Veterinary Medical Journal – Giza
Faculty of Veterinary Medicine, Cairo University
(ISSN 1110 – 1423)
Accredited from National Authority for Quality Assurance and Accreditation
Giza, 12211 - Egypt

Clinical and Biochemical Studies on the role of Histamine in Gastrointestinal Disorders in Dogs.

Sarwat, M. A.; Rakha, G. H.; El-Mashad, N. E.
Department of Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Cairo University, Giza, 12211.

Abstract

Canine Gastrointestinal Disorders is fairly common encounter in canine practice. Nevertheless, investigations of new underline causes options have been limited. This study carried out for assessing the role of histamine, as a player canine gastrointestinal disorders and adverse food reaction. This is a, controlled Study over a period of 25 months, designed for evaluation the role of histamine in canine gastrointestinal disorders, 16 adult dogs suffering from canine gastrointestinal disorders inclusion criteria were diarrhea, and vomiting which had been present for at least one day. Animals showed good responses to intramuscular injection of H_1R antagonist; pheniramine maleate and consequently the gastrointestinal disorders diminished, dogs were tolerated the treatment, and there were no adverse effects. Therefore, H_1R antagonist may be an alternative treatment option for patients with canine gastrointestinal disorders.

The success rate seems to be a promising option for the future treatment of canine gastrointestinal disorders in dogs. The use of H_1R antagonists will provide an additional choice beside H_2R blockers for the therapeutic options to treat GIT disorders in dogs. Results obtained in the present study showed that serum histamine level as well as symptoms had been decreased significantly after treatment

Keywords: histamine, dog, gastrointestinal disorder, H1R antagonist, pheniramine maleate.

Introduction

Histamine is short-acting endogenous amine, which is widely allocated throughout the body, is synthesized by histidine decarboxylase (HDC), which decarboxylates the semi essential amino acid L-histidine. Originally uncovered at the beginning of the 20th century, histamine was first chemically synthesized by Windaus and Vogt in 1907, in 1910, the first biological functions of histamine were reported by Barger, and Dale, (1910); Dale, and Laidlaw, (1910). They recognized that histamine had the ability to mimic smooth muscle–stimulating and vasodepressor action observed during anaphylaxis.

Jia-Liu, et al., (2014) stated that histamine was isolated from liver and lung tissue in 1927 before purification from other tissues succeeded and gave histamine its name based on the Greek word histos, which means tissue. Jia-Liu, et al., (2014) reported that histamine, 2-[4-imidazolyl] ethylamine, is an important mediator of anaphylactic reactions. Histamine is a decarboxylation product of histidine and is released from mast cells, basophiles, and a number of other a great many other various other cell types, including normal and malignant lymphocytes. Histamine is a potent of several biologic reactions and is one of the main mediators of allergic reactions. It can also stimulate and generate acute and chronic hypersensitive responses Koyama, et al., (2009).

Histamine exerts their actions via binding to four different G necessary protein coupled receptors (H_1, H_2, H_3, and H_4) through the entire body (Parsons and Ganellin, 2006). Histamine is produced by a multitude of cell types and is involved with many physiological functions, including cellular proliferation and differentiation, hematopoiesis, embryonic development, regeneration, and wound healing (Jutel 2002; Schneider, et al., 2002; Akdis and Blaser, 2003; MacGlashan, 2003, Dy-M and Schneider 2004).

This study was carried out for assessing the histamine in canine gastrointestinal disorders.

Material and methods

Control group included (C Gp, n=8); 6 males and 2 females. Breeds are 5 Mongrel, 1 Pit-bull and 1 Labrador retriever; they range from 9 months to 8 years old with an average of 3 year old, while gastrointestinal diseased dogs (GID Gp, n=8); 3 males and 5 females. Breeds are 6 mongrels, 1 Pit-bull and 1 German shepherd. They range from 6 months to 7 years old with an average of 3.5 year old. Physical examination, complete blood count, chemical examination and serum histamine estimation by histamine ELISA test were applied.

Drugs given to animals; anti-histaminic, antibiotics, anti-inflammatory, fluid therapy, antidiarrheal, and supplements treatment.

Trade name	Manufactor y	Active principle (s)	Usage, route, dose and duration
Avil®	Sanfi-aventis	Pheniramine maleate	1st generation H_1R antagonist, I.M admistration of 1.0-1.5 ml/35 kg bwt q 12 hrs.
Augmentine®	GSK	Amoxicillin-clavulenate	Antimicrobial for digestive and respiratory problems, I.M adminstration of 12.5-25 mg/kg bwt q 6-8 hrs.
Enroxine®	Alex	Enrofloxacin	Antimicrobial for digestive and respiratory problems, I.M adminstration of 10 mg/kg bwt q 6-8 hrs.
Fucidin®	LEO	Sodium fusidate 20 mg	Antimicrobial for skin problems, topical application twice/day.
Sodium chloride® 0.9%	Almutahedo on pharma	Sodium chloride 9 gm/l (sodium & chloride 154 mmol/l each)	I.V administration of 5-6 ml/min according to fluid deficit and = (Bwt × percent of dehydration × 10) = amount given for the first 5-6 hrs, then maintenance daily dose of 60-120 ml/day according to severity of the case.
Glucose® 5%	Almutahedo on pharma	Anhydrous glucose 50 gm & water upto 1000 ml.	
Sodiumchloride®0.9% & Glucose® 5%	OTSUKA pharma	Dextrose monohydrate 0.5 gm, Sodium chloride0.9 gmin each 100 ml water + water upto 1000 ml for injection	
Ringer's solution®	Almutahedo on pharma	NaCl 147.5 mmol/l KCL 4 mmol/l CaCL2 2.25 mmol/l Total CL 156 mmol/l	I.V administration according to fluid deficit and ongoing loss of fluids (vomiting).
Flagyl®	Alex/Sanofi -aventis	Metronidazole	I.V infusion of 8-15 mg/kg bwt every 12 hrs.
Vetalgin®	Intervet	Sodium metamizole	I.M injection of 1-1.5 ml/50 kg every 12 hrs
Antinal®	AMOUN	Nifuroxazide	Oral administrations of 1 capsule/dog every 6-8 hrs.
Multisanistol®	Chemi pharm	Vitamin A, B, C, D, E, Calcium, Ferrous, and Phosphate (43.5mg/5mg)	Oral administration of 5ml/dog every 8 hrs as a food supplement.
Immulant®	MEPACO	Echancea dry extract & Nigella sativa oil	Oral administration of 5ml/dog every 8 hrs as a dietary supplement.

Results and discussion

Results of histamine values, hematological parameters, biochemical and parameters values in both control group and GIT disorders group are shown in table 1, 2, 3, 4 and graph 1.

Table (1): Pulse rate (ppm), respiratory rate/min, body temperature (C), and mucous membrane among different groups

Item	Control group (CGp, n=8)		Gastro intestinal group (GIDGp, n=8)	
	Mean	Range	Mean	Range
Pulse rate (ppm)	122.7	80 - 139	129.1	78 - 144
Respiratory rate/min.	34.2	24 - 43	38.6	26 - 44
Body temperature (C)	38.8	38.5 - 39.0 C	38.7	38.4 -39.0 C
Mucous membrane	Salmon Pink color		Pale color	

Photo (1.A) Before treatment Photo (1.B) after treatment

Photo (1.A): case no. 1 Before treatment (suffers from gastritis).

Photo (1.B): case no. 1 After treatment

Table (2): Histamine values in control group (n = 8), GID group before, and GID group after treatment in dogs with GIT disorders (n = 8).

Variable	Control (Mean ± St. error)	GID before (Mean ± St. error)	GID after (Mean ± St. error)	P value	Significance
Histamine (µg/L)	1.33 ± 0.63 [a]	3.58 ± 0.67 [b]	2.96 ± 0.67 [a, b]	0.041*	Significant

a and b refer to degree of significance difference among groups (in which a<b).

* Weak significance ($P \leq 0.05$).

Table (3): Hematological parameters' values in control group (n = 8), GID group before, and GID group after treatment in dogs with GIT disorders (n = 8).

Variable	Control (Mean ± St. error)	GID before (Mean ± St. error)	GID after (Mean ± St. error)	P value	Significance
HGB (g/dl)	13.33 ± 0.69	12.46 ± 0.64	12.23 ± 0.64	0.486	Non Significant
RBCs (×10^12/L)	7.04 ± 0.30	6.32 ± 0.30	6.61 ± 0.30	0.225	Non Significant
HCT (%)	47.800 ± 2.02	41.914 ± 2.02	42.90 ± 2.02	0.116	Non Significant
MCV (fl)	67.37 ± 1.03	66.84 ± 1.03	66.33 ± 1.03	0.776	Non Significant
MCH (pg)	18.23 ± 0.53	19.17 ± 0.56	18.41 ± 0.56	0.455	Non Significant
MCHC (g/dl)	27.74 ± 0.50 [a]	29.28 ± 0.50 b	28.45 ± 0.50 [a, b]	0.114	Non Significant
PLT (×10^9/L)	289.63 ± 28.32 [a]	397.67 ± 32.70 b	383.17 ± 32.70 [b]	0.042*	Significant
WBCs (×10^9/L)	12.70 ± 84	13.45 ± 84	12.88 ± 84	0.807	Non Significant
Lymph. (%)	19.79 ± 3.95	19.60 ± 3.69	20.90 ± 3.69	0.764	Non Significant
Mon. (%)	5.41 ± 0.39	3.38 ± 0.39	3.98 ± 0.39	0.197	Non Significant
Gran. (%)	69.94 ± 2.29	70.24 ± 3.52	70.71 ± 3.52	0.739	Non Significant
Eos. (%)	5.43 ± 2.17	6.21 ± 2.32	3.26 ± 2.32	0.100	Non Significant

a and b refer to degree of significance difference among groups (in which a<b).

Table (4): Biochemical parameters'valuese in control group (n = 8), GID group before, and GID group after treatment in dogs with GIT disorders (n = 8).

Variable	Control (Mean ± St. error)	GID before (Mean ± St. error)	GID after (Mean ± St. error)	P value	Significance
GOT (u/L)	15.29 ± 1.74 [a]	22.50 ± 1.88 [b]	20.83 ± 1.88 [b]	0.029*	Significant
GPT (u/L)	23.63 ± 6.22	23.00 ± 6.22	33.00 ± 6.22	0.458	Non Significant
Creatinine (mg/dL)	0.79 ± 0.12	0.78 ± 0.12	0.76 ± 0.12	0.990	Non Significant
Urea (mmol urea/L)	33.38 ± 2.57	39.88 ± 2.57	34.25 ± 2.57	0.177	Non Significant
Sodium (mmol/L)	149.75 ± 3.39	140.38 ± 3.39	145.75 ± 3.38	0.170	Non Significant
Potassium (mmol/L)	4.26 ± 0.29 [a]	5.19 ± 0.29 [a]	5.29 ± 0.29 [b]	0.038*	Significant

Our results agree with finding of He-SH. et al., (2004); Buhner, et al., (2009), the pathological relevance of increased histamine levels at compromised sites is less well understood in disorders, such as inflammatory bowel disease and irritable bowel syndrom, histamine might negatively or positively influence parasitic or bacterial infections (Banu, et al., 1999; Jutel, et al., 2001; Beghdadi, et al., 2008; Buhner, et al., 2009). Cells of both the innate and adaptive immune response can be regulated by histamine (Jutel, 2002; Akdis and Blaser, 2003).

Amorim et al., (2016) stated that histamine have been documented in humans with chronic GI diseases such as Crohn's disease, ulcerative colitis, irritable colon syndrome and allergic enteropathy (Xie and He, 2006; Thurmond, 2010; Smuda and Bryce, 2011).

Inside the GI tract, histamine is involved in regulation of gastric acid production, muscle motility, and mucosal ion transport (Sander et al., 2006). Histamine also has a role in mucosal defense and neurotransmission (Peters and Kovacic, 2009).

Sullivant et al., (2016) stated that histamine receptors in the differing sections of the canine gastrointestinal tract will provide additional research opportunities to further explore the role of histamine and its receptors in canine enteropathies, as well as potential therapeutic options. All 4 histamine pains were readily identified.

Amorim et al., 2016; Smudaand Bryce, 2011; Thurmond, 2010 stated that histamine has been shown to have a natural part in many functions of the gastrointestinal (GI) tract, including neurotransmission, visceral nociception, and mucosal defenses (Peters and Kovacic, 2009; Deiteren et al., 2015).

Sander, et al., (2006) mentioned that intestinal epithelial cells might be protected from pathogenic infection because of, histamine signaling epithelial skin cells subjected to histamine resulting in having lower amounts of invasive intracellular pathogens in vitro, which was mediated simply by the H_1R. Duan, (2010) suggested that protection of the gastrointestinal mucosa from pathogen invasion might be dependent, in part, on the local concentration of histamine (Feng, et al., 2007; Wu-L, et al., 2007). Low levels of histamine might be protecting, whereas higher levels might be bad for epithelial protection from infection (Ciprandi, et al., 2003; Hou, et al., 2006).

Histamine is a potent signals geber of gastric acid release. Sources of histamine in the GI tract are enterochromaffin- like cells (ECL), mast cells, and neuronal fibres. ECL cells are under both humoral and neurological regulation and are triggered by inflammation in the gastric mucosa (Repka-Ramirez and Baraniuk, 2002; Peters, and Kovacic, 2009; Jadidi-Niaragh, abd Mirshafiey, 2010).

Dogs with IBD have increased GI tract mast cell denseness, and several have high levels of urine N-methyl histamine, a metabolite of histamine (Berghoff et al., 2014). In 2008, the dog H_4 receptor was cloned, and PCR techniques were used to distinguish H_4 receptor expression in the canine small intestine (Jiang et al., 2008).

Mast cells may get involved in inflammatory processes of the intestine through the release of a variety of inflammatory mediators, such as histamine. (Le, et al., 1995; Winterkamp, 2002; He-SH, et al., 2004; Peters, and Kovacic, 2009; Kumar, and Sharma, 2010; Berghoff, 2011).

The inhibition of H_1 receptor activity by the application of two recognized histamine H_1 antagonists (triprolidine or mepyramine) reduced the ability of posterior hypothalamic explants to protect the hippocampus from KA-induced cellular death Panula, et al., (2007), histamine seems to have significant neuroprotective results. It is thus which the increased histamine level and histamine release noticed in both ischemic conditions and hibernation represent a relevant physiological response to neurological stress and anoxia, startingup the opportunity of the therapeutic use of histamine and/or histamine receptorlegends' to treat related clinical conditions Panula, et al., (2007).

Dogs have relatively few adverse results to H_1R antagonists when used at appropriate doses; nevertheless , effective clinical response in allergic disorder management is variable.

H_2R antagonists such as famotidine and ranitidine are widely used to treat peptic ulcers, and gastrointestinal bleeding in puppies and cats. In humans, H_2R antagonists are being used not just for gastroesophageal reflux disease and healing gastric, duodenal, and esophageal ulcers but also in the elimination of gastrointestinal ulcers in critically ill patients.

Conclusion

This investigation advocates that H_1R antagonist; pheniramine maleate and consequently the gastrointestinal disorders diminished, dogs were tolerated the treatment, and there were no adverse effects. Therefore, H_1R antagonist may be an alternative treatment option for patients with Canine Gastrointestinal Disorders and adverse food reaction (CAFR). Future investigation into the mechanism with controlled clinical studies using a large number of patients will be necessary to provide supporting evidence for this potential treatment. The success rate seems to be a promising option for the future treatment of Canine Gastrointestinal Disorders and adverse food reaction (CAFR) in dogs. The use of H_1R antagonists will provide an additional choice beside H_2R blockers for the therapeutic options to treat GIT disorders in dogs. Results obtained in the present study showed that serum histamine level as well as symptoms had been decreased significantly after treatment

References

Akdis CA., Blaser K., (2003): Histamine in the immune regulation of allergic inflammation. J Allergy Clin Immunol; 112:15-22.

Amorim, I., Taulescu, M. A., Day, M. J., Catoi, C., Reis, C. A., Carneiro, F., & Gärtner, F. (2016): Canine Gastric Pathology: A Review. Journal of Comparative Pathology, 154(1), 9–37.

Banu Y, Watanabe T., (1999): Augmentation of antigen receptor-mediated responses by histamine H1 receptor signaling. J Exp Med; 189:673-82.

Barger G., Dale HH., (1910): Chemical structure and sympathomimetic action of amines. J Physiol 1910; 41:19-59.

Beghdadi W, Porcherie A, Schneider BS, Dubayle D, Peronet R, Huerre M, (2008): Inhibition of histamine-mediated signaling confers significant protection against severe malaria in mouse models of disease. J Exp Med 2008; 205: 395-408.

Berghoff, N., & Steiner, J. M. (2011): Laboratory Tests for the Diagnosis and Management of Chronic Canine and Feline Enteropathies. Veterinary Clinics of North America - Small Animal Practice, 41(2), 311–328.

Berghoff, N., Hill, S., Parnell, N.K., Mansell, J., Suchodolski, J.S., Steiner, J.M., (2014): Fecal and urinary N-methylhistamine concentrations in dogs with chronic gastrointestinal disease. Vet. J. 201, 289–294.

Brown, R.E., Stevens, D.R. and Haas, H.L. (2001): The physiology of brain histamine Prog. Neurobiol. 63,637–672.

Buhner S, Li Q,Vignali S, Barbara G, De Giorgio R, StanghelliniV, (2009): Activation of human enteric neurons by supernatants of colonic biopsy specimens from patients with irritable bowel syndrome. Gastroenterology 2009; 137:1425-34.

Ciprandi G, Tosca MA, Cosentino C, Riccio AM, Passalacqua G, Canonica GW. (2003): Effects of fexofenadine and other antihistamines on components of the allergic response: adhesion molecules. J Allergy Clin Immunol 2003; 112(suppl): S78-82.

Dale HH, Laidlaw PP. (1910): The physiological action of beta-iminazolylethylamine. J Physiol; 41:318-44.

Deiteren, A., De Man, J. G., Pelckmans, P. A., & De Winter, B. Y. (2015): Histamine H4 receptors in the gastrointestinal tract. British journal of pharmacology, 172(5), 1165-1178.

Duan L, Chen X, Alexander JW. (2010): Regulatory effect of histamine on the barrier function of intestinal mucosal. J Gastrointest Surg; 14:1180-5.

Dy M, Schneider E. (2004): Histamine-cytokine connection in immunity and hematopoiesis. Cytokine Growth Factor Rev 2004; 15:393-410.

Feng BS, He SH, Zheng, PY, Wu L, Yang, PC. (2007): Mast cells play a crucial

role in Staphylococcus aureus peptidoglycan-induced diarrhea. Am J Pathol 2007;171: 537-47.

He SH. (2004): Key role of mast cells and their major secretor products in inflammatory bowel disease. World J Gastroenterol 2004; 10:309-18.

Hou YF, Zhou YC, Zheng XX, Wang HY, Fu YL, Fang ZM, et al. (2006): Modulation of expression and function of Toll-like receptor 3 in A549 and H292 cells by histamine. Mol Immunol; 43:1982-92.

Jadidi-Niaragh, abd Mirshafiey, (2010): Le Berre N, Heresbach D, Kerbaol M, et al. Histological discrimination of idiopathic inflammatory bowel disease from other types of colitis. J Clin Pathol 1995; 48(8):749–53.

Jia Liu, Lei Wang, Wenjuan Hu, Xiaoyan Chen, Dafang Zhong(2014): Development of a UHPLC–MS/MS method for the determination ofplasma histamine in various mammalian species Journal of Chromatography B, 971 (2014) 35–42

Jiang CG, Liu FR, Yu M, Li JB, Xu HM (2010): Cimetidine induces apoptosis in gastric cancer cells in vitro and inhibits tumor growth in vivo. Oncol Rep 23: 693–700.

Jutel M, Watanabe T, Akdis M, Blaser K, Akdis CA. (2002): Immune regulation by histamine. Curr Opin Immunol; 14:735-40.

Koyama, A. Takeuchi, C. Tode, M. Shimizu, I. Morita, M. Nobukawa, M.Nobukawa, N. Kobayashi, (2009): J. Chromatogr. B 877 207–212.

Kumar V, Sharma A. (2010): Mast cells: emerging sentinel innate immune cells with diverse role in immunity. Mol Immunol; 48:14–25.

MacGlashan D Jr. (2003): Histamine: a mediator of inflammation. J Allergy Clin Immunol; 112(suppl):S53-9. 1.

Parsons, M.E., Ganellin, C.R., (2006): Jan. Histamine and its receptors. British Journal of Pharmacology 147 (Suppl. 1), S127eS135

Peters, L. J., & Kovacic, J. P. (2009): Histamine: metabolism, physiology, and pathophysiology with applications in veterinary medicine. Journal of Veterinary Emergency and Critical Care, 19(4), 311–328.

Raber, J., (2007): Mar. Histamine receptor-mediated signaling during development and brain function in adulthood. Cellular and Molecular Life Sciences 64 (6), 735e741.

Repka-Ramirez, M.S., Baraniuk, J.N., (2002): Histamine in health and disease. Clinical Allergy and Immunology 17, 1e25.

Sander LE, Lorentz A, Sellge G, Coeffier M, Neipp M, Veres T, et al. (2006); Selective expression of histamine receptors H1R, H2R, and H4R, but not H3R, in the human intestinal tract. Gut 2006; 55:498-504.

Schneider E, Rolli-Derkinderen M, Arock M, Dy M. (2002): Trends in histamine research: new functions during immune responses and hematopoiesis. Trends Immunol 2002;23:255-63.

Smuda, C., Bryce, P.J., (2011): New developments in the use of histamine and histamine receptors. Curr. Allergy Asthma Rep. 11, 94–100.

Sullivant, A., Mackin, A., Pharr, T., Cooley, J., Wills, R., & Archer, T. (2016): Histamine: metabolism, physiology, and pathophysiology with applications in veterinary medicine. Veterinary Immunology and Immunopathology, 182, 29–36.

Thurmond, R.L., (2010): Histamine in Inflammation. Landes Bioscience and Springer Science and Business Media, 136 pp.

Thurmond, R.L., Desai, P.J., Dunford, P.J., et al., (2004): A potent and selective histamine H4 receptor antagonist with anti-inflammatory properties. Journal of Pharmacology and Experimental Therapeutics 309 (1), 404e413

Winterkamp S, Weidenhiller M, Otte P, (2002): Urinary excretion of N-methylhistamine as a marker of disease

activity in inflammatory bowel disease. Am J Gastroenterol; 97(12):3071–7.

Wu L, Feng BS, He SH, Zheng PY, Croitoru K, Yang PC (2007): Bacterial peptidoglycan breaks down intestinal tolerance via mast cell activation: the role of TLR2 and NOD2. Immunol Cell Biol 2007;85:538-45.

Xie Ska, H., He, S., (2005): Roles of histamine and its receptors in allergic and inflammatory bowel disease. World J. Gastroenterol. 11, 2851–2857

الملخص العربى

دراسات اكلينيكية وبيوكيمائية على دور الهستامين فى امراض الجهاز الهضمى فى الكلاب

مؤمن احمد ثروت –جمال رخا –ناجى المشد

تعد اضطرابات الجهاز الهضمي ضمن تلك الأمراض الشائعة إلى حد ما في الكلاب. ومع ذلك، فإن البحث عن أسباب جديدة مؤدية لهذه الأمراض أصبح محدودا. وتقوم هذه الدراسة على تقييم دور الهستامين فى أمراض الجهاز الهضمى فى الكلاب، وقد استغرقت الدراسة حوالى 25 شهرا باستخدام عدد 16 كلبا بالغا تعانى من اضطرابات فى الجهاز الهضمي وتتمثل هذه الاضطرابات فى حالات إسهال وتقيؤ تم تشخيصها لمدة يوم واحد على الأقل. أظهرت الحيوانات استجابات جيدة عند حقنها عضليا بعقار الفينرامين ماليات وهو أحد عقاقير الجيل الأول من مضادات الهستامين وعليه حدث تقلص لاضطرابات الجهاز الهضمي. تقبلت الكلاب العلاج بدون أى أعراض جانبية ممت يجعل استخدام عقاقير الجيل الأول من مضادات الهستامين علاجا بديلا فى حالات اضطرابات الجهاز الهضمي في الكلاب.

ويعطى معدل النجاح الذى حدث فى الدراسة مؤشرا قويا على وجود خيار واعد فى المستقبل من مضادات الهستامين متمثلا فى عقاقير الجيل الأول من مضادات الهستامين إلى جانب عقاقير الجيل الثانى من مضادات الهستامين فى علاج اضطرابات الجهاز الهضمي في الكلاب.

وقد أظهرت نتائج الدراسة الحالية أن معدلات الهستامين فى سيرم الدم وكذلك الأعراض تقلصت بشكل ملحوظ فى تلك الحالات بعد استخدام علاج الفينرامين ماليات