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Determining the Effect of Ultraviolet Light on the Development of Hydatid Cyst in Mice

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ARTICLE INFO	ABSTRACT
Article history: Received 05 Jun 2026 Revised 20 Jul. 2026 Accepted 24 Aug. 2026 Available online 15-09-2026 Keywords: Hydatid cyst Mice Protoscoleces Viability	Cystic echinococcosis, caused by <i>Echinococcus granulosus</i>, remained a major zoonotic disease of public health and economic importance. This study evaluated the effects of ultraviolet irradiation on hydatid cyst development in mice and associated histopathological changes. Viable protoscoleces recovered from fertile hydatid cysts were exposed to 18,000, 27,000, 54,000, or 81,000 erg/mm²/s. Swiss albino mice (five per group) were inoculated intraperitoneally with 400 irradiated protoscoleces; controls received non-irradiated protoscoleces. Four weeks later, all mice were challenged with 2,000 viable non-irradiated protoscoleces. After six months, the mice were sacrificed, and cyst establishment, distribution, size, fertility, and histopathological alterations were evaluated. Irradiation significantly affected cyst establishment and development. Establishment rates were 22.6% at 18,000 and 27,000 erg/mm²/s and 42.0% at 54,000 and 81,000 erg/mm²/s, compared with 100% in controls. Most cysts occurred in the liver and peritoneal cavity and were generally smaller and less developed in irradiated groups. Histopathological examination revealed hepatic necrosis, inflammatory cell infiltration, coagulative necrosis, edema, emphysema, and disrupted tissue architecture, indicating reduced viability and pathogenicity of irradiated protoscoleces. Irradiation significantly reduced protoscolex infectivity and developmental capacity, resulting in fewer, smaller cysts and marked histopathological alterations compared with controls. Lower doses produced greater inhibitory effects than higher doses. These findings suggested that ultraviolet irradiation could reduce parasite infectivity and provided a basis for further investigation of its potential applications in cystic echinococcosis control and the development of immunoprophylactic strategies.

1. Introduction

Cystic echinococcosis (CE), also known as hydatid disease, is a chronic zoonotic infection caused by the larval stage of *Echinococcus granulosus*. It remains one of the most important neglected parasitic diseases worldwide because of its considerable impact on both human health and livestock production (WHO, 2023). The disease is endemic in many regions, including the Middle East, North and East Africa, Central Asia, South America, and Mediterranean countries, where close contact between livestock and dogs facilitates parasite transmission. Besides causing significant

morbidity in humans, cystic echinococcosis is responsible for substantial economic losses due to reduced livestock productivity, condemnation of infected organs at slaughter, and the high costs of diagnosis and treatment (WHO, 2023).

The life cycle of *E. granulosus* involves canids as definitive hosts and herbivorous animals as intermediate hosts, whereas humans become accidental intermediate hosts through ingestion of parasite eggs. Following ingestion, the released oncospheres migrate through the bloodstream and develop into hydatid cysts, primarily in the liver and lungs. Fertile hydatid cysts contain numerous protoscoleces, which represent the infective stage responsible for

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transmission to the definitive host and for the development of secondary hydatid cysts following cyst rupture or experimental inoculation. Consequently, reducing the viability and infectivity of protoscoleces is considered an important strategy for interrupting parasite transmission and limiting disease progression.

Ultraviolet (UV) radiation is a well-established physical agent capable of inducing irreversible damage to nucleic acids, proteins, and cellular membranes through photochemical reactions and oxidative stress, thereby reducing cellular viability and infectivity. UV irradiation has also been shown to reduce the viability of parasites and other pathogenic organisms through DNA damage, oxidative stress, and disruption of cellular functions. Recent studies have demonstrated its effectiveness against helminth eggs, highlighting its potential as a non-chemical strategy for parasite control (Mahmoud et al., 2023). Because of these biological effects, UV irradiation has received increasing attention as a potential parasite attenuation strategy that may reduce infectivity while preserving sufficient antigenicity to stimulate protective immune responses (Hassan, 2022; Al-Obaidi, 2024).

Several studies have investigated the effects of UV irradiation on *E. granulosus* protoscoleces. Hassan (2022) demonstrated that sublethal doses of UV irradiation significantly reduced protoscolex viability. Alsudani (2006) further showed that the effects of UV irradiation were influenced by preservation medium and pH, with hydatid cyst fluid providing greater protection than normal saline and neutral pH favoring prolonged parasite survival. Karim and Suleiman (2023) confirmed that UV irradiation reduced both protoscolex viability and evagination in a dose-dependent manner. More recently, Al-Obaidi (2024) reported that pH plays a critical role in modulating the biological response of irradiated protoscoleces. Ahmed et al. (2024) demonstrated that mice immunized with UV-irradiated protoscoleces developed significant protection against secondary hydatid infection, suggesting that UV attenuation may preserve parasite antigenicity while reducing infectivity. Furthermore, Zhao et al. (2025)

highlighted the importance of preservation conditions in determining the survival and infectivity of irradiated protoscoleces.

Despite these advances, most previous studies have focused primarily on evaluating the in vitro effects of UV irradiation on protoscolex viability, survival, and evagination. Comparatively little information is available regarding the influence of different UV irradiation doses on the subsequent establishment and development of hydatid cysts in vivo, particularly with respect to the associated histopathological alterations in infected tissues. Therefore, the relationship between UV-induced attenuation of protoscoleces and the progression of secondary hydatid cysts remains insufficiently understood.

Accordingly, the present study was designed to evaluate the effect of different doses of ultraviolet irradiation on the establishment and development of hydatid cysts in experimentally infected mice. In addition, histopathological changes associated with UV-irradiated protoscoleces were investigated to provide further insight into the biological effects of UV irradiation and to assess its potential application as a parasite attenuation strategy for the control of cystic echinococcosis and future vaccine development.

2. Methodology

2.1 Sample collection and experimental animals

Hydatid cysts were collected from naturally infected camels slaughtered at Tambool abattoir, Central Eastern Sudan. The camels served only as the source of fertile hydatid cysts from which viable *Echinococcus granulosus* protoscoleces were recovered.

Male Swiss albino mice weighing 25–30 g were used as the experimental animal model. Recovered protoscoleces were exposed to different doses of ultraviolet (UV) irradiation before being intraperitoneally inoculated into the mice to evaluate the effect of UV irradiation on hydatid cyst development.

2.2 Pathological examination

Postmortem examination was carried out immediately after slaughter. Different organs

were examined carefully for the presence of any cyst. Protoscoleces from fertile cysts recovered from camels were counted and injected intraperitoneally into the mice. The number of scoleces was 400 in the first dose and 2000 in the challenge dose. Protoscoleces were irradiated with UV rays with different doses: 18000 Erg/mm²/sec for 20 minutes, 27000 Erg/mm²/sec for 30 minutes, 54000 Erg/mm²/sec for 60 minutes, and 81000 Erg/mm²/sec for 90 minutes. Twenty-five mice, 20 weeks of age, were divided into five groups of five mice each. Group 1 received 400 protoscoleces exposed for 20 minutes with UV irradiation (18000 Erg/mm²/sec). Group 2 received 400 protoscoleces exposed for 30 minutes with UV irradiation (27000 Erg/mm²/sec). Group 3 received 400 protoscoleces exposed for 60 minutes with UV irradiation (54000 Erg/mm²/sec). Group 4 received 400 protoscoleces exposed for 90 minutes with UV irradiation (81000 Erg/mm²/sec). Group 5 received 400 protoscoleces unexposed to UV irradiation and was considered the untreated control. Four weeks post-infection, all groups of mice were challenged intraperitoneally with an equal number of 2000 protoscoleces. All groups of mice were sacrificed after 6 months and established cysts were counted and examined.

3. Results and discussion

Cysts were subjected to irradiation with UV light at four different doses (18000, 27000, 54000 and 81000 Erg/mm²/sec). The establishment rates compared to the controls were found to be 22.6%, 22.6%, 42.00% and 42.00%, respectively (Table 1, Fig. 1). The differences in establishment rates between the irradiated groups and the controls were found to be statistically insignificant.

UV irradiation at 18000 Erg/mm²/sec (20 min) revealed establishment of 3 cysts in one mouse and 2 cysts in another and only one cyst in the remaining 2 mice. In this group, all the cysts encountered were sterile and small in size (Table 2, Fig 2, Fig 3). They were located either in the liver (5) or peritoneum (2).

Certain parameters were considered. These were:

- 1- Morphological changes of the protoscoleces.
- 2- Viability of the cyst.
- 3- Development of the cyst.

The establishment rate was determined using the formula:

$$\text{Establishment rate} = \frac{\text{Number of cysts in each group}}{\text{Number of cysts in the control group}} \times 100$$

2.3 Histopathology

Representative samples from the infected organs were fixed in 10% normal saline for histopathological studies. After fixation, the tissue (cyst wall and surrounding tissue) was divided into three equal parts. Samples were dehydrated in descending grades of alcohol, cleared in xylene and embedded in paraffin wax. After blocking, sections 5 µm thick were cut using a rotary microtome. Tissue sections were floated in a water bath at 48 °C and collected on glass slides. Sections were further deparaffinized in xylene and dehydrated in descending grades of alcohol then washed in distilled water. Sections were stained using haematoxylin and eosin (H&E) as described by Drury and Wallington (1967).

In mice irradiated with 27000Erg/mm²/sec (30 min), 2 mice showed 2 cysts each and one mouse showed 3 cysts and one mouse showed no cyst. All cysts were small in size and located in the liver (4) and liver + peritoneal cavity (3). Two cysts were sterile and one cyst was fertile (Table 3, Fig 4, Fig 5).

In the group irradiated with 54,000 erg/mm²/sec (60 min), hydatid cysts developed in four of the five mice. Three mice each developed three cysts, whereas one mouse developed four cysts, giving a total of 13 cysts. All cysts were small in size and were distributed in the liver (3 cysts), lung (3 cysts), and liver with the peritoneal cavity (7 cysts). Most cysts were fertile, while three cysts located in the lungs were sterile (Table 4; Figs. 6 and 7).

In the group irradiated with 81,000 erg/mm²/sec (90 min), four mice developed

cysts, with two mice harboring three cysts each, one mouse harboring five cysts, and one mouse harboring two cysts, resulting in a total of 13 cysts. All cysts were small and were located in the liver (8 cysts) or in the liver together with the peritoneal cavity (5 cysts). Ten cysts were fertile, whereas three cysts were sterile (Table 5; Figs. 8 and 9).

As expected, the control group exhibited the highest level of cyst establishment. The number of cysts ranged from 5 to 11 per mouse, with a total of 31 cysts. All cysts were small and were distributed in the liver, peritoneal cavity, and kidneys. Of the 31 cysts, 22 were fertile and 9 were sterile (Table 6; Figs. 10 and 11).

Table 1: Effect of irradiation with UV light in different groups

Dose / exposure time	Total number of cysts established	Establishment rate	Expected Responses	Residual	Probability
18000Erg/20	7	22.60%	5.600	1.400	0.280
27000Erg/30	7	22.60%	8.400	-1.400	0.280
54000Erg/60	13	42.00%	10.400	2.600	0.173
81000Erg/90	13	42.00%	15.600	-2.600	0.173
Control	31	100%	28.039	2.961	0.904

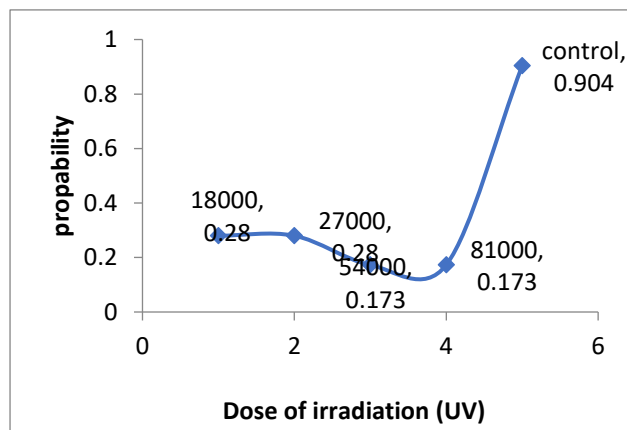


Figure 1: Effect of irradiation with UV in different groups

Table 2: Effect of irradiation with UV 1800Erg (20min) on size & stage of the cyst:

Mouse No.	No. of cysts	site	size	case
1	2	Liver & peritoneal	small	sterile
2	3	liver	small	sterile
3	1	liver	small	sterile
4	1	peritoneal	small	sterile
total	7	-	-	-

$$\text{Establishment rate} = 7 \div 31 \times 100 = 22.6\%$$

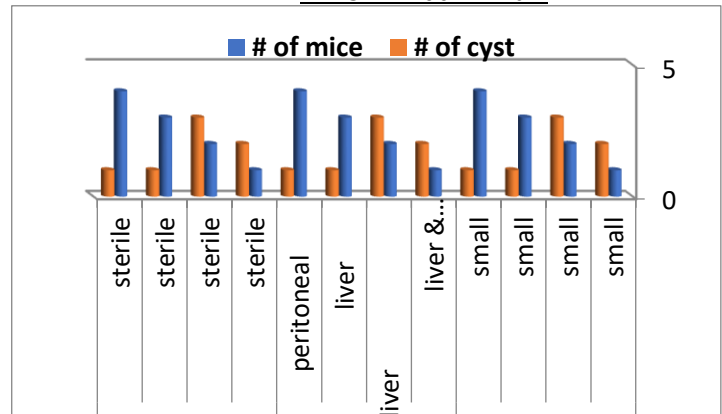


Figure 2: Effect of irradiation with UV 1800Erg (20 min) on size & stage of the cyst

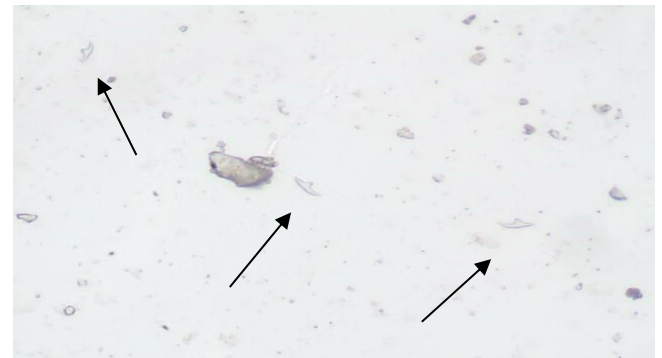


Figure 3: Mice inoculated with scoleces irradiated with 18000Erg/mm/sec (hooks)

Table 3: Effect of irradiation with UV 27000Erg (30min) on size & stage of the cyst:

Mouse No.	No. of cysts	site	Size	case
1	3	Liver & peritoneal	Small	sterile
2	2	liver	Small	sterile
3	2	liver	Small	fertile
4	0	0	0	0
total	7	-	-	-

$$\text{Establishment rate} = 7 \div 31 \times 100 = 22.6\%$$

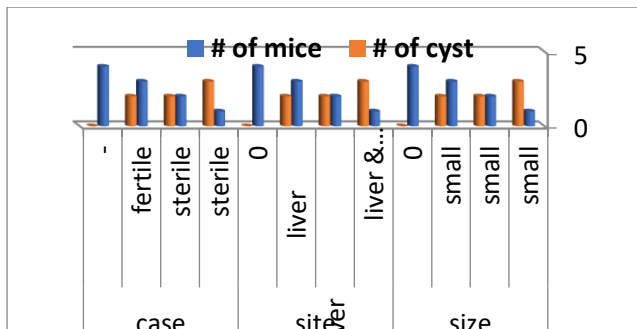


Figure 4: Effect of irradiation with UV 27000Erg (30min) on size & stage of the cyst:

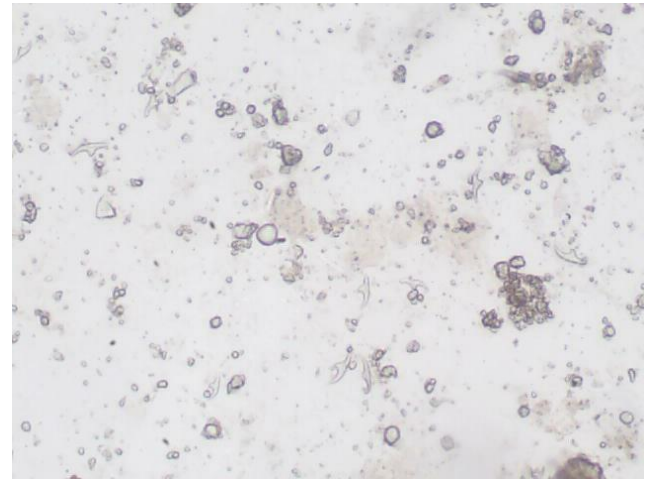


Figure 7: Mice inoculated with scoleces irradiated with 54000 Erg/mm/sec (hooks).

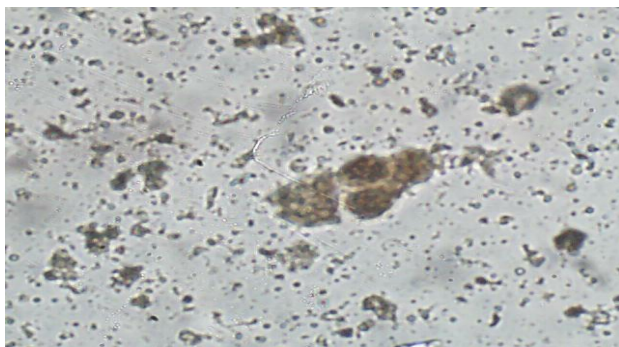


Table 4: Effect of irradiation with UV 54000Erg (60min) on size & stage of the cyst:

Mouse No.	No. of cysts	site	size	case
1	3	Liver & peritoneal	small	fertile
2	3	liver	small	fertile
3	4	Liver & peritoneal	small	fertile
4	3	lung	small	sterile
total	13	-	-	-

Establishment rate= $13 \div 31 \times 100 = 41.9\%$

Table 5: Effect of irradiation with UV 81000Erg (90min) on size & stage of the cyst:

Mouse No.	No. of cysts	site	size	case
1	5	Liver & peritoneal	small	sterile
2	2	liver	small	sterile
3	3	Liver	small	fertile
4	3	liver	small	sterile
Total	13	-	-	-

Establishment rate= $13 \div 31 \times 100 = 41.9\%$

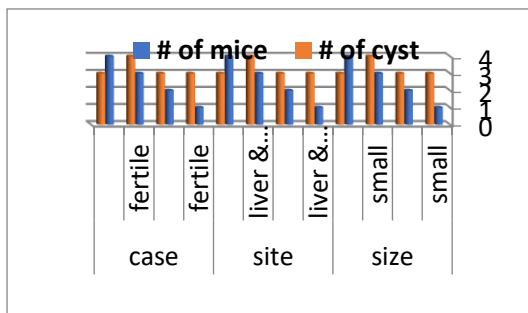


Figure 6: Effect of irradiation with UV 54000Erg (60min) on size & stage of the cyst

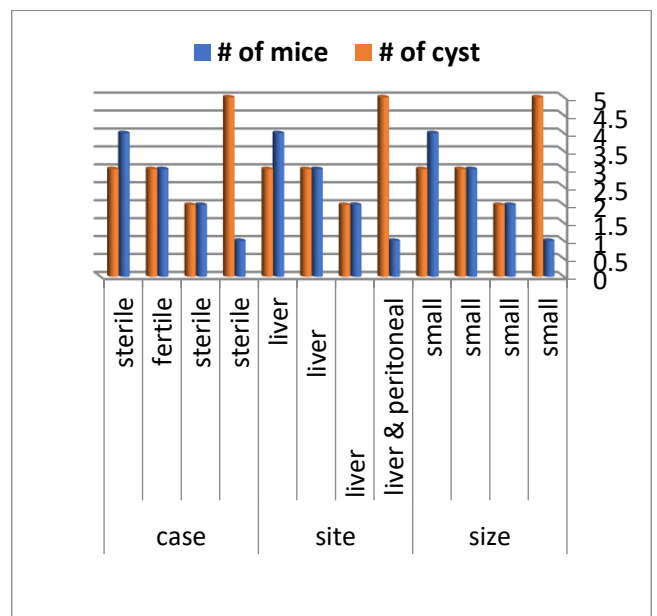


Figure 8: Effect of irradiation with UV 81000Erg (90min) on size & stage of the cyst

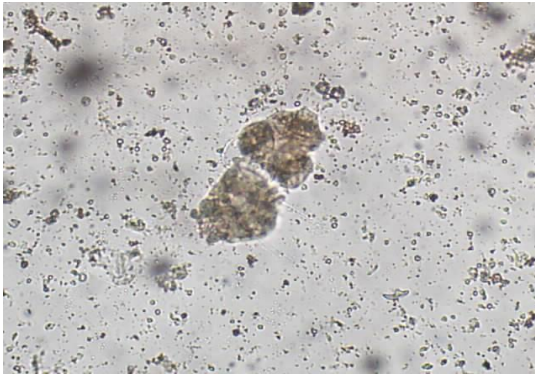


Figure 9: Mice inoculated with scoleces irradiated with 81000Erg/mm/sec(hooks).

Table 6: The Controls of irradiation with UV light.

Mouse No.	No. of cysts	Site	size	case
1	9	Liver & peritoneal	small	Sterile
2	11	Kidney & liver & peritoneal	small	sterile
3	6	Liver	small	fertile
4	5	Peritoneal	small	sterile
Total	31	-	-	-

$$\text{Establishment rate} = 31 \div 31 \times 100 = 100\%$$

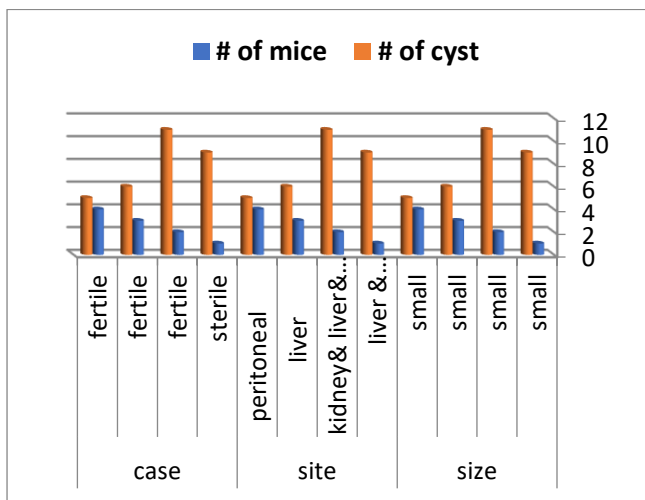


Figure 10: The Controls of irradiation with UV light.

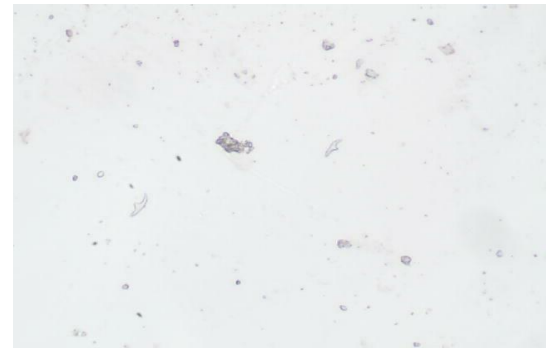


Figure 11: Control group (hooks and scoleces).

3.1 Histopathology

Liver:

In group 1, there was a very severe infiltration with necrotic hepatic cells. The cysts were very small compared to groups 2 and 3. Very few scoleces and hepatic cells were observed. In group 2, there was a very severe diffuse necrosis in hepatic cells, the cyst size was smaller compared to cysts in group 3. There was also diffuse coagulative necrosis, ruptured cysts and few scoleces (Fig 12). In group 3, there was no inflammatory reaction around small cysts; however, there were necrotic hepatic cells in the area. It was observed that the central vein was enlarged with dilated sinusoids (Fig 13). In the control group, very severe oedema, coagulative necrosis which was bordered by a narrow zone of inflammatory cell infiltrates, tissue debris and extravascular erythrocytes forming infarct-like structure was observed. In this group, large cysts and scoleces with hooks were very clear and the size of the central vein was changed (Fig 14).

Lung:

Oedema was observed in group 3. Severe emphysema and atelectasis were also observed. Scoleces were clearly visible; however, a few were observed in the section (Fig. 15).

Kidney:

Adipose tissue inflammatory cells were observed (Fig 16).

Muscles:

In group 4, the muscles surrounding the cyst were ruptured, with inflammatory cell infiltration inside the cyst. Very few scoleces were found inside the cyst, with coagulative muscle necrosis and muscle dissociation (Fig. 17).

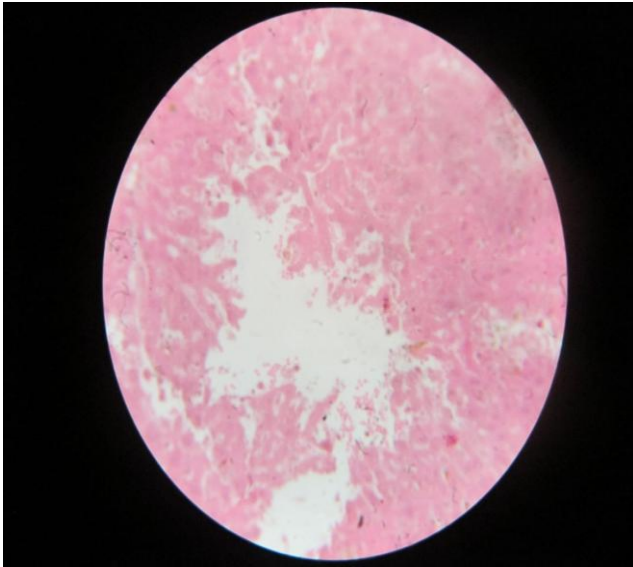


Figure 12: Histopathology of a liver exposed to 27000 Erg.

a: Ruptured cyst ,b : Scoleces ,c : Coagulative necrosis

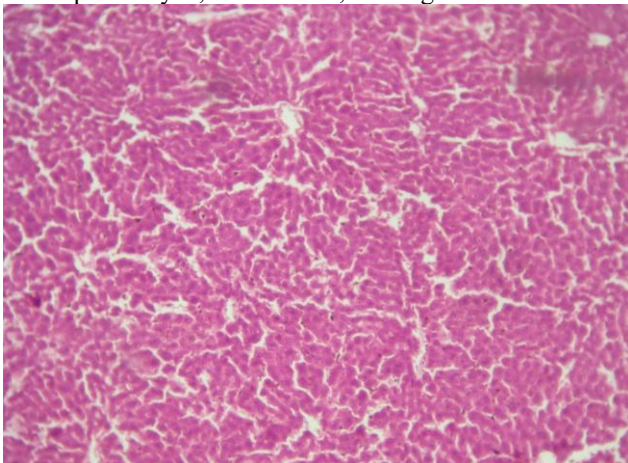


Figure 13: Histopathology of a liver exposed to 54000 Erg.

a : Central vein , b :Dilated sinusoids

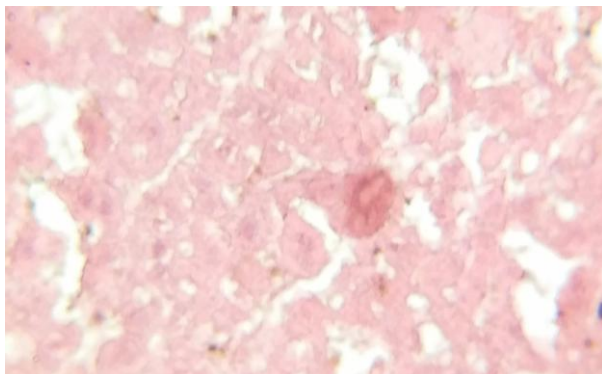


Figure 14: Histopathology of a liver in the control group.

a : Necrotic cells, b :Scoleces

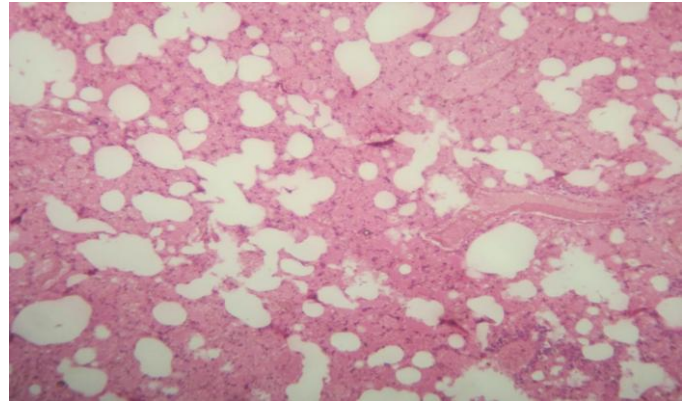


Figure 15: Histopathology of a lung exposed to 54000 Erg.

a: Emphysema, b : Oedema.

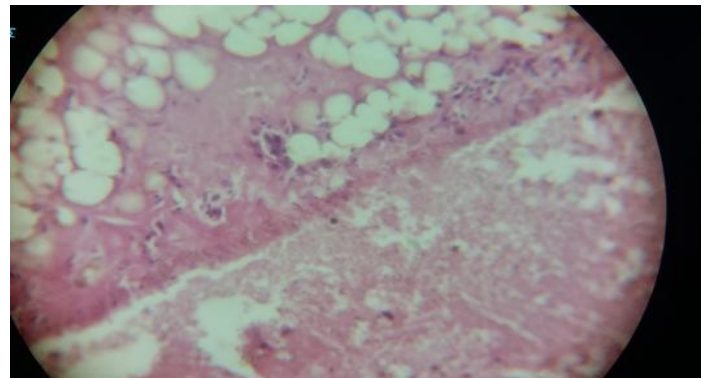


Figure 16: Histopathology of a kidney exposed to 81000 Erg.

a: Fat tissue, b: Cyst wall, c: Cyst fluid

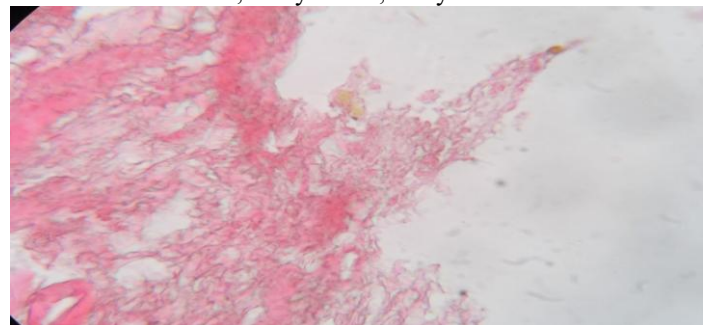


Figure 17: Histopathology of a muscle exposed to 81000 Erg.

b: Scoleces, c: Coagulative muscles.

3.2 Discussion

The present study demonstrated that ultraviolet (UV) irradiation significantly influenced the infectivity and subsequent development of *Echinococcus granulosus* protoscoleces in experimentally infected mice. Exposure to UV irradiation reduced the

establishment rate of hydatid cysts compared with the non-irradiated control group, and the resulting cysts were generally fewer in number, smaller in size, and associated with marked histopathological alterations. These findings indicate that UV irradiation adversely affects the biological activity of protoscoleces and limits their capacity to establish secondary hydatid cysts.

The present findings are consistent with previous reports demonstrating that UV irradiation exerts a detrimental effect on *E. granulosus* protoscoleces. Earlier investigations demonstrated that the biological response of protoscoleces to UV radiation is influenced not only by irradiation dose but also by environmental conditions, particularly the preservation medium and pH. Alsudani (2006) reported that hydatid cyst fluid (HCF) provided greater protection against UV-induced damage than normal saline (NS), allowing irradiated protoscoleces to survive for longer periods, while neutral pH favored parasite survival compared with acidic and alkaline conditions. Similarly, increasing UV exposure progressively reduced protoscolex viability in a dose-dependent manner. In the present study, the highest irradiation dose (54,000 erg/mm²/sec) markedly reduced protoscolex survival, with HCF consistently preserving viability longer than NS under all pH conditions. These findings further confirm the protective role of HCF while emphasizing the importance of irradiation dose and environmental conditions in determining parasite survival.

The reduction in viability observed after UV irradiation is further supported by studies evaluating structural alterations in *E. granulosus* protoscoleces. Karpisheh et al. (2023) demonstrated that decreased viability was associated with severe ultrastructural damage, including disruption of the tegument, hook disorganization, and loss of cellular integrity following exposure to protoscolicidal agents. Shnawa et al. (2022) reported that reduced viability assessed by eosin exclusion staining was accompanied by marked morphological degeneration of protoscoleces. Mahmoudvand et al. (2023) also confirmed that decreased protoscolex viability was associated

with profound structural degeneration and impaired biological activity. Although these studies evaluated different protoscolicidal approaches, their observations collectively support the present findings that UV irradiation compromises parasite viability through progressive structural damage.

While previous studies mainly evaluated the immediate effects of UV irradiation on protoscolex viability under in vitro conditions, the present study extends these observations by demonstrating that reduced viability is directly reflected in diminished in vivo infectivity. Mice inoculated with irradiated protoscoleces developed substantially fewer and smaller hydatid cysts than those in the control group, indicating that UV irradiation impaired the developmental capacity of the parasite after inoculation. This finding provides direct evidence that attenuation induced by UV exposure persists beyond the laboratory setting and significantly reduces the establishment of secondary hydatid cysts in the host.

An interesting finding of the present study was that the lower irradiation doses (18,000 and 27,000 erg/mm²/sec) produced greater inhibition of cyst establishment than the higher doses (54,000 and 81,000 erg/mm²/sec). Although this observation appears inconsistent with the expected dose-response relationship, similar variations have been reported previously, suggesting that parasite survival after irradiation may depend on several interacting factors, including irradiation conditions, preservation medium, parasite metabolic status, DNA repair mechanisms, and host immune responses following inoculation (Karim & Suleiman, 2023; Zhang et al., 2025). These findings indicate that parasite attenuation cannot be explained solely by irradiation dose and emphasize the need for further molecular investigations to clarify the mechanisms underlying this response.

Histopathological examination further confirmed the biological effects of UV irradiation. Irradiated groups exhibited extensive hepatic degeneration characterized by coagulative necrosis, inflammatory cell infiltration, hepatocellular degeneration, edema, and disruption of normal tissue architecture.

Similar pathological alterations were observed in lung, kidney, and muscle tissues, although their severity varied among irradiation groups. These observations agree with those reported by Karpisheh et al. (2023) and Mahmoudvand et al. (2023), who demonstrated that structural degeneration of protoscoleces following protoscolicidal treatment was accompanied by marked tissue alterations and reduced parasite viability. Collectively, these findings indicate that UV irradiation not only decreases parasite survival but also interferes with normal cyst development and parasite pathogenicity.

The reduction in cyst number, size, and fertility observed in irradiated groups further supports the concept that attenuation of protoscoleces reduces parasite infectivity. Ahmed et al. (2024) demonstrated that mice immunized with UV-irradiated protoscoleces developed significant protection against secondary *E. granulosus* infection, indicating that UV irradiation can reduce parasite infectivity while preserving antigenic components capable of stimulating protective immune responses. The present findings support this hypothesis, as irradiated protoscoleces exhibited markedly impaired development and produced fewer fertile cysts than the non-irradiated controls.

The present study differs from previous investigations by integrating parasitological and histopathological evaluations under in vivo conditions. Whereas earlier studies primarily focused on immediate changes in protoscolex viability following UV exposure under laboratory conditions, the current study demonstrated that these biological effects extend to reduced cyst establishment, impaired cyst development, and marked tissue alterations in experimentally infected mice. This comprehensive evaluation provides stronger evidence that UV irradiation influences not only parasite survival but also its infective potential and pathogenicity within the host.

From a practical perspective, these findings suggest that UV irradiation may represent a promising parasite attenuation strategy. By reducing protoscolex infectivity while preserving sufficient antigenicity to stimulate host immune responses, UV irradiation may

contribute to the development of safer experimental vaccines and complementary control strategies against cystic echinococcosis. Nevertheless, additional studies are required to determine the optimal irradiation dose and to elucidate the molecular and immunological mechanisms responsible for parasite attenuation.

A limitation of the present study is the relatively small number of experimental animals and the absence of molecular and immunological analyses explaining the observed reduction in infectivity. Future investigations should evaluate oxidative stress markers, apoptosis-related pathways, DNA damage, gene expression, cytokine profiles, and long-term protective immunity following inoculation with UV-irradiated protoscoleces.

Overall, the present study demonstrates that UV irradiation significantly reduces the infectivity and developmental capacity of *E. granulosus* protoscoleces while inducing marked histopathological alterations in infected tissues. These findings expand current knowledge regarding the biological effects of UV irradiation and provide experimental evidence supporting its potential application in parasite attenuation, immunoprophylaxis, and future control strategies against cystic echinococcosis.

4. Conclusions

Exposure to ultraviolet (UV) radiation significantly affected the formation and development of protoscoleces in mice. Irradiated primary heads produced fewer and smaller cysts compared to the control group, indicating reduced infectivity and impaired growth. Lower UV doses (18,000 and 27,000 erg/mm²/s) showed greater inhibition of cyst formation compared to higher doses. Histopathological examination confirmed marked histological changes and reduced viability of primary heads after irradiation. These findings suggest that UV radiation exposure may represent a promising approach for controlling echinococcosis and could contribute to future vaccine development strategies.

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