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***Research Article** (Open Access)*

DOSE-DEPENDENT HISTOPATHOLOGICAL IMPACTS OF FORMALDEHYDE EXPOSURE ON GILL, LIVER, MUSCLE, AND HEART TISSUES OF LABEO ROHITA: IMPLICATIONS FOR FRESHWATER AQUACULTURE HEALTH MANAGEMENT

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ABSTRACT
Formaldehyde is commonly used in aquaculture as a disinfectant and parasitic treatment; however, excessive exposure can cause toxic effects in fish. The present study evaluated the histopathological changes induced by different concentrations of formaldehyde in *Labeo rohita* (Rohu), an important freshwater aquaculture species. Fish were exposed to four concentrations of formaldehyde (0.3, 0.5, 0.7 and 0.9 mL in 15 L of water) along with a control group under laboratory conditions. Each experimental tub contained 15 L of dechlorinated water with 5–6 fish. The exposure durations were 48, 72 and 96 hours. Control fish showed normal tissue architecture in all examined organs. In contrast, fish exposed to formaldehyde exhibited several histopathological alterations, and the severity of lesions increased with concentration and exposure time. In the gills, epithelial lifting, lamellar fusion, hyperplasia, vascular dilation and necrosis were observed. Liver sections showed cytoplasmic degeneration, hepatocyte hypertrophy, pyknosis, hemolysis, thrombus formation and focal necrosis. Muscle tissues revealed fibre atrophy, inflammatory cell infiltration, blood congestion and haemorrhage. Cardiac tissues displayed myofibrillar fragmentation, myocardial degeneration, vascular collapse and necrosis. The results clearly demonstrate that formaldehyde exposure induces significant structural damage in vital organs of *Labeo rohita*. The observed alterations confirm the toxic potential of formaldehyde and indicate that prolonged or high-dose exposure may pose serious risks in aquaculture practices. Therefore, careful regulation and controlled application of formaldehyde are necessary to minimize its harmful effects on cultured fish.

Keywords: *Labeo rohita, formaldehyde, histopathological, gills, liver, muscle, heart*

INTRODUCTION
Water is a fundamental resource essential for life on Earth, yet its quality is deteriorating due to increasing pollution from industrial, agricultural, and domestic activities. The discharge of pesticides, heavy metals, detergents, sewage, and industrial waste has led to severe contamination of water bodies, posing risks to aquatic life and human health (Simon, 2017). Many of these pollutants exhibit acute toxicity, while others persist in the environment, bio accumulate in organisms, and disrupt ecological balance (Sheela *et al*., 2011). Heavy metals, in particular, are of significant concern as they are non-degradable and can enter aquatic ecosystems through industrial effluents, soil erosion, and leaching, ultimately accumulating in fish and other organisms (Sindayigaya *et al.,* 1994). The increased use of chemical fertilizers and pesticides has further contributed to freshwater contamination, making it unsuitable for both aquatic species and human consumption (Akhtar *et al*., 2009). Additionally, climate change-induced variations in temperature and precipitation have exacerbated the stress on aquatic ecosystems, affecting species reproduction, feeding patterns, and habitat stability (Schmeller *et al.,* 2018).

Formaldehyde
Formalin (aqueous formaldehyde gas 37%), which replaced malachite green in the treatment of parasitic diseases, has been highlighted for its effectiveness (Buchmann and Kristensson 2003). Several research articles

stated that the addition of formaldehyde illegally in food items such as fish, meat, seafood, vegetables, and fruits to extend shelf life is one of the primary forms of food adulteration (Bianchi *et al.,* 2007, Fappiano *et al.,* 2022, Jinadasa *et al.,* 2022). The increased concentration of Formalin on aquatic water reduces the amount of oxygen circulated, causes respiratory distress among the fishes, loss of balance, gulping for air, vertical movement of fishes, excessive accumulation of mucus and death (Ayuba *et al.,* 2013). Thus, treatment with formalin has been investigated for use in commercial fish farms, analyzing its efficacy, toxicity and which doses are safe for the fish species. In other words, better prophylaxis and treatment protocols and procedures are investigated (Burka *et al*).

Labeo Rohita

Rohu is a silvery white belly and a brownish or bluish back. The body is short with rounded abdomen and the dorsal profile is arched and slightly concave above the occiput, and more arched in comparison to the Barbels are slender and filamentous which arise from the upper lip. Upper lips are present and the lower lip is very thick. Dorsal fin is inserted above the tip of pelvic fin and has 7-9 rays ventral one. It has wide mouth bounded by fleshy lower and upper lips. The eyes are large and situated in the anterior half of the head. Pores may be present on the snout. The anal fin has 4-6 rays out of which is five are branched. Caudal fin is homocercal. It is distributed throughout India; it occurs in rivers throughout much northern, central and eastern India, Bangladesh, Nepal, and Myanmar and has been introduced into some of the rivers of Peninsular India, and Sri Lanka.

Histology And Histopathology

Histology is the study of the structure of biological material and how individual components are structurally and functionally related. It is central to biological and medical science since it stands at the cross roads between biochemistry, molecular biology and physiology on the one side, and disease processes and their effects on the other. Knowledge of normal histological appearance is essential if abnormal diseased structures are to be recognized. As understanding of histology is vital to comprehend biochemical and physiological processes, and to gain insight into how abnormalities of structure lead to disorders of function and biochemistry, and result in disease (Stevens and Lowe, 1991). Animal histology is aimed primarily to morphologically clarify the fine structure of the animal body. However, more recently, the main aims of histology changed to focus on the study of the function of the body at the tissue level and the clarifications of the physiological functions from the view point of cellular correlation. The main purpose of histopathology is to diagnose disease from pathological changes in tissues (Takashi, 1982).

MATERIALS AND METHODS

Collection of Fishes

Live *Labeo rohita* with an average body weight of approximately 22 g and body length ranging from 10 to 20 cm were collected from fish seed suppliers near Pullarampakkam, Thiruvallur district, Tamil Nadu. The collected fish were transported to the laboratory in insulated boxes at a density of 20 fish per box to minimize physical and physiological stress during transportation. Upon arrival, the fish were acclimatized to laboratory conditions for a period of one week in a clean plastic tub with a capacity of 15 L containing dechlorinated and aerated tap water. During the acclimatization period, the water quality parameters were maintained at a temperature of around 20 °C, pH 7.2, and dissolved oxygen level of approximately 8 mg/L. After acclimatization, the fish were exposed to different concentrations of formaldehyde (0.3, 0.5, 0.7, and 0.9 mL in 15 L of water) for exposure periods of 48, 72, and 96 hours. Each experimental tub contained 15 L of dechlorinated water with 5–6 fish. Rearing environment as

Control group: Fed with fish pellet and was not treated with formalin

- Fish treated with 0.3ml concentration of formal dehydrate in 15 liters of water.
- Fish treated with 0.5ml concentration of formal dehydrate in 15 liters of water.
- Fish treated with 0.7ml concentration of formal dehydrate in 15 liters of water.
- Fish treated with 0.9ml concentration of formal dehydrate in 15 liters of water.

Acute Toxicity

1/3 OF LC_{50} was chosen as the acute toxicity dose and the studies were carried out for three different hours such as 48hrs, 72hrs, and 96hrs. The fishes were sacrificed after each different hrs to collect the tissues (gills, liver, muscle, and heart) for histological studies and enzyme studies.

Histopathological Study

A histopathological study was conducted to examine tissue alterations in experimental fishes exposed to formaldehyde. The stomach tissues of rats fed with formaldehyde-contaminated fish showed destruction and granulation of the protective mucus layers, along with detachment from the secretory layer, indicating potential adverse health effects (Kundu *et al*., 2020).

Tissue Collection and Preparation

Gill, liver, muscle, and heart tissues were collected from both control and experimental fishes. The isolated tissues were washed thoroughly with ice-cold saline to remove blood clots and immediately fixed in 10% formalin for preservation. Control fish tissues were also fixed following the same protocol.

Fixation and Dehydration

The fixed tissues were washed thoroughly in distilled water and subjected to a graded series of alcohol solutions for dehydration. Isopropyl alcohol (99%) was used as a substitute for ethyl alcohol. The tissues were passed through progressively increasing concentrations of alcohol, with one change per hour. Since absolute alcohol is not readily miscible with paraffin, the tissues were de-alcoholised before paraffin infiltration.

Embedding, Sectioning, and Antigen Retrieval

The tissues were cleaned briefly using xylene to prevent brittleness and then transferred to a molten wax bath at 54°C–56°C for 2–3 hours for infiltration and impregnation. Paraffin wax with a melting point of 56°C–58°C was used for routine embedding. Tissue blocks were prepared using Leuckhard (L) blocks, and thin sections (4µm) were obtained using a rotary microtome.

Histochemical and Cytochemical Staining

Tissue sections were stained using the Haematoxylin and Eosin (H&E) staining technique, which provides a clear distinction between cellular components (Stevens & Lowe, 1991). Stained sections were mounted in DPX for microscopic examination. Photomicrographs were taken to document observations. The histological structure of normal tissues was carefully compared with the histopathological changes observed in the experimental tissues exposed to formaldehyde.

RESULTS AND DISCUSSION

The histopathological analysis of *Labeo rohita* exposed to formaldehyde at different concentrations (0.3 ml, 0.5 ml, 0.7 ml, and 0.9 ml) revealed progressive tissue damage in the gills, liver, muscle, and heart. The severity of these alterations increased in a dose-dependent manner. In contrast, the control group (unexposed fish) exhibited normal tissue architecture without any pathological changes, indicating that formaldehyde exposure was the primary cause of the observed damage.

Gills

Control Group:

The gill structure in the control group showed well-organized primary and secondary lamellae with intact epithelial cells. The blood vessels within the gill filaments appeared normal, without signs of congestion or haemorrhage.

Formaldehyde-Exposed Groups:

At lower formaldehyde concentrations (0.3–0.5 ml), the gills exhibited epithelial lifting, basal hyperplasia, and fusion of lamellae as an adaptive response to the toxicant. At higher concentrations (0.7–0.9 ml), more severe changes such as dilation of blood vessels, rupture of marginal filaments, and widespread necrosis were observed. These changes suggest impaired respiratory function due to reduced surface area for gas exchange, leading to hypoxia. The epithelial lifting and hyperplasia indicate a defence mechanism against toxicants, while vascular dilation and necrosis suggest excessive oxidative stress and disruption of cellular integrity.

Liver

Control Group:

The liver of the control fish exhibited normal hepatocyte structure with well-defined nuclei, clear cytoplasm, and intact hepatic cords. No signs of inflammation, necrosis, or vascular congestion were observed.

Formaldehyde-Exposed Groups:

The liver showed cytoplasmic degeneration, hepatocyte hypertrophy, and aggregation at lower concentrations (0.3–0.5 ml). As the dose increased (0.7–0.9 ml), severe haemolysis between hepatocytes, pyknosis (nuclear shrinkage), thrombus formation, and focal necrosis were evident. These alterations suggest hepatic stress due to formaldehyde-induced oxidative damage, leading to mitochondrial dysfunction and hepatocyte apoptosis. The presence of thrombus formation indicates impaired circulation and potential clotting abnormalities, which further exacerbate hepatic dysfunction.

Muscle

Control Group:

The muscle tissues of the control fish exhibited well-organized muscle fibres with no signs of atrophy, inflammation, or vascular congestion. The cellular structure remained intact with no evidence of necrosis.

Formaldehyde-Exposed Groups:

At lower concentrations (0.3–0.5 ml), the muscles exhibited atrophy, inflammatory cell infiltration, and edema. At higher concentrations (0.7–0.9 ml), haemorrhage, blood congestion, and extensive necrosis were observed. The presence of inflammatory cells suggests an immune response triggered by oxidative stress and formaldehyde toxicity. The haemorrhage and congestion indicate vascular damage, potentially caused by endothelial cell dysfunction and increased permeability.

Heart

Control Group:

The heart tissue of the control fish displayed normal cardiac muscle structure, with well-aligned myofibrils and intact vascularization. No signs of myocardial fragmentation or inflammatory infiltration were observed.

Formaldehyde-Exposed Groups:

At lower formaldehyde concentrations, the cardiac tissue exhibited myofibrillar fragmentation and early myocardial changes. With increased exposure (0.7–0.9 ml), the heart tissue showed extensive damage, including destruction of myofibrils, pericardial edema, and vascular impairment. These pathological changes indicate oxidative stress-induced apoptosis and necrosis, leading to myocardial dysfunction. The destruction of myofibrils suggests impaired contractility, which could result in circulatory failure and reduced oxygen supply to vital organs.

CONCLUSION

The present investigation demonstrates that formaldehyde exposure induces pronounced, dose-dependent histopathological alterations in *Labeo rohita*, with observable severity corresponding to increasing concentrations (0.3 ml to 0.9 ml). Compared to the control group, which exhibited normal tissue architecture, the exposed specimens showed significant damage across vital organs, including the gills, liver, muscle, and heart. Histological lesions such as lamellar fusion, cytoplasmic degeneration, haemorrhage, necrosis, and myocardial disruption were progressively intensified with higher exposure levels. These findings clearly reflect the deleterious impact of formaldehyde on fish physiology and health, underscoring its toxic potential even at sub lethal doses. The study emphasizes the urgent need for stricter regulatory controls on formalin use in aquaculture and advocates for the development and application of safer, eco-friendly alternatives to minimize harm to aquatic life and ensure environmental sustainability.

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Authors' Contributions:
Farheen Khanam.N: Conception and design, conducted experiment, writing of the original draft; manuscript preparation – review and editing; methodology, slide preparation; **Dr. B. Dilshad Begum** - final validation of the manuscript and supervision.

Consent to Participate
Both the authors contributed to the study. Both authors read and approved the final manuscript.

Consent for Publication
Both the authors gave their consent for research publication.

Disclosure Statement
No potential conflict of interest was reported by the author(s).

Additional Information

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HISTOPATHOLOGY IMAGES

CONTROL

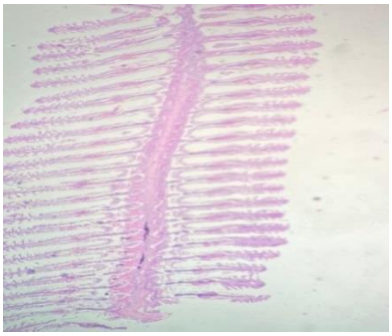


FIG NO 1. GILLS IN *LABEO ROHITA* - CONTROL

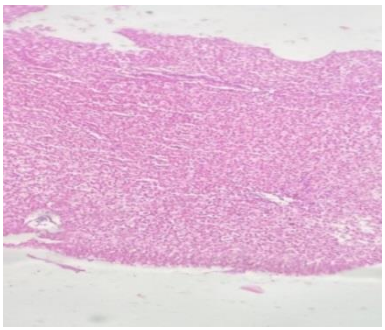


FIG NO 2. LIVER IN *LABEO ROHITA* - CONTROL

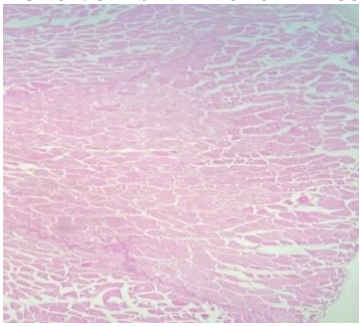


FIG NO 3. MUSCLE IN *LABEO ROHITA* - CONTROL

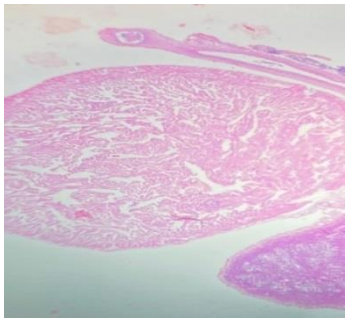


FIG NO 4. HEART IN *LABEO ROHITA* - CONTROL

GILLS

0.3 ml CONCENTRATION AT 48 HOURS

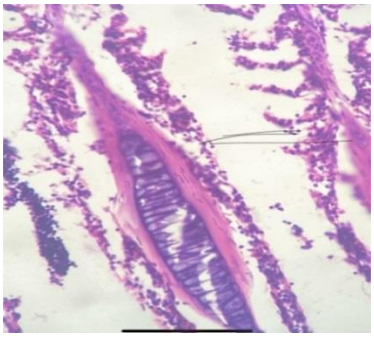


FIG NO 5. FUSION OF GILL LAMELLAE AND EPITHELIAL LIFTING

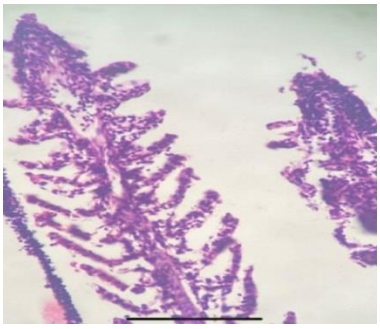


FIG NO 6. BASAL HYPERPLASIA AND DISTAL HYPERPLASIA

0.3ml CONCENTRATION AT 72 HOURS

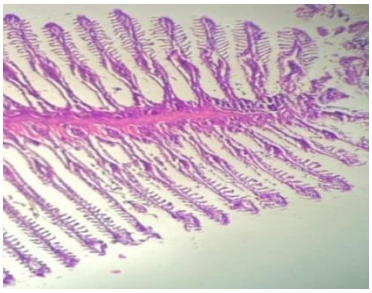


FIG NO 7. BASAL HYPERPLASIA, DISTALHYPERPLASIA AND EPITHELIAL LIFTING

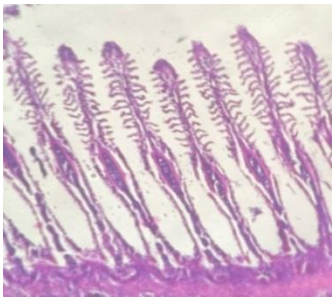


FIG NO 8. BASAL HYPERPLASIA, DISTALHYPERPLASIA AND EPITHELIAL LIFTING

0.3ml CONCENTRATION AT 96 HOURS

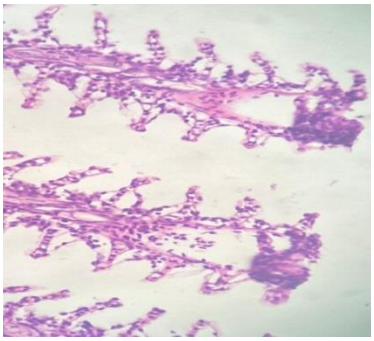


FIG NO 9. DISTALHYPERPLASIA DISORGANIZED LAMELLAE LAMELLAE AND EPITHELIAL LIFTING

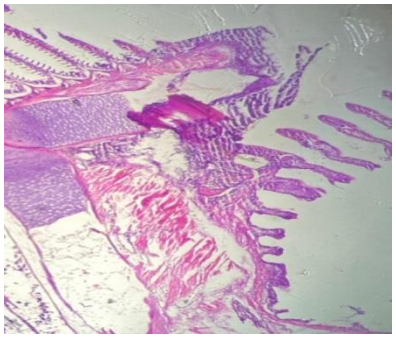


FIG NO 10. DISTALHYPERPLASIA, DISORGANIZED AND NECROSIS

0.5 ml CONCENTRATION AT 48 HOURS

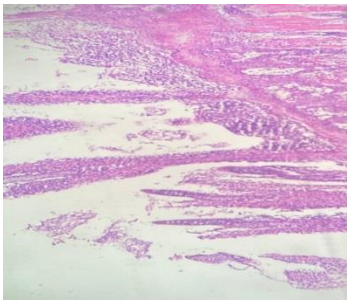


FIG NO 11. FUSION OF LAMELLAE DISORGANIZED LIFTING

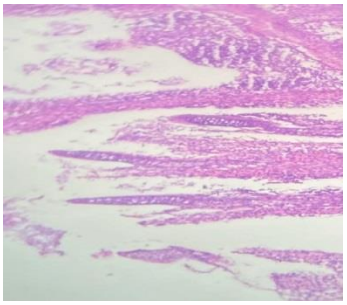


FIG NO 12. FUSION OF LAMELLAE, LAMELLAE AND EPITHELIAL EPITHELIAL LIFTING

0.5 ml CONCENTRATION AT 72 HOURS

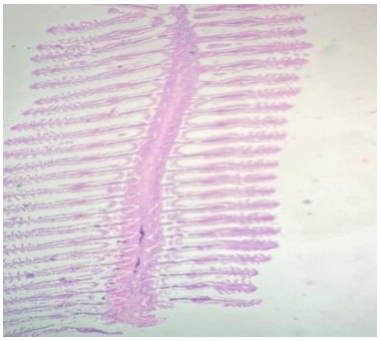


FIG NO 13. NECROSIS FORMATION

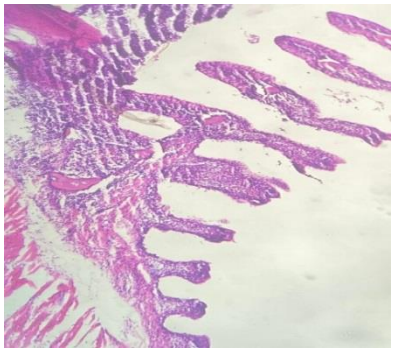


FIG NO 14.DILATION OF BLOOD VESSELS, AND DENSE INFLAMMATORY REACTION

0.5 ml CONCENTRATION AT 96 HOURS

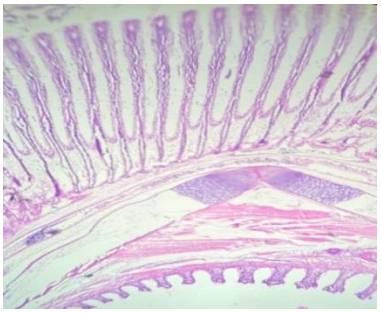


FIG NO 15. EPTHELIAL LIFTING DISORANIZATION OF LAMELLAE, BASAL HYPERPLASIA

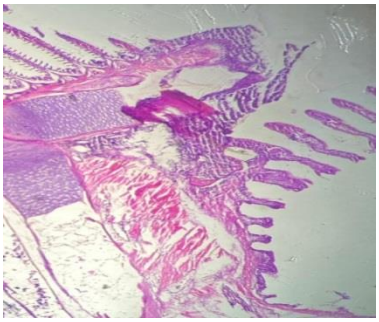


FIG NO 16. DILATION OF BLOOD VESSELS,AND DENSE INFLAMMATORY REACTION AND DEGENARATION OF LAMELLAE

0.7 ml CONCENTRATION AT 48 HOURS

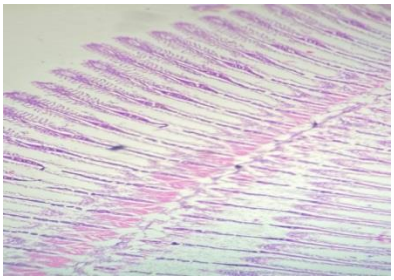
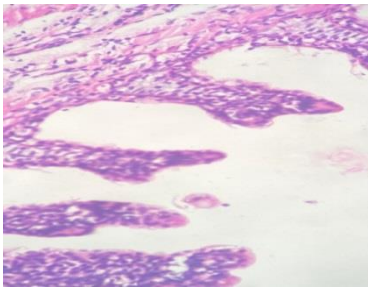


FIG NO: 17. FUSION OF LAMELLAE, DISTIL HYPERPLASIA AND EPITHELIAL LIFTING



FIGNO: 18 DEGENERATION AND NECROSIS OF LAMELLAE

0.7 ml CONCENTRATION AT 72 HOURS

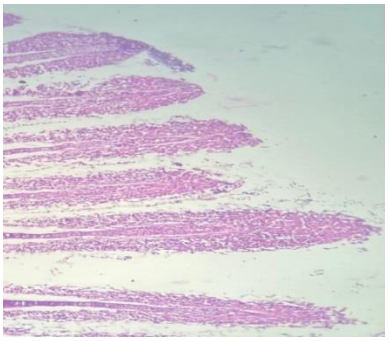


FIG NO19.DILATION OF BLOOD VESSELS, IN GILL ARCH AND DENSEINFLAMMATORY REACTION AND DEGENARATION

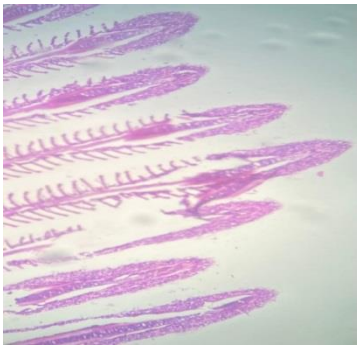


FIG NO 20 DEGENARATION AND NECROSIS

0.7 ml CONCENTRATION AT 96 HOURS

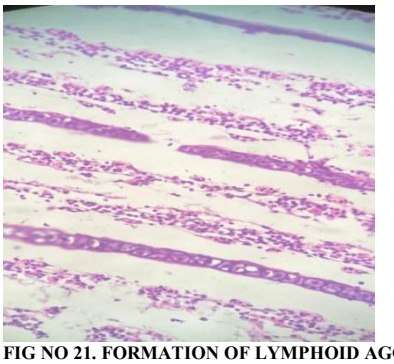


FIG NO 21. FORMATION OF LYMPHOID AGGREGATES INFLAMMATORY REACTION, NECROSIS DILATION OF BLOOD VESSELS IN GILL ARCH

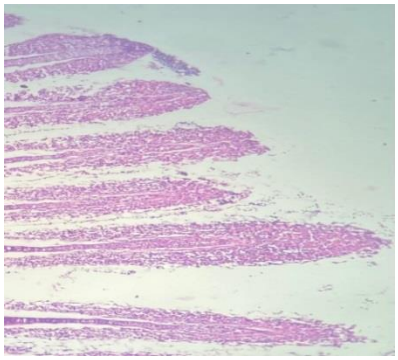


FIG NO 22. NECROSIS DILATION OF BLOOD VESSELS IN GILL ARCH

0.9 ml CONCENTRATION AT 48 HOURS

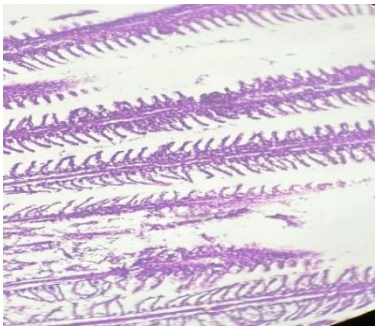


FIG NO 23.RUPTURE OF MARGINAL FILAMENTS, FORMATION OF LYMPHOID AGGREGATES INFLAMMATORY IN GILL ARCH

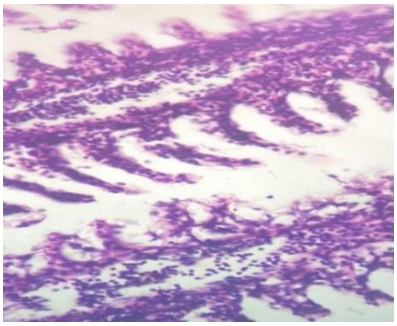


FIG NO 24.RUPTURE OF MARGINAL FILAMENTS, FORMATION REACTION OF LYMPHOID AGGREGATES INFLAMMATORY, NECROSIS DILATION OF BLOOD VESSELS,

0.9 ml CONCENTRATION AT 72 HOURS

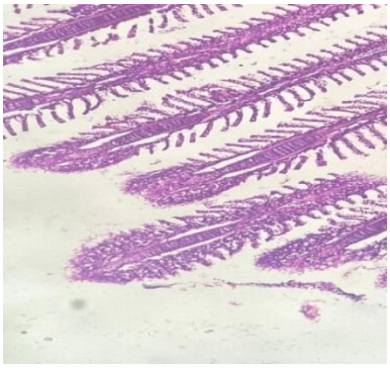


FIG NO 25.RUPTURE OF MARGINAL FILAMENT NECROSIS, DILATION OF BLOOD VESSELS IN GILL ARCH

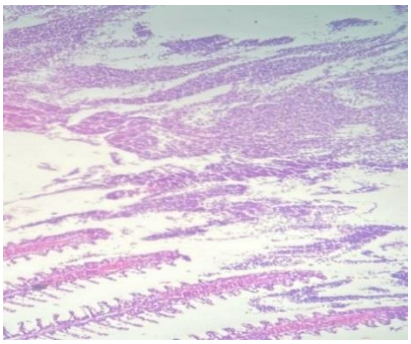


FIG NO 26. DENSE INFLAMMORATY REACTION RUPTURE OF MARGINAL FILAMENTS, NECROSIS, DILATION OF BLOOD VESSELS IN GILL ARCH

0.9 ml CONCENTRATION AT 96 HOURS

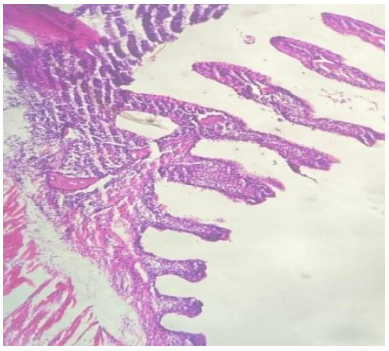


FIG NO 27. DENSE INFLAMMORATY REACTION RUPTURE OF MARGINAL FILAMENTS, NECROSIS, BLOOD VESSELS IN GILL ARCH

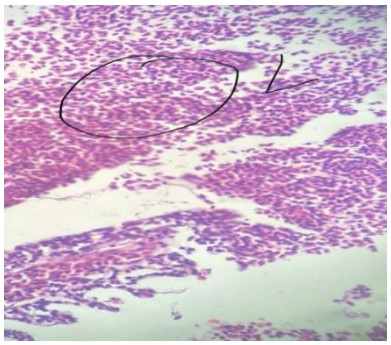


FIG NO 28. FORMATION OF LYMPHOID AGGERATES DENSE INFLAMMORATY REACTION RUPTURE OF DILATION OF MARGINAL FILAMENTS, NECROSIS, DILATION OF BLOOD VESSELS IN GILL ARC

LIVER

0.3 ml CONCENTRATION AT 48 HOURS

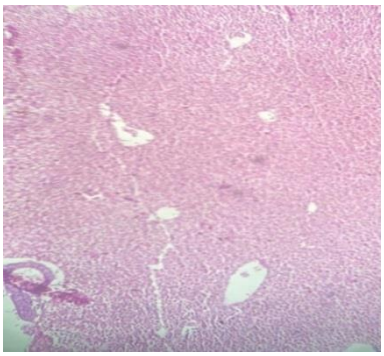


FIG NO 29. LYMPHOCYTIC INFILTRATION

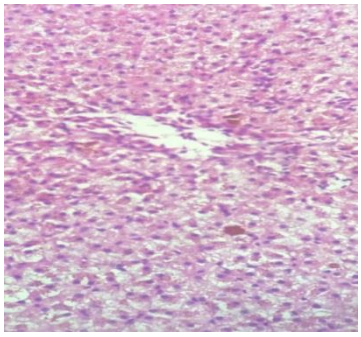


FIG NO 30. LYMPHOCYTIC INFILTRATION, CYTOPLAMIC IN AGGREGATION

0.3 ml CONCENTRATION AT 72 HOURS

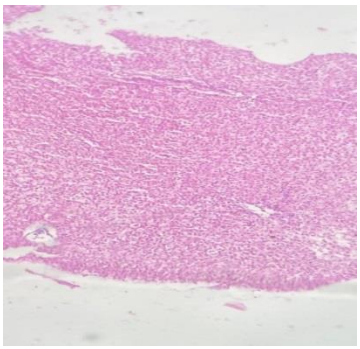


FIG NO 31. LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION).

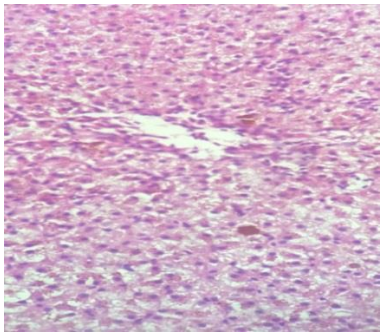


FIG NO 32. MILD FATTY CHANGES LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION

0.3 ml CONCENTRATION AT 96 HOURS

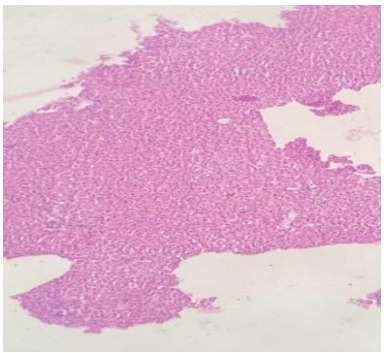


FIG NO 33 .AGGREGATION OF HEPATOCYTES LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION

.

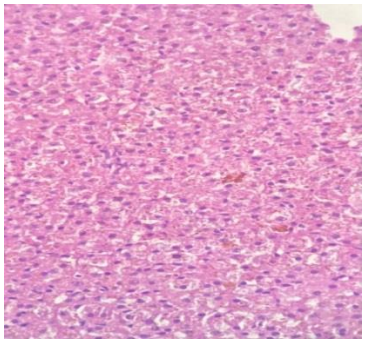


FIG NO 34. PYKNOSIS OF NUCLEI, AGGREGATION OF HEPATOCYTES LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION

0.5 ml CONCENTRATION AT 48 HOURS

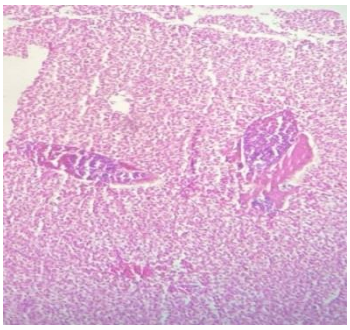


FIG NO 35. PYKNOSIS OF NUCLEI, AGGREGATION OF HEPATOCYTES LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION.

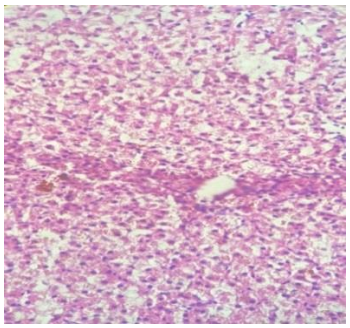


FIG NO 36. MODERATE INFLAMMATORY FATTY CHANGE PYKNOSIS OF NUCLEI, AGGREGATION OF HEPATOCYTES LYMPHOCYTIC INFILTRATION,

0.5 ml CONCENTRATION AT 72 HOURS

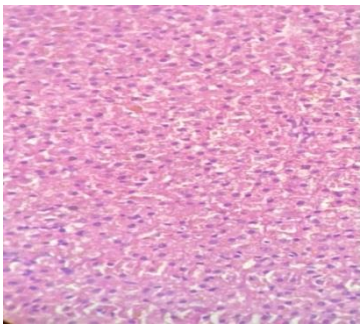


FIG NO 37. HYPERTROPY OF HEPATOCYTES MODERATE INFLAMMATORY FATTYCHANGE PYKNOSIS OF NUCLEI LYMPHOCYTIC INFILTRATION

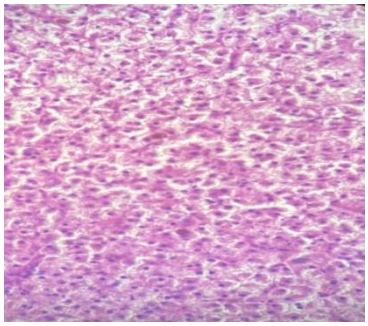


FIG NO 38. MILD TO MODERATE INFLAMMATORY FATTY CHANGES, PYKNOSIS OF NUCLEI, AGGREGATION OF HEPATOCYTES LYMPHOCYTIC INFILTRATION

0.5 ml CONCENTRATION AT 96 HOURS

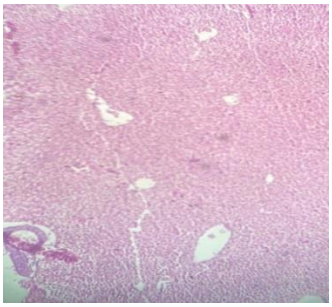


FIG NO 39. CYTOPLASMIC DEGENERATION THROMBUS FORMATION

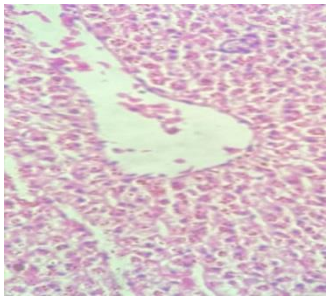


FIG NO 40. HAEMOLYSIS OF HEPATOCYTES PYKNOCYSIS OF NULECUS AND NEROSIS

0.7 ml CONCENTRATION AT 48 HOURS

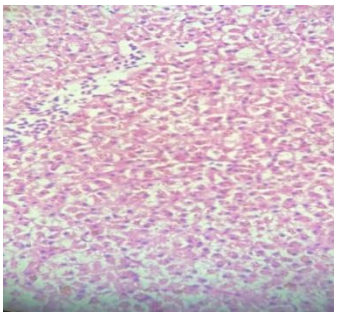


FIG NO 41. HAEMOLYSIS OF HEPATOCYTES PYKNOCYSIS OF NULECUS AND NEROSIS

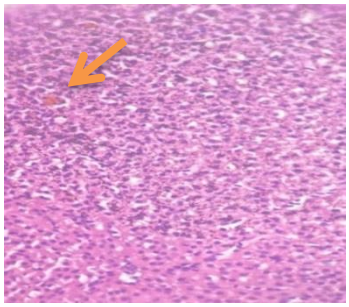


FIG NO 42. CHRONIC LIVER DAMAGE AND BILE STASIS, MODERATE INFLAMMATORYFATTYCHANGE PYKNOSIS OF NUCLEI LYMPHOCYTIC INFILTRATION

0.7 ml CONCENTRATION AT 72 HOURS

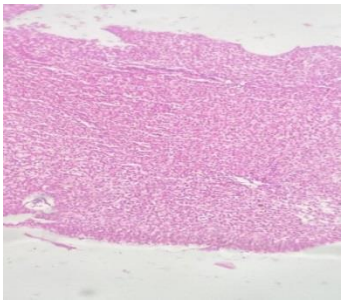


FIG NO 43. CHRONIC LIVER DAMAGE AND BILE STASIS MODERATE INFLAMMATORY FATTYCHANGE PYKNOSIS OF NUCLEI LYMPHOCYTIC INFILTRATION

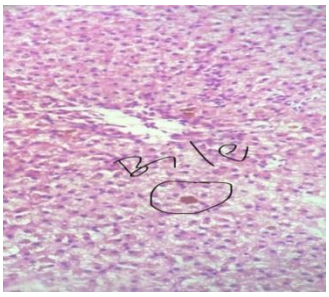


FIG NO 44. EXTENSIVE FATTY CHANGES CHRONIC LIVER DAMAGE AND BILE STASIS, MILD TO MODERATE INFLAMMATORY FATTYCHANGE PYKNOSIS OF NUCLEI LYMPHOCYTIC INFILTRATION

0.7 ml CONCENTRATION AT 96 HOURS

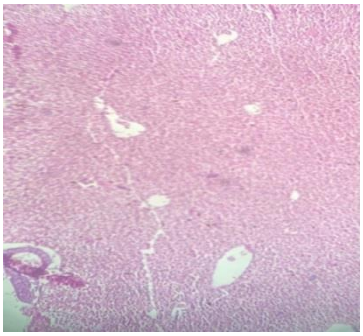


FIG NO 45. CYTOPLASMIC DEGENERATION THROMBUS FORMATION

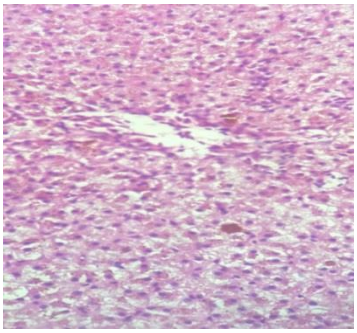


FIG NO 46. MILD FATTY CHANGES LYMPHOCYTIC INFILTRATION CYTOPLAMIC AGGREGATION

0.9 ml CONCENTRATION AT 48 HOURS

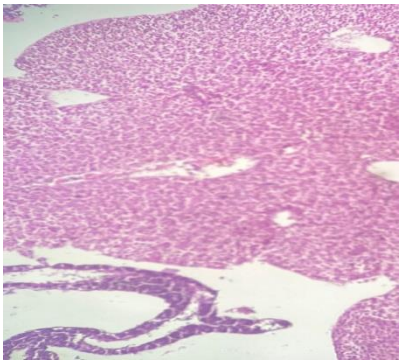


FIG NO 47.PYKNOSIS OF NUCLEI ,FOCAL NECROSIS MODERATE FATTY CHANGES LYMPHOCYTIC INFILTRATION, CYTOPLAMIC AGGREGATION

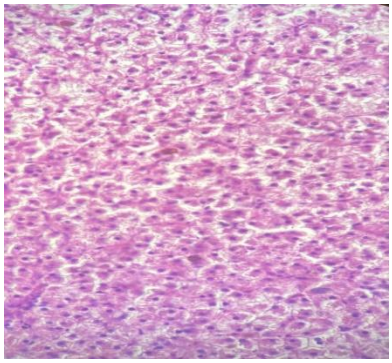


FIG NO 48. HAEMOLYSIS OF HEPATOCYTES PYKNOCYSIS OF NULECUS AND NEROSIS

0.9 ml CONCENTRATION AT 72 HOURS

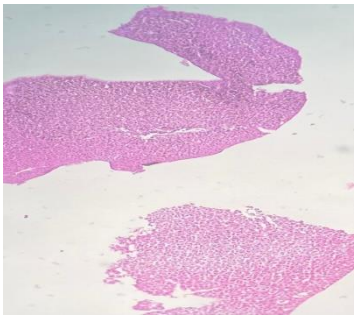


FIG NO 49. HYPERTROPY OF HEPATOCYTES PYKNOCYSIS OF NULECUS, LYMPHOCYTIC INFILTRATION AND NEROSIS *ROHITA*

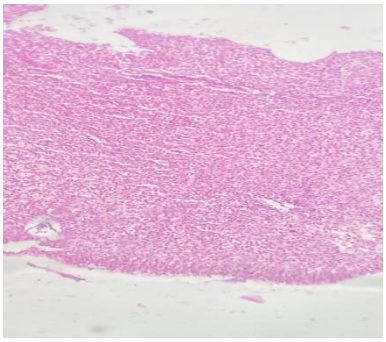


FIG NO 50. THROMBUS FORMATION, DEGENERATION HYPERTROPY OF HEPATOCYTES PYKNOCYSIS OF NULECUS LYMPHOCYTIC INFILTRATION AND NEROSIS

0.9 ml CONCENTRATION AT 96 HOURS

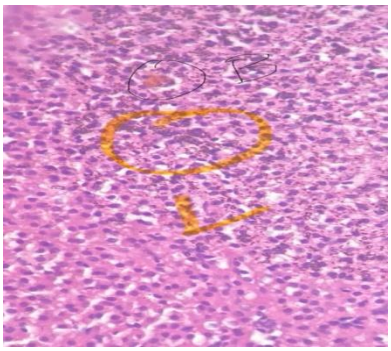


FIG NO 51. LIVER WITH BILE STASIS, PYKNOCYSIS OF NULECUS, LYMPHOCYTIC INFILTRATION AND NEROSIS

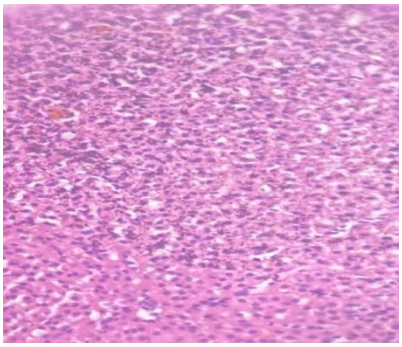


FIG NO 52. LIVER WITH BILE STASIS, PYKNOCYSIS OF NULECUS, LYMPHOCYTIC INFILTRATION AND NEROSIS

MUSCLE
0.3 ml CONCENTRATION AT 48 HOURS

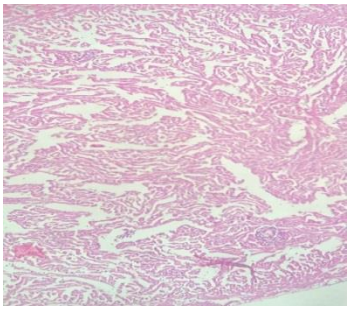


FIG NO 53. DILATION WITH IN BLOOD VESSLES, INFLAMMATORY CELLULAR AGGREGATION

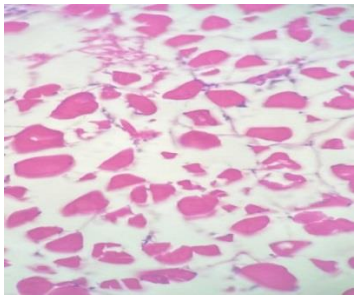


FIG NO 54.EDEMA IN EPITHELIAL CELLS, BLOOD CONGESTION INFLAMMATORY CELLULAR AGGREGATION

0.3 ml CONCENTRATION AT 72 HOURS

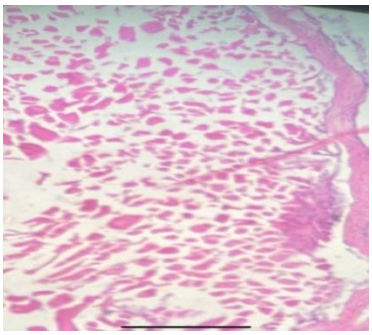


FIG NO 55.DILATION OF BLOOD VESSELS, ATROPHY MUSCULATURE, NECROTIC MUSCLE FIBRE EDEMA IN EPITHELIAL CELLS , BLOOD CONGESTION INFLAMMATORY CELLULAR AGGREGATION

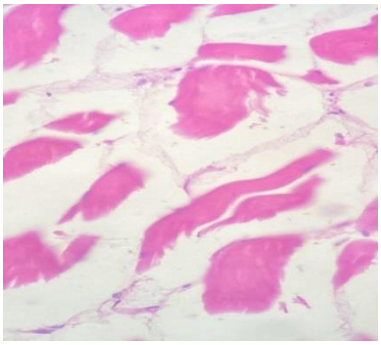


FIG NO 56.CALCIFICATION, ATROPHIC MUSCLE FIBRE NECROSIS AND SEVERE DEGENARATION

0.3 ml CONCENTRATION AT 96 HOURS

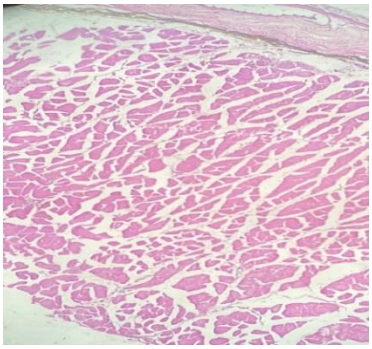


FIG NO 57.INFLAMMATORY CELL AGGEREGATION HAEMORRHAGE BETWEEN BLOOD VESSELS AND EDEMA IN EPTHILIAL CELLS

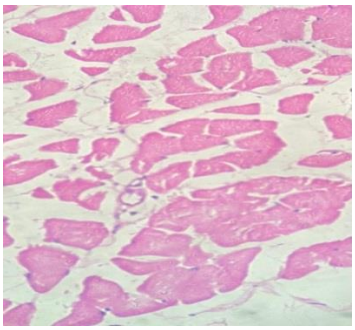


FIG NO 58. DILATION OF BLOOD VESSELS INFLAMMATORY CELL AGGEREGATION HAEMORRHAGE BETWEEN BLOOD VESSELS AND EDEMA IN EPTHILIAL CELLS

0.5 ml CONCENTRATION AT 48 HOURS

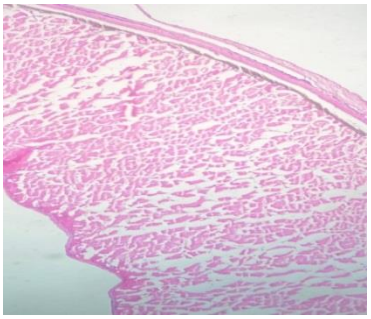


FIG NO 59.MUSCLES WITH FOCUS OF DISTROPIC CALCIFICATION

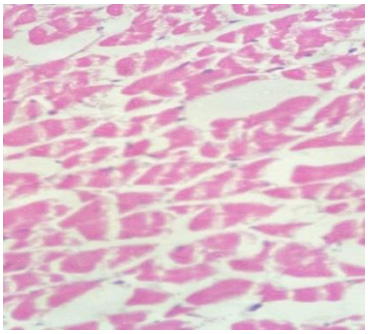


FIG NO 60. CALCIFICATION APPEARS AS A BLUISH BASOPHILIC MATERIAL AND MUSCLES FIBRES WITH DISTROPIC CALCIFICATION

0.5 ml CONCENTRATION AT 72 HOURS

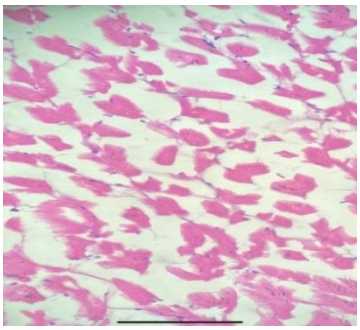


FIG NO 61. SEVERE DEGENERATION AND NECROSIS OF MUSCLE FIBRE CALCIFICATION APPEARS AS A BLUISH BASOPHILICMATERIAL

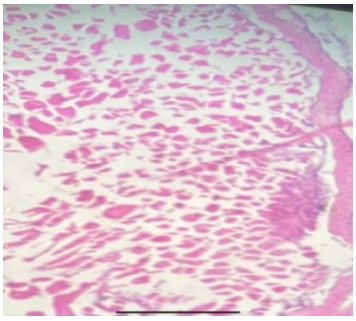


FIG NO 62.DILATION OF BLOOD VESSELS, ATROPHY MUSCULATURE NECROTIC MUSCLE FIBRE EDEMA IN EPITHELIAL CELLS , BLOOD CONGESTION INFLAMMATORY CELLULAR AGGREGATION

0.5 ml CONCENTRATION AT 96 HOURS

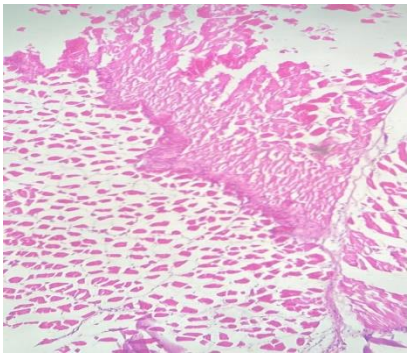


FIG NO 63. DILATED BLOOD VESSELS WITH MUSCULAR ATROPHY, NECROTIC FIBRES, EPITHELIAL EDEMA, BLOOD CONGESTION, AND INFLAMMATORY CELL AGGREGATION.

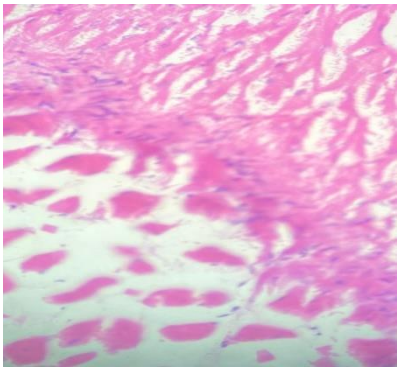


FIG NO 64. VASCULAR DILATION, MUSCULAR ATROPHY, NECROTIC FIBRES, EPITHELIAL EDEMA, CONGESTION, AND INFLAMMATORY INFILTRATION

0.7 ml CONCENTRATION AT 48 HOURS

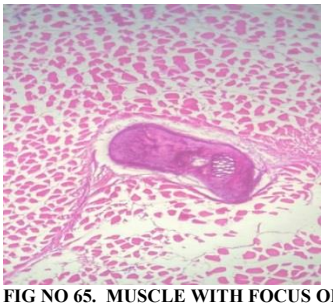


FIG NO 65. MUSCLE WITH FOCUS OF DYSTROPHIC CALCIFICATIONAPPEARS AS BASOPHILIC MATERIAL

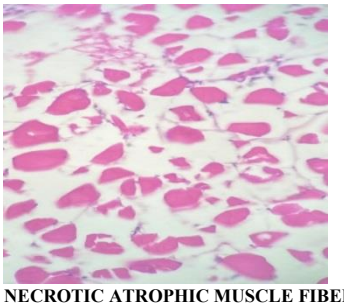


FIG NO 66. NECROTIC ATROPHIC MUSCLE FIBERS

0.7 ml CONCENTRATION AT 72 HOURS

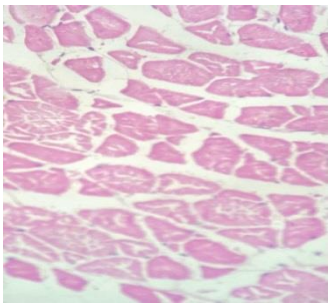


FIG NO 67.INFLAMMATORY CELL AGGREGATION, BLOOD CONGESTION EDEMA PERSISTS

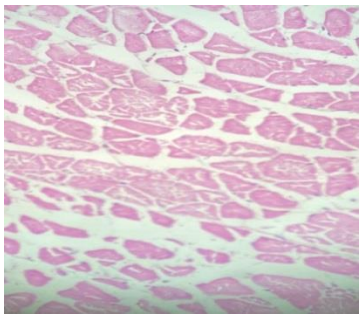


FIG NO 68. HAEMORRHAGE AND INFLAMMATORY CELL AGGREGATION, BLOOD CONGESTION EDEMA PERSISTS

0.7 ml CONCENTRATION AT 96 HOURS

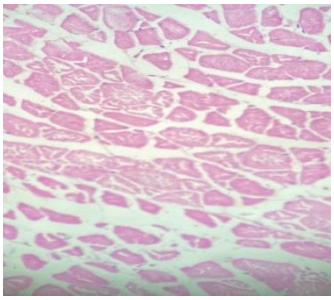


FIG NO 69. HAEMORRHAGE AND INFLAMMATORY CELL AGGREGATION, BLOOD CONGESTION EDEMA PERSISTS

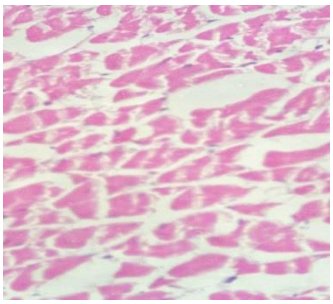


FIG NO 70. SEVERE DEGENERATION AND NECROSIS OF MUSCLE FIBRE CALCIFICATION APPEARS AS A BLUISH BASOPHILIC MATERIAL

0.9 ml CONCENTRATION AT 48 HOURS

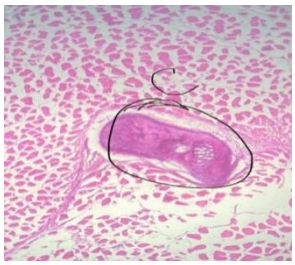


FIG NO 71. SEVERE MUSCLE FIBRE DEGENERATION WITH NECROSIS AND DYSTROPHIC CALCIFICATION, APPEARING AS BLUISH BASOPHILIC MATERIAL

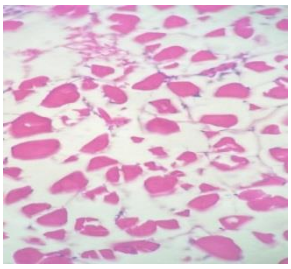


FIG NO 72. NECROTIC ATROPHIC MUSCLE FIBRE

0.9 ml CONCENTRATION AT 72 HOURS

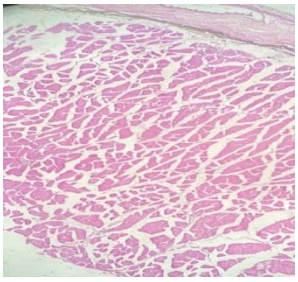


FIG NO 73.BLOOD CONGESTION, DILATION OF BLOOD VESSELS AND NECROTIC ATROPHIC MUSCLE FIBRE

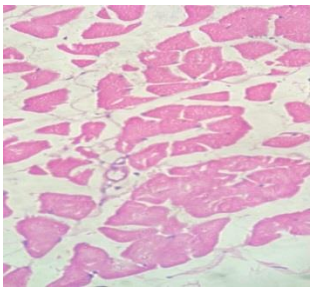


FIG NO 74. INFLAMMATORY CELL AGGEREGATION EDEMA EPITHELIAL CELLS, BLOOD CONGESTION AND DILATION OF BLOOD VESSELS

0.9 ml CONCENTRATION AT 96 HOURS

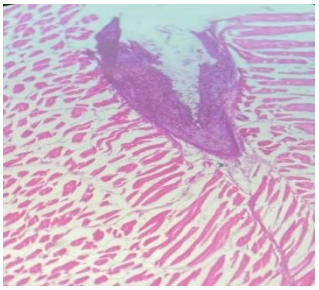


FIG NO 75. SEVERE DEGENERATION OF MYOFIBRILS, NECROSISANDDISTROPHIC CALCIFICATION AS BLUISH BASOPHILIC MATERIAL

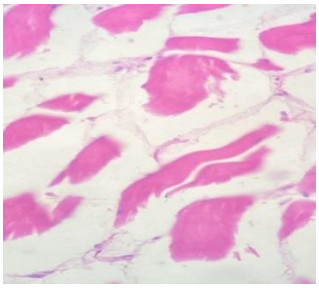


FIG NO 76. SEVERE DEGENERATION OF MYOFIBRILS NECROSIS AND DISTROPHIC CALCIFICATION

HEART

0.3 ml CONCENTRATION AT 48 HOURS

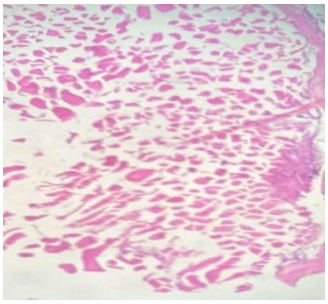


FIG NO 77. CELLULAR INFILTRATION AND MYOCARDIAL CHANGES

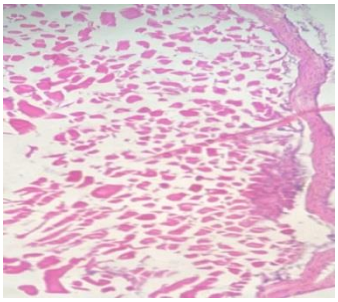


FIG NO 78. CELLULAR INFILTRATION AND MYOCARDIAL CHANGES`

0.3 ml CONCENTRATION AT 72 HOURS

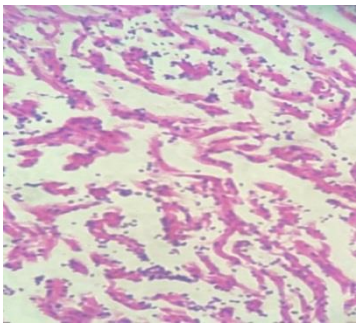


FIG NO 79. FRAGMENTED MYOFIBRILS, CELLULAR INFILTRATION

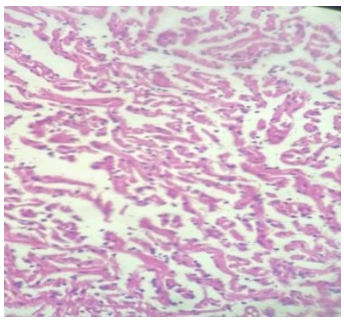


FIG NO 80. FRAGMENTED MYOFIBRILS CELLULAR INFILTRATION AND MYOCARDIAL CHANGES

0.3 ml CONCENTRATION AT 96 HOURS

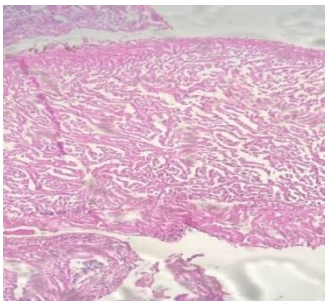


FIG NO 81.DESTRUCTED AND FRAGMENTED MYOFIBRILS AND MYOCARDIAL CHANGES

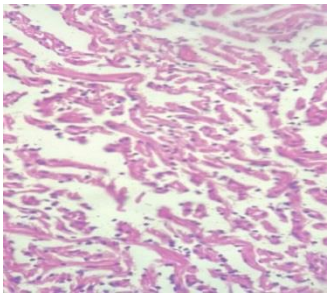


FIG NO 82. FRAGMENTED MYOFIBRILS CELLULAR INFILTRATION AND MYOCARDIAL CHANGES

0.5 ml CONCENTRATION AT 48 HOURS

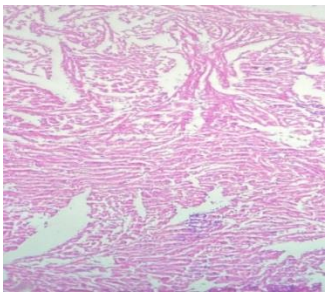


FIG NO 83. DAMAGE OF BLOOD VASULAR SYSTEM EDEMA IN PERICARIAL LAYER

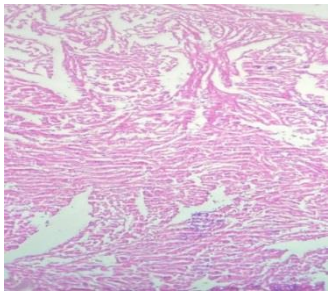


FIG NO 84. DAMAGE OF BLOOD VASULAR SYSTEM EDEMA IN PERICARIAL LAYER

0.5 ml CONCENTRATION AT 72 HOURS

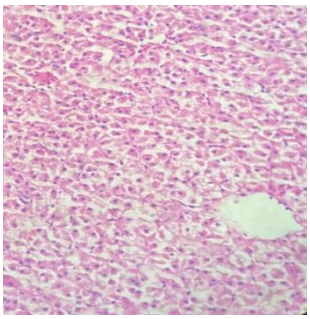


FIG NO 85. DAMAGE OF BLOOD VASULAR SYSTEM EDEMA IN PERICARIAL LAYER

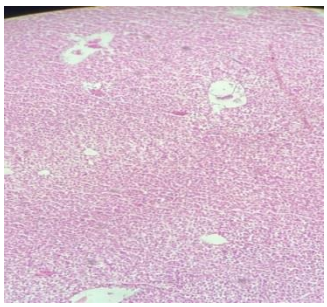


FIG NO 86. CELLURAL INFILTRATION AND FRAGMENTED MYOFIBRILS EDEMA INPERICARIAL LAYER

0.5 ml CONCENTRATION AT 96 HOURS

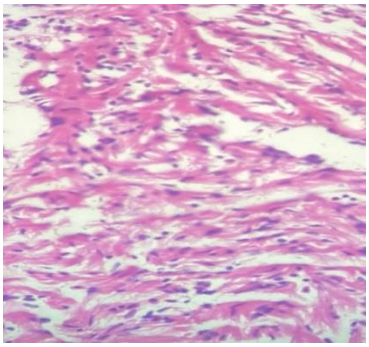


FIG NO 87. CALCIFICATION OF MYOFIBRILS AND CELLURAL INFILTRATION

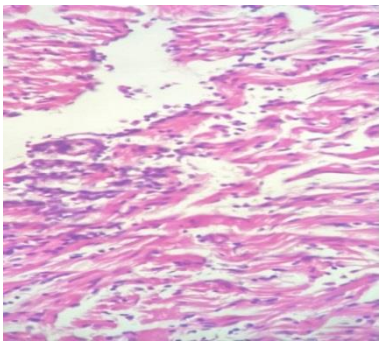


FIG NO 88. SEVERE MYOCARDIAL CHANGES AND CALCIFICATION OF MYOFIBRILS CELLURAL INFILTRATION AND EDEMA IN PERICARIAL LAYER

0.7 ml CONCENTRATION AT 48 HOURS

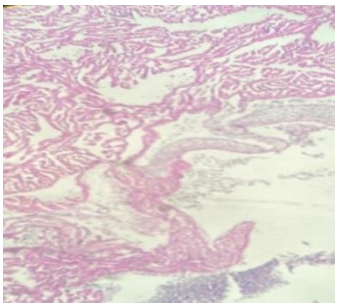


FIG NO 89. SEVERE MYOCARDIAL CHANGES AND CALCIFICATION OF MYOFIBRILS IN PERICARIAL LAYER

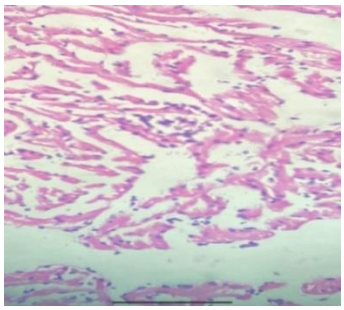


FIG NO 90 FRAGMENTED SEVERE MYOCARDIAL CHANGES AND CALCIFICATION OF MYOFIBRILS

0.7 ml CONCENTRATION AT 72 HOURS

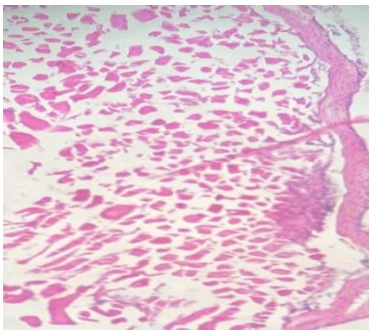


FIG NO 91. DAMAGE OF BLOOD VASULAR SYSTEM AND EDEMA IN PERICARIDAL LAYER

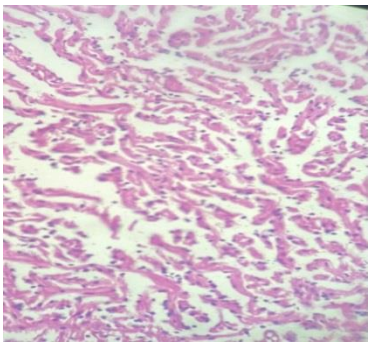


FIG NO 92. FRAGMENTED SEVERE MYOCARDIAL CHANGES

0.7 ml CONCENTRATION AT 96 HOURS

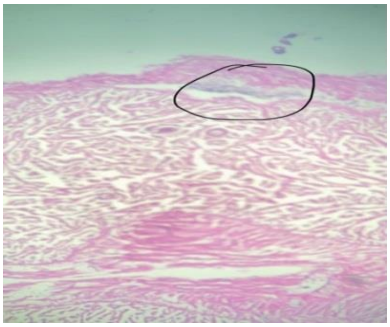


FIG NO 93. FRAGMENTED SEVERE MYOCARDIAL CHANGES AND CALCIFICATION OF MYOFIBRILS

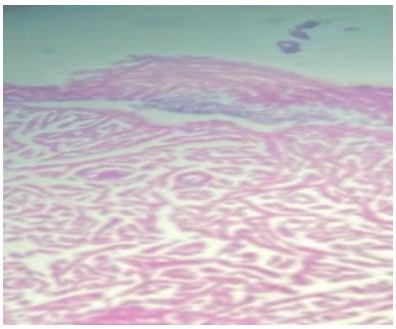


FIG NO 94. FRAGMENTED SEVERE MYOCARDIAL CHANGES AND CALCIFICATION OF MYOFIBRILS

0.9 ml CONCENTRATION AT 48 HOURS

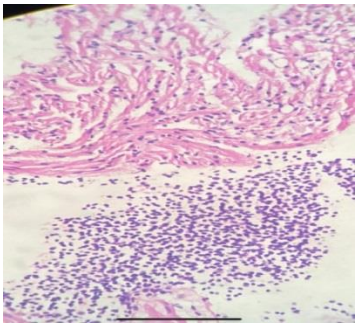


FIG NO 95. CELLULAR INFILTRATION CALCIFICATION EDEMA IN PERICARDIAL LAYER

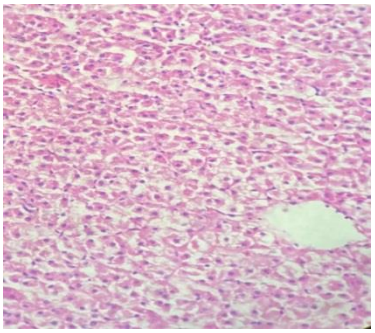


FIG NO 96. DESTRUCTION OF MYOFIBIRILS CAUSING EXTENSIVE DAMAGE

0.9 ml CONCENTRATION AT 72 HOURS

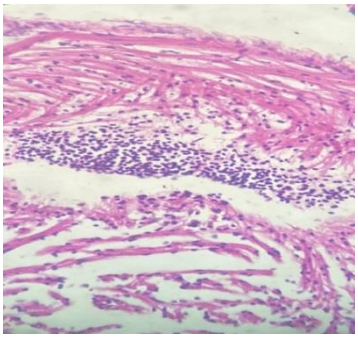


FIG NO 97. DILATION OF BLOOD VESSLES EXTENSIVE DAMAGE FRAGMENTED MYOFIBRILS AND CELLULAR INFILTRATIONS

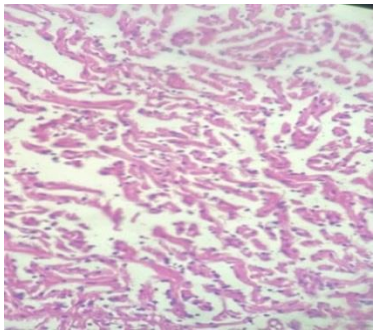


FIG NO 98. DESTRUCTION OF MYOFIBIRILS CAUSING EXTENSIVE DAMAGE

0.9 ml CONCENTRATION AT 96 HOURS

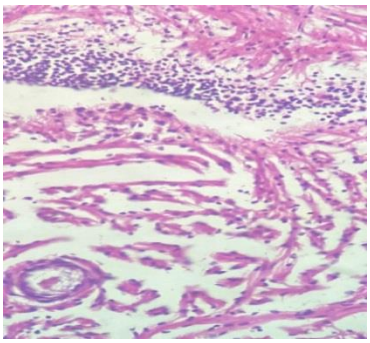


FIG NO 99. NECROSIS CALCIFICATION OF MYOFIBRILS AS BLUISH MATERIAL EDEMA IN PERICARIDAL

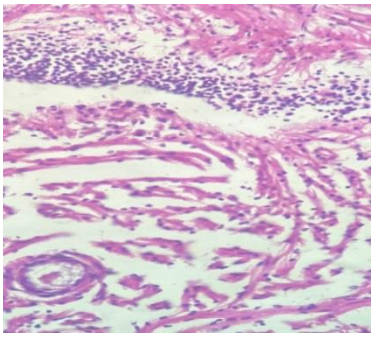


FIG NO 100. NECROSIS WITH BLUISH CALCIFICATION AND OF MYOFIBRILS, EDEMA IN THE PERICARDIAL LAYER LAYER, AND MYOFIBRIL DESTRUCTION CAUSING EXTENSIVE DAMAGE

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