure and cataractogenesis (28).

Previous research also supported the conclusions of Neriishi et al (28). At 1 Gy exposure, a study by Hall et al (5) reported a 1.49 OR for PSCC in infants treated with radiation therapy for skin hemangiomas, and Worgul et al (29) reported an OR of 1.42 for PSCCs when examining 8,607 Chernobyl cleanup workers. These studies reporting an increased risk of cataracts with low doses of radiation supported the idea of a threshold as low as 0.5 Gy or no threshold at all (13,18,19).

Occupational Radiation Exposure Studies: Dose Accumulation and Cataract Prevalence

Multiple epidemiologic and clinical studies investigating occupational radiation exposure to health care professionals have confirmed the prevalence of radiation-induced cataracts in the medical community. In 2008, Chodick et al (11) published results of a 1983–2004 cohort study of 35,705 US radiology technologists

(initially cataract-free), aged 24–44 years, followed up with two detailed questionnaires. They reported 2,382 cataracts and 647 cataract extractions, with 25% of the cataracts occurring before 50 years of age. Results showed a mean radiation dose to the lens of 28.1 mGy in the entire cohort, and, after being adjusted for other known confounding factors, suggested a possible risk of cataractogenesis at low radiation doses. Just three diagnostic head or neck radiography procedures were found to increase the risk of cataractogenesis. The dose–to–risk of cataract relationship was strongest with subjects younger than 50 years old, with PSCCs being the most prevalent type of opacity found in this age group (11). In addition, a study on Finnish physicians (12) reported an OR for any lens opacities of 0.13 (95% confidence interval, −0.02 to 0.28) per 10 mSv of whole-body cumulative effective dose.

Organizations such as the ICRP, the United Nations Scientific Committee on the Effects of Atomic Radiation, the International Atomic Energy Agency, and the National Council on Radiation Protection and Measurements have also attempted to raise awareness and evaluate the risk of cataracts from a chronic occupational radiation exposure by publishing annual reports and guidelines and initiating collaborative studies (25,26,30–32). In the past few years, the steady increase in the number of medical procedures requiring radiation has led the United Nations Scientific Committee on the Effects of Atomic Radiation to convene a task force to analyze and record worldwide radiation exposures from the medical field (32). In an epidemiologic study by the occupational cataracts and lens opacities in interventional cardiology (O'CLOC) group in 2013, Jacob et al (10,33) investigated the risk of cataract in French interventional cardiologists and electrophysiologists. The retrospective assessment showed a cumulative eye lens exposure ranging from 25 mSv to more than 1,600 mSv, as estimated by taking into account the number of procedures performed, the average radiation dose per procedure, and the various radiation protection equipment used (33). A mean exposure of 423 mSv was found for a mean working time of 22 years, with an OR of 3.8 (95% confidence interval, 1.3–11.4) for the development of PSCCs in exposed medical personnel compared with nonexposed controls. These data suggested that approximately 25% of these professionals were exposed to more than the revised ICRP threshold of 500 mSv. In addition, the new ICRP annual exposure limit of 20 mSv/y was surpassed at least once by 60% of cardiologists during the study period (33).

Indeed, interventional cardiologists have become the focus of many similar studies attempting to estimate occupational lens dose and cataract prevalence in this group. The study of Ciraj-Bjelac et al (34) found a lifetime cumulative dose to the lens of 3.7 Gy ± 7.5 (range, 0.02–43Gy) over an average of 9 years of work, and Vano et al (35) found a cumulative occupational lens dose of 6.0 Sv ± 6.6 (range, 0.1–27 Sv) over a mean working time of 14 years. These results were, like those of the O'CLOC study (33), estimated from the numbers of procedures performed by the interventionalist, the baseline eye lens dose per procedure type, and the radiation protection conditions. The doses were likely overestimated in these studies, as lens opacities were found in only 38% of participants by Vano et al (35), less than what would be expected with medians of such high values (33). The wide discrepancies between estimated levels of exposure and the PSCC incidence was hypothesized to be secondary to a lack of systematic monitoring, leading to inaccurate estimates of occupational exposure (33). However, all these studies ultimately did show an increase in the crude relative risk of radiation cataractogenesis, which was still significant after being adjusted for confounding factors and compared with the control subjects (33–35).

Another important source of discrepancies in studies reporting the prevalence of PSCCs comes from the use of different classification systems and grading schemes available. For example, the reported prevalence of posterior subcapsular opacities in the O'CLOC (10), Vano et al (34), and Ciraj-Bjelac et al (35) studies were evaluated at 17%, 38%, and 52%, respectively. Ciraj-Bjelac et al (35) and Vano et al (34) used a modified Merriam–Focht system specifically designed for posterior lens opacities (36), whereas the O'CLOC study (10) used the Lens Opacities Classification System III (37), a system that evaluates and grades any type of cataract. With this grading system, the ophthalmologist compares the morphology of the lens fiber in all regions of the lens (nuclear, cortical, and posterior subcapsular) and compares it to reference slides to determine the severity of the opacities in each region (37). All these grading systems also differ in the methods used to assess the opacities, including dilated slit-lamp biomicroscopy, retroillumination, and Scheimpflug imaging (6). These factors constitute a major limiting factor when comparing studies and further complicate pooled analysis (6). Hence, for future studies, standardization of the cataract grading systems is critical for an effective analysis of prevalence (6).

Protocol, Clinical Applications, and Their Issues

Monitoring and protection. Radiation dose monitoring devices are essential to assess the physician's occupational risk. Because of the lack of a direct quantification method to measure eye lens dose, studies such as those of the O'CLOC group (33), Vano et al (34), and Ciraj-Bjelac et al (35) have sought to use various formulae to estimate retrospective assessment of eye lens exposure. These methods are not reliable or reproducible

for the assessment of radiation dose as a result of inherent differences in patients, physicians, procedures, and protective equipment used (38,39). In addition, many physicians unfortunately do not routinely use personal dosimeters even when they are made available (40).

$H_p(3)$ is the most accurate operational quantity for eye lens dose (41). However, the dosimeters provided to clinicians often only include effective dose/whole-body dosimeter $H_p(10)$ and local skin dose/partial body dosimeter $H_p(0.07)$, and calibration for $H_p(3)$ is impossible (40,41). In this context, $H_p(0.07)$ is used to provide the closest dose estimate to the lens (38,40).

Recently, studies have explored the use of electronic active dosimeters that provide physicians with real-time maximum dose rates and total cumulative dose for a single procedure (42,43). Such devices could serve as an occupational dose education and awareness tool for physicians while potentially increasing compliance with the use of protective equipment and other dose-reduction techniques (42,43). To decrease chronic radiation exposure, there has to be strict adherence and appropriate use of the active (patient dose reduction techniques) and passive (room and personal protection devices) components of radiation protection (**Table**).

Radiation to the eye in the fluoroscopy room mainly comes from scatter radiation from the patient (44,45). Central to any dose reduction strategy is first and foremost to decide if the examination to be performed is clinically indicated, and if the use of the fluoroscopy guidance is essential. If the procedure is deemed appropriate, careful planning of the procedure to optimize the use of radiation is necessary (44). Strategies to reduce radiation dose to the patient include shorter fluoroscopy time, decreased frame rate, use of collimation, avoidance of magnification, decreased digital subtraction angiography sequence acquisitions, increased distance between the source and the patient, and decreased distance between the patient and the image receptor (44).

Table. Protective Measures to Decrease Radiation Exposure in Interventional Radiology

	Passive measures
Personal protection	Protective glasses
	Lead apron
	Lead gloves
	Thyroid shield
Room-specific protection	Suspended screens
	Suspended drapes
	Disposable shielded drapes
	Active measures
Operator-specific strategies	Decrease length of fluoroscopy
	Decrease frame rate
	Use collimation
	Avoid magnification
	Decrease number of DSA acquisitions
	Increase distance between patient and source
	Decrease distance between patient and image receptor
	Increase distance between operator and source if possible

DSA = digital subtraction angiography.

Passive strategies such as room shielding devices protect against the scatter radiation and include ceiling-suspended shields and shielded drapes (44). Ceiling-suspended shields, when used properly, were shown to reduce occupational exposure doses by as much as 100-fold and therefore should always be used if interventionalist positioning is not compromised as a result (44). Similarly, drapes placed around the image intensifier were shown to provide an approximate 90% dose exposure reduction to the endoscopist during endoscopic retrograde cholangiopancreatography (45). Other measures to reduce scatter radiation include the use of protective disposable shielded drapes, which showed a 23% reduction in total radiation dose to the operators in a randomized controlled trial (46) and dose reduction to the hands and chest of interventionalists of 49% and 55%, respectively, during endovascular aortic repair procedures (47).

Finally, to achieve the greatest reduction in dose exposure, room-shielding equipment must be combined with personal protective devices. These include protective aprons, thyroid shields, gloves, and eyewear for physicians (44). The use of lead eyewear that is properly fitted and has lateral eye shielding can reduce eye lens dose by a factor of 2.1 for the tube-side eye and 0.8 for the other eye (48). Unfortunately, evidence suggests poor compliance, with only 25% of interventionalists and 36% of cardiologists wearing glasses during procedures (41). Although current technology has the ability to provide major protective benefits to physicians, ineffective protocols and poor compliance need to be addressed in future guidelines.

Proficiency and treatment procedure. Without comprehensive evidence-based guidelines, effective dose monitoring tools, and the use of appropriate protective equipment and dose-reducing strategies, physicians are at risk of pathologic manifestations from their occupational radiation exposure. This might impact the physician's professional proficiency, quality of life, and career span (6). For example, because of their central position, PSCCs may cause significant visual impairment and decrease contrast sensitivity and present a specific surgical group of patients that is likely to be younger than the majority of patients with senile cataracts (49).

CONCLUSIONS

Throughout the present paper, the numerous potential risk factors of cataractogenesis are presented, and the pathogenesis of radiation-induced cataracts is reviewed.

A brief parallel was made between radiation-induced cataractogenesis and oncogenesis, two processes that share multiple similarities. The recent IRCP guidelines changes were described, and the studies that led to these changes were presented. Future areas of research include the study of specific genetic traits that could potentially affect an individual's vulnerability to radiation and more precise and homogeneous ophthalmologic diagnostic tools for the diagnosis of PSCCs. In the current interventional radiology field, there is an urgent need to recognize that there is no safe dose of radiation. Will we one day see posterior chamber cataracts as the proverbial canary in the coal mine? To prevent the detrimental effects of occupational radiation on health workers, there must be a strict worldwide application of the recent lower radiation threshold guidelines, a more effective means of monitoring radiation exposure, and, finally, the consistent use of appropriate radiation-protection strategies.

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CME TEST QUESTIONS: MARCH 2017

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The CME questions in this issue are derived from the article "Radiation Cataractogenesis: The Progression of Our Understanding and Its Clinical Consequences" by Khan et al.

1. Ionizing radiation is associated with which one of the following?
   a. Cortical cataracts.
   b. Uveitis.
   c. Corneal defects.
   d. Posterior subcapsular cataracts.

2. Posterior subcapsular cataracts (PSCCs) are associated with all of the following statements EXCEPT:
   a. PSCCs can cause significant visual impairment even with minor opacities in the posterior pole of the lens visual axis.
   b. PSCCs due to ionizing radiation have been viewed as "cancer-like pathology."
   c. The only known cause for PSCC is exposure to ionizing radiation.
   d. PSCCs are histologically characterized by vacuoles of opacification that coalesce and form conglomerates.

3. Factors that influence radiation-induced cataractogenesis include all of the following EXCEPT:
   a. Age.
   b. Male gender.
   c. Genetic mutations such as ATM polymorphism.
   d. Body mass index (BMI) $>25 kg/m^2$.

4. What are the occupational exposure limits to the lens established by International Commission on Radiological Protection (ICRP) 2007 guidelines?
   a. 500mSv threshold for lifetime exposure, 20mSv per year for annual occupational exposure limit.
   b. 500mSv threshold for lifetime exposure, 50mSv per year for annual occupational exposure limit.
   c. 1000mSv threshold for lifetime exposure, 50mSv per year for annual occupational exposure limit.
   d. 1000mSv threshold for lifetime exposure, 1000mSv per year for annual occupational exposure limit.

5. True or false: Recent studies indicated that radiation cataractogeneis may have a component that is independent of the dose (stochastic effect)
   a. True
   b. False