Iraqi Journal of Veterinary Sciences

www.vetmedmosul.com

The correlation of the forkhead transcription factor foxo1 expression and histopathological pancreatic lesions in dogs

F.M. Abed[1], A.N. Abduljawaad[2], O.A. Al-Mahmood[3], K.H. Al-Mallah[1] and H.Kh. Ismail[1]

[1]Department of Pathology and Poultry Diseases, [2]Department of Anatomy, College of Veterinary Medicine, [3]Department of Veterinary Public Health, College of Veterinary Medicine, University of Mosul, Mosul, Iraq

Article information

Article history:
Received 30 September, 2024
Accepted 01 November, 2024
Published online 01 January, 2025

Keywords:
Dog
FOXO1
Pancreas
Immunohistochemistry
Histopathology

Correspondence:
F.M. Abed
firasabed@uomosul.edu.iq

Abstract

The objective of the current research was to characterize the correlation of Forkhead transcription factor and histopathologic findings of dogs' pancreatic lesions. The study revealed a negative linear correlation (Correlation Coefficient 0.96) between lesions frequences and FOXO1 (lesions frequences move in opposing directions). Lesion frequences reduce as FOXO1 rises. Samples collected from the necropsy unit of veterinary teaching hospital at University of Mosul, college of veterinary medicine, to determine and confirm any histopathologic evidence of specific lesions these specimens histologically examined. In dogs, pancreatic lesions can range from mild inflammation (pancreatitis) to more severe conditions such as pancreatic cancer. Focusing on histopathological changes can help identify the correlation with FOXO1 expression. Various types of lesions were observed in the 17 pancreatic samples from dogs; however, none have been showed any evidence of neoplasia. Despite a lack of gross lesions, 14 samples (82.35%) exhibited microscopic pathological changes. Three samples (17.64%) revealed normal histology. In total, 12 types of microscopic lesions were identified. Among the 14 samples, degenerative changes, congestion, edema, coagulative and fat necrosis, as well as inflammation were detected in some samples. Multiple lesions, including fibrosis, hyperplasia, and atrophy, were observed in other samples. Additionally, cyst formations, metaplasia, and granulation tissue were determined. Dogs were randomly chosen, and all pancreatic samples were assessed through histopathological and immunohistochemical evaluation. This study concludes that lesions frequences move in opposing directions. Lesion frequences increase as FOX01 descend.

DOI: 10.33899/ijvs.2025.156229.4066,

Introduction

The canine pancreatitis model has frequently been utilized because of its resemblance to the condition in humans (1). The pancreas is situated in the dorsal region of both the epigastric and mesogastric sections of the abdomen, just below the liver. Canine liver is divided into 4 lobes namely: the right lobe, the left lobe, the caudate lobe. The right lobe is thin and slender, while the left lobe is shorter, thicker, and wider. These lobes are connected at the body, which is located caudomedial to the pylorus. The right lobe extends caudally within the mesoduodenum (2). An understanding of the function of FOXO1 in pancreatic health and illness tissue can be gained by examining its expression in connection with histological pancreatic lesions in dogs. Cancer research places a lot of emphasis on FOXO1 because it is known to be involved in several cellular functions, such as metabolism, cell cycle regulation, and apoptosis (3). FOXO1, part of the Foxo1 family, is important for regulating various cellular processes such as metabolism, cell control, and stress resistance (4). In the study of pancreatic lesions in dogs, examining FOXO1 expressions can offer valuable

insights into the molecular mechanisms driving pancreatic diseases and potentially highlight new therapeutic targets (5). The relationship between the expression of the forkhead transcription factor FOXO1 and pancreatic lesions is an attractive research topic (6). Minor variations exist in the configuration of the exocrine pancreas across most mammals; nevertheless, anatomical and functional variances are evident across other members of the animal kingdom due to variations in body shape and metabolism (7). Most physiological systems across animals, including humans, are considered similar. There are, however, crucial differences in specific diseases, especially pancreatic cancer (8). The new exertions to fund large associations and exploration into pancreatic tumors are appreciated (2,9,10)

Diagnosing pancreatic diseases in dogs remains difficult due to the variability in clinical signs, which often do not align with medical pathology or histopathological outcomes. Thus, the present study was to characterize correlation of the Forkhead Transcription Factor and histopathologic findings of dogs' pancreatic lesions.

Materials and methods

Ethical approval

This study was approved by the University of Mosul's Ethical Committee for Animal Research (UoM.Dent/A.DM.L.1/22) for the period extending from November 30, 2021, to December 31, 2022.

Case selection

The study involved seventeen pancreatic specimens collected from dogs from August 2021 to February 2022 at the necropsy unit at the College of Veterinary Medicine, University of Mosul. Mixed-breed dogs were euthanized for reasons unrelated to pancreatic issues. Intense investigations were conducted into all internal organs, pancreas resected with other internal organs for further pathological studies. Dogs ranged in age from 1.8 to 10 years and weighed between 17.8 and 41.1 kg. Dogs with a history of each specimen were inspected for lesions and any variations in the pancreatic size, color, and texture, then composed, chilled, and transferred to the lab.

Fixative

This study utilized 10% Neutral-Buffered Formalin (NBF) as the fixative. The samples were collected and then immersed in the solution (11-13).

Histopathological examinations

Three blinded pathologists reviewed and graded hematoxylin and eosin-stained pancreatic slides using a published grading scheme (14,15). Each slide was examined for inflammations, pancreatic necrosis, edema, fibrosis, atrophy, and hyperplasia.

Immunohistochemistry

Samples preserved in 10% neutral buffered formalin for 48 hours and then stained using Hematoxylin and Eosin (H&E). The tissue sections underwent immunohistochemistry (IHC), Commencing with dewaxing in xylene, followed by rehydration in ethanol, and concluding with a wash in phosphate-buffered saline. Endogenous peroxidase activity was suppressed by a 3% hydrogen peroxide solution in methanol for 30 minutes. The tissue specimens were then frozen at -25℃ for one hour and then incubated with primary antibodies at 4℃ overnight (16-18). The primary antibodies involved anti-rabbit IHC of paraffin-embedded using FOX01 polyclonal antibody (Catalog No. E-AB-70144, Elabscience, USA) at dilution of 1:200. FOXO1 is a transcription factor which acts as a regulator of cell responses to oxidative stress. Quantification of IHC was achieved rendering to the following semiquantitative scores based on the proportion of positively stained cells: score 1 (≤25 % + cells), score 2 (26–50 % + cells), score 3 (51–75 % + cells), and score 4 (76–100 % + cells) (19,20).

Statistical analysis

Descriptive and inferential statistics were performed using JMP Pro16 software (2021 SAS Institute Inc., North Carolina, USA). Descriptive statistics included frequences. To determine the correlation between lesions frequences and FOX01, linear correlation was used. Results were significant with a $P<0.05$. JMP®, Pro 16. (2021). SAS Institute Inc., Cary, NC, 1989-2021 (21,22).

Results

Histopathological findings

Microscopic examination revealed typical pancreatic structures with all histological elements, including the well-defined endocrine elements (islets of Langerhans) scattered within the darker exocrine components (ducts and glands). Various types of lesions were observed in the 17 pancreatic samples from dogs; however, none have been showed any evidence of neoplasia. Despite a lack of gross lesions, 14 samples (82.35%) exhibited microscopic pathological changes. Three samples (17.64%) revealed normal histology. In total, 12 types of microscopic lesions were identified. Among the 14 samples, degenerative changes, congestion, edema, coagulative and fat necrosis, as well as inflammation were detected in some samples. Multiple lesions, including fibrosis, hyperplasia, and atrophy, were observed in other samples. Additionally, cyst formations, metaplasia, and granulation tissue were determined, Figure 1 illustrates observed lesions. Among the 14 samples, degenerative changes, congestion, and edema were seen in some sections. Other tissue sections displayed some specific lesions such as coagulative and fat necrosis, as well as inflammation.

Figure 2 reveals multiple lesions, including fibrosis, hyperplasia, and atrophy, which were observed in different pancreatic slides sections. Furthermore, frequently determined lesions were cyst formations. Additionally, several tissue sections showed metaplasia and granulation tissue. Table 1 summarizes the frequency of lesions that are seen in all examined tissue samples in ten microscopic field.

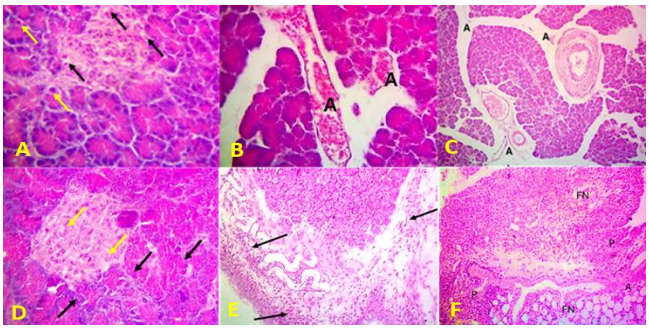

Figure 1: A panel of histopathological sections displays different types of lesions that were seen in many samples, upper left picture shows a degenerative change in both pancreatic (black arrows) and acini cells (yellow arrows) H and E 400X (A). The upper middle picture shows sever congestion of pancreatic blood vessels letter H and E 400X (B). The upper right picture reveals interlobar edema H and E 100X (C). Yellow arrows show the pancreatic cells and acini cells coagulative necrosis (black arrows) H and E 400X D. Infiltrations of inflammatory cells represented by black arrows H and E 100X (E). Fat pancreatic necrosis (FN) with well detection of purulent inflammation (p) and interlobular fibrosis A, as shown in lower right picture (E). H and E 100X.

Immunohistochemical findings

Our immunohistochemistry results matched our expectations. FOXO1 IHC expressions varied in different sections corresponding to specific detected lesions, as illustrated in figure 3 and 4. There is a negative linear correlation (Correlation Coefficient: - 0.96) between lesions frequences and FOX01 (lesions frequences move in opposing directions) ($P<0.05$). Lesion frequences reduce as FOXO1 rises. Granulation tissue, metaplasia, atrophy, cysts, fat necrosis, and coagulative necrosis had low scoring data (score 1) for the expression of the intranuclear transcription factor (FOXO1) in these pancreatic pathological findings. Inflammation, congestion, and edema showed a score 2 expression of FOXO1. Fibrosis, hyperplasia, and degenerative changes exhibited higher levels of FOXO1 expression, with a score of 3, compared to other pathological changes. Score 4 was recorded in sections with no lesions only. Figure 5 illustrates FOXO1 immunohistochemical reactions against recombinant protein corresponding to mouse FOXO1 polyclonal Antibody in pancreatic tissues sections with specific score for each picture.

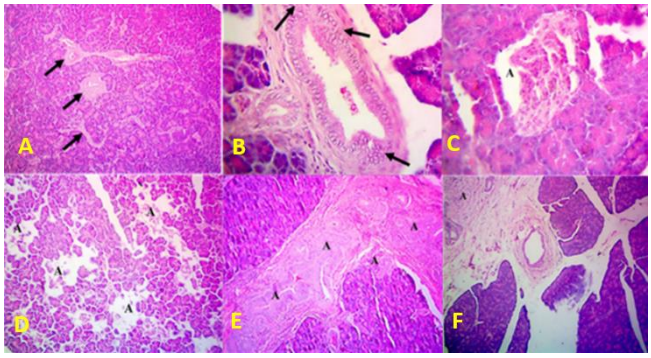

Figure 2: A panel of histopathological sections displays different types of lesions that were seen in many samples, upper left picture shows intralobular fibrosis (black arrows) H and E 100X (A). The upper middle picture shows hyperplasia of columnar epithelial cells of interlobular pancreatic duct (black arrows) H and E 400X (B). The upper right picture reveals pancreatic atrophy H and E 400X (C). Letter (A) shows the pancreatic cyst formations H and E 400X (D). Metaplasia of pancreatic tissue to fibrous tissue H and E 400X (E). Presence of granulation tissue in focal area with infiltration of monomorphic inflammatory cells and fibroblast as shown in lower right picture H and E 400X (E).

Table 1: The frequencies of lesions

No	Lesions	Frequencies
1	Granulation tissue	6
2	Metaplasia	6
3	Inflammations	3
4	Fibrosis	3
5	Hyperplasia	3
6	Atrophy	3
7	Cyst	6
8	Degenerative changes	2
9	Fat necrosis	7
10	Congestions	5
11	Coagulative necrosis	7
12	Edema	4

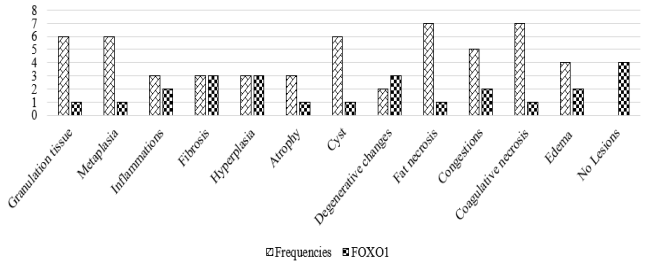

Figure 3: There is a negative linear correlation (Correlation Coefficient: - 0.96) between lesions frequences and FOXO1 (lesions frequences move in opposing directions) ($P<0.05$). Lesion frequences reduce as FOXO1 rises.

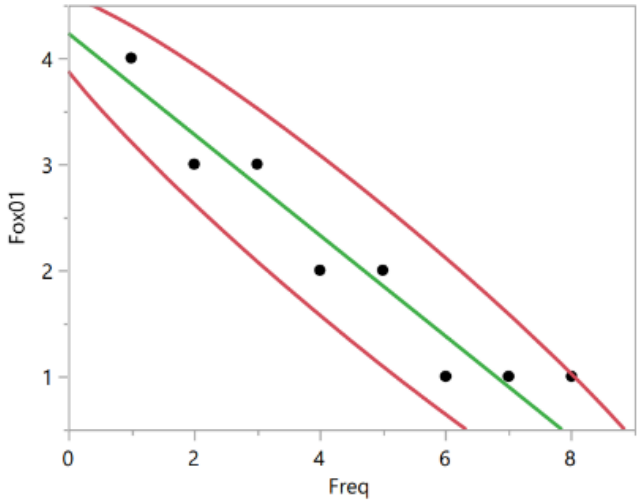

Figure 4: Lesion frequences reduce as FOX01 rises.

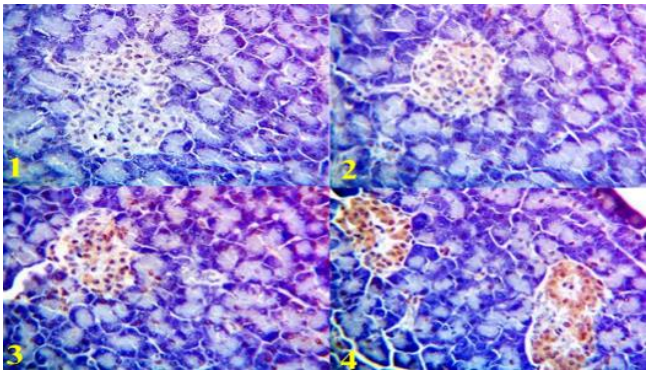

Figure 5: Our findings of immunoreactivity of (β-cells) a specialized endocrine cell located within the pancreatic islets (1) represent ≤25 % of positive cells, a weak intranuclear expression of FOXO1 (2) 26–50 % positive cells immunoreactivity, a moderate intranuclear immunoreactivity against FOXO1 polyclonal antibody (3) 51–75 % positive cells reaction of FOXO1(4)76–100 % which represent a strong positive immunohistochemical reactions of FOXO1. All images magnified at 400X.

Discussion

Diagnosing pancreatic diseases in dogs remains difficult due to the inconsistent symptoms, without constantly aligning with histopathological findings (23). Foxo1 plays an essential function in the development and development of pancreatic diseases, exclusively in the perspective of pancreatic lesions (7,24). This result is consistent with our findings in terms of sever lesions having a great impact on FOX01 expression levels. Research has demonstrated that FOXO1 plays a role in regulating pancreatic beta-cell function and offers protection against beta-cell failure caused by oxidative stress. This regulation is essential for preserving pancreatic health and preventing lesions (7). This aligns with our data, which indicates that the frequency of multiple lesions increases when low levels of FOXO1 are observed. Abnormal regulation of FOXO1 activity has been associated with metabolic issues and tissue malfunction, potentially leading to the development of pancreatic lesions (6,22) There is limited data published on medical, histopathological, and clinicopathological in dogs with chronic pancreatitis and any correlations between these aspects (25). Recently, two studies detailed specific observations in a minor group of dogs with histologically definite chronic pancreatitis. However, these studies were conducted in the UK and primarily involved English Cocker Spaniels and Cavalier King Charles Spaniels, which the authors recommended might exhibit a unique form of chronic pancreatitis (25). Severe disorders are a major risk factor for pancreatitis in both humans and dogs. It's essential to determine the underlying cause, which may involve evaluating endocrine disorders, and to manage it effectively (26). These findings highlight the challenges we faced in this study when trying to predict pancreatic abnormalities based solely on clinical investigations. Regarding fibrosis frequently observed, it may be caused by prolonged necrosis and apoptosis, as well as inflammatory reactions (27). Additionally, duct obstruction associated with hyperplasia, detected using the H&E standard staining technique, might contribute to fibrosis. These findings agree with previous interesting studies, mentioning that pancreatic fibrosis can be triggered by processes such as necrosis, apoptosis, inflammation, or duct obstruction. The initial event that induces fibrogenesis typically involves injury to the interstitial mesenchymal cells, duct cells, and/or acinar cells (28). In this context beta cell availability will be affected and foxo1 expression level and intensities is variable, the data aligns with the hypothesis that Foxo1-deficient beta cells maintain normal survival rates yet undergo dedifferentiation (29). Atrophies pancreatic tissue was detected in several sections in this study. Within pancreatic atrophy, inflammatory reaction, if it occurred, was less noticeable this pathological finding (30,27). In pancreatic acinar atrophy, the targeted destruction of enzyme-creating acinar cells results in maldigestion, which are typical markers of exocrine pancreatic deficiency (31). Differentiated cells have the capability to achieve tissue repair and regeneration. Under particular conditions, they can yet transform into other cell types. This process, known as trans differentiation, can lead to tissue metaplasia (32). Collecting pancreatic tissue samples from dogs with varying degrees of pancreatic lesions might have different scenario in terms of specific pathological changes which could be predisposing factors of Diabetes mellitus DM. This statement agrees with many histological studies on pancreatic health, injured, pregnancy, or obese adult humans and dogs have not reported significant β-cell containing with proliferative markers (32). Pancreatic metaplasia's studies incorporate the alteration of exocrine acinar cells other

nonspecific cell types morphologically, as acinar cells gradually lose zymogen granules, their apical cytoplasm diminishes while the lumen of the acini enlarges. These cells also lose their polarity and adopt cuboidal to columnar shape, resembling the ductal precursors of the embryonic pancreas during this process (33,34).

Conclusion

Even in the absence of significant signs of pancreatic abnormalities, various histopathological changes can affect pancreatic function in dogs and influence transcription factors such as FOXO1revealing a negative linear correlation (Correlation Coefficient 0.96) between lesions frequences and FOX01 (lesions frequences move in opposing directions) ($P<0.05$). Lesion frequences reduce as FOX01 rises.

Acknowledgment

We express our gratitude to the University of Mosul, College of Veterinary Medicine, Department of Pathology in accomplishing this work.

Conflict of interest

The authors have no conflicts of interest.

References

1. de Vries C, Konukiewitz B, Weichert W, Klöppel G, Aupperle-Lellbach H, Steiger K. Do canine pancreatic neuroendocrine neoplasms resemble human pancreatic neuroendocrine tumours? A comparative morphological and immunohistochemical investigation. J Comp Pathol. 2020;181:73-85. DOI: 10.1016/J.JCPA.2020.10.001
2. McGuigan A, Kelly P, Turkington RC, Jones C, Coleman HG, McCain RS. Pancreatic cancer: A review of clinical diagnosis, epidemiology, treatment and outcomes. World J Gastroenterol. 2018;24(43):4846. DOI: 10.3748/WJG.V24.I43.4846
3. Jiramongkol Y, Lam EF. FOXO transcription factor family in cancer and metastasis. Cancer Metastasis Rev. 2020;39(3):681. DOI: 10.1007/S10555-020-09883-W
4. Lu H, Huang H. FOXO1: A potential target for human diseases. Curr Drug Targets. 2011;12(9):1235-1244. DOI: 10.2174/138945011796150280
5. Liu L, Cheng Z. Forkhead Box O (FoxO) transcription factors in autophagy, metabolic health, and tissue homeostasis. In: Turksen K, editor. Autophagy in Health and Disease: Potential Therapeutic Approaches. Switzerland: Humana Cham; 2018. 47-69 p. DOI: 10.1007/978-3-319-98146-8_4
6. Buteau J, Accili D. Regulation of pancreatic β-cell function by the forkhead protein FoxO1. Diabetes Obes Metab. 2007;9(2):140-146. DOI: 10.1111/J.1463-1326.2007.00782.X
7. Son WC, Faki K, Mowat V, Gopinath C. Spontaneously occurring extra-islet endocrine cell proliferation in the pancreas of young Beagle dogs. Toxicol Lett. 2010;193(2):179–82. DOI: 10.1016/j.toxlet.2010.01.005
8. Whitley EM. Comparative Pancreatic Pathology. Whitley EM, editor. Pathobiology of Human Disease. USA: Elsevier; 2014. 1101–1123 p. DOI: 10.1016/B978-0-12-386456-7.03415-8
9. Jiang Z, Tian J, Zhang W, Yan H, Liu L, Huang Z, Lou J, Ma X. Forkhead protein FoxO1 acts as a repressor to inhibit cell differentiation in human fetal pancreatic progenitor cells. J Diabetes Res. 2017;2017(1):6726901. DOI: 10.1155/2017/6726901
10. Maejima Y, Nah J, Aryan Z, Zhai P, Sung EA, Liu T, Takayama K, Moghadami S, Sasano T, Li H, Sadoshima J. Mst1-mediated phosphorylation of FoxO1 and C/EBP-β stimulates cell-protective mechanisms in cardiomyocytes. Nat Commun. 2024;15(1):6279. DOI: 10.1038/s41467-024-50393-y
11. Al-Sabawy HB, Rahawy AM, Al-Mahmood SS. Standard techniques for formalin-fixed paraffin-embedded tissue: A Pathologist's perspective. Iraqi J Vet Sci. 2021;35:2021-2127. DOI: 10.33899/ijvs.2021.131918.2023
12. Al-Mallah KH. Pathological study of the relation between the incidences of claws lesions and carpo-digital joint capsule lesions in front limbs of sheep slaughtered in Mosul. Iraqi J Vet Sci. 2021;35(4):693-697. DOI: 10.33899/ijvs.2021.127788.1530
13. Saeed MG, Al-Hamdany E, Ismail HK. Pathological and histomorphometric study of comparative gastric ulcer induced by indomethacin, aspirin, and ethanol in rats. Iraqi J Vet Sci. 2023;37(2):339-346. DOI: 10.33899/ijvs.2022.134659.2389
14. Newman SJ, Steiner JM, Woosley K, Williams DA, Barton L. Histologic assessment and grading of the exocrine pancreas in the dog. J Vet Diagn Invest. 2006;18(1):115-118. DOI: 10.1177/104063870601800119
15. Ahmad AI, Al-Haaik AG. Age-related pancreatic tissue postnatal histogenesis in the albino rat (Rattus norvegicus): A morphological, morphometrical, and immunohistochemical study with emphasis on alpha and beta cells. Iraqi J Vet Sci. 2024;38(1):29-36. DOI: 10.33899/ijvs.2023.140777.3089
16. Al-Ayash AA, Abed FM. Impact of fixations on anti-genetic determinants of CD4, CD68, and Pax8 immunohistochemistry: A comparative study of NBF, Zink formalin, and Bouin's fluid. Iraqi J Vet Sci. 2024;38(3):623-629. DOI: 10.33899/ijvs.2024.147460.3495
17. Al-Mahmood SS, Khalil KW, Edreesi AR. Histopathology and Immunohistochemistry of tumors in animals attending veterinary teaching hospital. Iraqi J Vet Sci. 2022;36(2):2022-2309. DOI: 10.33899/ijvs.2021.130114.1733
18. Mohammed MJ, Kadhim HM. The hepatoprotective effects of the polyphenol-enriched n-butanol fraction of Cnicus benedictus against carbon tetrachloride-induced liver fibrosis in rats: In vivo study. Toxicol Rep. 2025;14:101850. DOI: 10.1016/J.TOXREP.2024.101850
19. Al-Jameel W, Al-Mahmood SS, Al-Saidya AM. Correlation between p53 and Mdm2 expression with histopathological parameters in cattle squamous cell carcinomas. Vet World. 2022;15(1):10. DOI: 10.14202/VETWORLD.2022.10-15
20. Abed FM, Rahawi AM, Al-Sabaawy HB, Dark MJ, Abduljawaad AN. Pathological and immunohistochemical findings of prostate glands from clinically normal dogs. Am J Anim Vet Sci. 2023;18(4):317-26. DOI: 10.3844/AJAVSP.2023.317.326
21. Sadeeq N, Beski S. Effect of nettle on productive performance of broilers and its biochemical, histological, immunological, and antioxidant characteristics. Mesopotamia J Agric. 2024;52(1):106-21. DOI: 10.33899/MJA.2024.144667.12971
22. Simsek N, Kaya M, Kara A, Can I, Karadeniz A, Kalkan YI. Effects of melatonin on islet neogenesis and beta cell apoptosis in streptozotocin-induced diabetic rats: An immunohistochemical study. Domest Anim Endocrinol. 2012;43(1):47-57. DOI: 10.1016/J.DOMANIEND.2012.02.002
23. Aupperle-Lellbach H, Törner K, Staudacher M, Stadler C, Tress U, Grassinger JM, Müller E, Weber CN. Histopathological findings and canine pancreatic lipase immunoreactivity in normal dogs and dogs with inflammatory and neoplastic diseases of the pancreas. J Vet Intern Med. 2020;34(3):1127-1134. DOI: 10.1111/JVIM.15779
24. Ali MA, Mustafa NG. A comparative histopathological study of pancreas, intestine, and liver of experimentally induced diabetes in rats. Iraqi J Vet Sci. 2023;37(4):971-8. DOI: 10.33899/IJVS.2023.138343.2791

25. Watson PJ, Roulois A, Scase T, Holloway A, Herrtage ME. Characterization of chronic pancreatitis in English Cocker Spaniels. J Vet Intern Med. 2011;25(4):797-804. DOI: 10.1111/J.1939-1676.2011.0744.X
26. Lim SY, Cridge H, Twedt DC, Ohta H, Nuruki T, Steiner JM. Management of acute-onset pancreatitis in dogs: A Narrative Review. J Am Vet Med Assoc. 2024;262(9):1231-1240. DOI: 10.2460/JAVMA.24.02.0107
27. Mostafa SL, Mohammed YA. Histomorphometrical and histochemical study of the pancreas on the local dogs (Canis lupus familiaris). Iraqi J Vet Sci. 2022 Oct 1;36(4):913-22. DOI: 10.33899/IJVS.2022.132567.2105
28. Klöppel G, Detlefsen S, Feyerabend B. Fibrosis of the pancreas: The initial tissue damage and the resulting pattern. Virchows Arch. 2004;445(1):1-8. DOI: 10.1007/S00428-004-1021-5
29. Talchai C, Xuan S, Lin HV, Sussel L, Accili D. Pancreatic β cell dedifferentiation as a mechanism of diabetic β cell failure. Cell. 2012;150(6):1223-1234. DOI: 10.1016/J.CELL.2012.07.029
30. Wiberg ME, Saari SM, Westermarck E. Exocrine pancreatic atrophy in German Shepherd Dogs and Rough-coated Collies: An end result of lymphocytic pancreatitis. Vet Pathol. 1999;36(6):530-541. DOI: 10.1354/VP.36-6-530
31. Wiberg ME. Pancreatic acinar atrophy in German shepherd dogs and rough-coated collies. Etiopathogenesis, diagnosis and treatment. A review. Vet Q. 2004;26(2):61-75. DOI: 10.1080/01652176.2004.9695169
32. Lardon J, Bouwens L. Metaplasia in the pancreas. Differentiation. 2005;73(6):278-286. DOI: 10.1111/J.1432-0436.2005.00030.X
33. Moshref M, Tangey B, Gilor C, Papas KK, Williamson P, Loomba-Albrecht L, Sheehy P, Kol A. Concise review: Canine diabetes mellitus as a translational model for innovative regenerative medicine approaches. Stem Cells Transl Med. 2019;8(5):450-455. DOI: 10.1002/sctm.18-0163
34. Marstrand-Daucé L, Lorenzo D, Chassac A, Nicole P, Couvelard A, Haumaitre C. Acinar-to-ductal metaplasia (ADM): on the road to pancreatic intraepithelial neoplasia (PanIN) and pancreatic cancer. Int J Mol Sci. 2023;24(12):9946. DOI: 10.3390/ijms24129946

الارتباط بين تعبير عامل نسخ فوكسو وآفات البنكرياس النسيجية المرضية في الكلاب

فراس محمد عبد[١]، احمد نزار عبد الجواد[٢]، عمر احمد عبدالله[٣]، كرم هاشم الملاح[١] و هناء إسماعيل خليل[١]

[١]فرع الأمراض وأمراض الدواجن، [٢]فرع التشريح، [٣]فرع الصحة العامة البيطرية، كلية الطب البيطري، جامعة الموصل، الموصل، العراق

الخلاصة

تم اختيار الكلاب المحلية من وحدة التشريح المرضي بشكل عشوائي وبدون انتقائية، وتم دراسة جميع عينات البنكرياس من خلال التقييم النسيجي المرضي وكيمياء النسجي المناعي. كان الهدف من هذه الدراسة هو توصيف الارتباط بين عامل نسخ فوكسو والنتائج النسيجية المرضية لآفات البنكرياس في عينات الكلاب. كشفت الدراسة عن وجود ارتباط خطي سلبي (معامل الارتباط ٠,٩٦) بين تكرار الآفات والتعبير المناعي لبروتين فوكسو (تكرار الآفات ترتبط بغلاقة عكسية مع ارتفاع التعبير البروتيني المناعي لبروتين فوكسو تحت مستوى معنوية أي أن تقل تكرار الآفات مع ارتفاع فوكسو، تم جمع العينات من وحدة التشريح في المستشفى التعليمي البيطري بجامعة الموصل، كلية الطب البيطري للتقصي عن أي آفات نسيجية مرضية ممكن تسجيلها. من المتوقع أن تتراوح آفات البنكرياس من التهاب خفيف (التهاب البنكرياس) إلى حالات أكثر شدة مثل سرطان البنكرياس. يمكن أن يساعد التقصي في إيجاد الآفات والتعرف على التغيرات النسيجية المرضية في تحديد العلاقة مع التعبير المناعي لبروتين فوكسو. لوحظت أنواع مختلفة من الآفات في عينة البنكرياس السبعة عشر والمأخوذة من الكلاب المحلية ومع ذلك، لم يظهر أي دليل على وجود آفات عيانية لتواجد الأورام حيث أظهرت ١٤ عينة (٨٢,٣٥٪) تغيرات مرضية مجهرية. في حين ثلاث عينات (١٧,٦٤٪) لم تظهر أي تغيرات نسجية تذكر. في المجموع، تم تحديد ١٢ نوعا من الآفات المجهرية. من بين العينات الأربعة عشر تم الكشف عن التغيرات التنكسية والاحتقان والوذمة والتخثر ونخر الدهون، وكذلك الالتهاب في بعض العينات. تم تسجيل آفات متعددة، بما في ذلك التليف والتضخم والضمور. بالإضافة إلى ذلك، تم تحديد تكوينات الكيس، والحؤول، والأنسجة الحبيبية. تستنتج هذه الدراسة إلى أن تكرار الآفات تتجه في علاقة ترابطية عكسية مع التعبير البروتيني المناعي لبروتين عامل النسخ فوكسو.