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Study of Nausea and Vomiting can be Symptoms of Many Different Conditions

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Abstract: ***Before the twentieth century, drugs used for the treatment of diseases were obtained from natural sources like plants, animals, microorganisms, and minerals, and among them, plants were the major source of natural drugs. At present, most of the drugs are obtained from synthetic and biosynthetic sources. The nature was once served as the source of all medicaments and plants, especially the higher plants have been continuing the service since antiquity as important sources of novel compounds useful directly as medicinal agents, as model compounds for synthetic or semi-synthetic structure modifications and optimization, as biochemical and/or pharmacological probes, and as sources of inspiration for generations of synthetic organic medicinal chemists. Plant-derived compounds which have recently under- gone development include the anti-cancer agents, taxol and camptothecin, the Chinese antimalarial drug, artemisinin, and the East Indian Ayurvedic drug, for-skolin. These and many other examples serve to illustrate the continuing value of plant-derived secondary metabolites as viable compounds for modern drug devel- opment (Newman et al. 2003; Newman and Cragg 2007). Natural sources are most primitive and abundant. Drugs obtained from the natural sources include a. Plant, b. Animal, c. Microbial, d. Marine, e. Mineral, and f. Geographical sources. Plant, Animal, Microbial and Marine may be put under common heads—the biological sources.***

Keywords: ***Morning sickness during pregnancy, Gastroenteritis (infection of your intestines) and other infections, Migraines, Motion sickness, Food poisoning Medicines, including those for cancer chemotherapy, GERD (reflux) and ulcers. Intestinal obstruction***

I. INTRODUCTION OF VOMITING

Vomiting is the forceful expulsion of stomach contents through the mouth, caused by humoral stimulation of the chemoreceptor trigger zone (CRTZ) or neural stimulation of the emetic center. The CRTZ is activated and controlled by neurotransmitter manipulation at the receptor level. Clinical signs preceding vomiting may include ptyalism, tachycardia, depression, hiding, and yawning. Gastritis, gastrointestinal ulceration, pancreatitis, motion sickness, uremia, chemo-therapy, and drug administration are common initiating causes of vomiting. This article reviews the anatomic and physiologic aspects of the vomiting reflex and its neurotransmitters, associated receptors, and rational management.

II. ANATOMY

The act of vomiting is composed of three phases: nausea, retching, and expulsion of proximal duodenal and gastric contents. Nausea is the conscious recognition of sub-conscious excitation in an area of the medulla that is closely associated with the vomiting center. This excitation is caused by irritative impulses coming from the gastroin-testinal tract, lower brain, or cerebral cortex.Ptyalism, tachycardia,nervousness, hiding or seeking attention, shivering, and yawning are all characteristic signs of nausea triggered by general activation of the sympathetic and parasympa-thetic branches of the autonomic nervous system. Hypersalivation stimulates swallowing, which stimu-lates relaxation of the gastroesopha-geal sphincter. The bicarbonate-rich saliva secreted by the salivary glands in the mouth lubricates the esopha-gus and helps neutralize the stomach's acidic environment before vomiting. 8,13 Before retching, abo-ral gastric and esophageal motility diminishes and the lower esopha-geal and pyloric sphincters relax. Retching is the second phase of vomiting and begins with the on setof a retrograde giant contraction.20,21 This contraction is a single-phase, ret-rograde, peristaltic motion that emp-ties the proximal duodenal contents into the stomach.20–22 It is followed by deep inspiratory movements, force-ful contractions of the abdominal muscles and diaphragm, and closure of the glottis. These actions produce negative intra thoracic pressure and positive intra abdominal pressure, facilitating the movement of gastric contents into the esophagus. Before expulsion, the respiratory center is inhibited and the nasopharynx and glottis close to prevent pulmonary aspiration and nasal regurgitation of the gastric contents. The third and last phase of vomiting is the expul-sion of stomach contents through the mouth.

III. ANTIHISTAMINES

Antihistamines can intercept cholinergic and histaminic nerve transmission responsible for vestibular stimulation of the vomiting center. Drugs in this classification include diphen-hydramine, dimenhydrinate, and meclizine. These drugs display H1 – antihistaminergic properties and are mainly used to control the clinical signs of motion sickness. Mild seda-tion, xerostomia, and drowsiness are some of the adverse effects. Meclizine can be terato-genic if administered at high doses. Cats do not have histamine receptors in the CRTZ, and antihistaminic drugs do not control their vomiting.

IV. SEROTONIN ANTAGONISTS

Serotonin antagonists are specific inhibitors of 5-HT-serotonergic receptors. They control vomiting by acting on receptors located on the periphery of vagal nerve terminals and centrally on the CRTZ. These receptors are normally stimulated by serotonin released from the entero chromaffin cells of the small intestine in response to damage to the gastro-intestinal mucosa. Ondansetron, a member of this class of antiemetic drugs, has been shown to control vomiting in dogs and is used in dogs receiving radiation and chemotherapy when metoclopramide and other antiemetics fail to control vomiting. Dolasetron, another member of this group, acts on recep-tors in the CRTZ. 25 Both of these drugs are used extensively in human medicine, and they seem to be safe antiemetic alternatives in veterinary medicine. However, they are not effective in controlling vomiting caused by motion sickness. Side effects of these drugs that have been reported in people include electrocardiographic changes, including PR and QT prolongation and QRS widening, that are believed to be caused by sodium channel blockage by dolasetron metabolites. Diarrhea, headache, dizziness, and musculoskeletal pain have been reported as well. These medications can be expensive.

V. OTHER DRUGS

Other drugs used to control vomiting centrally include yohimbine, diazepam, dexamethasone, propofol, and mirtazapine. Yohimbine, a pure α2 -adrenergic antagonist, is a very potent antiemetic used in dogs and cats. It may cause CNS excitement, excessive sedation, muscle tremors, tachypnea, ptyalism, and hyperemic mucous membranes. Diazepam relieves nausea and vomiting in people. Studies with animal models and clinical trials in human medicine suggest that this drug suppresses the vestibular system. The antiemetic properties of corticosteroids are incompletely understood, but their mechanism involves the activation of glucocorticoid receptors in the medulla, especially the emetic center in cats. Dexamethasone has been shown to be useful in controlling chemotherapy-associated nausea and vomiting in human patients and dogs. Propofol, an alkylphenol derivative, is used as an antiemetic in people with chemotherapy-asso-ciated nausea and vomiting that is unresponsive to serotonin antagonists or dexamethasone. It has been proposed that its antiemetic mechanism involves reduction of the serotonin concentration in the CRTZ via γ-aminobutyric acid activity and 5-HT3 serotonin receptor antagonism. Mirtazapine is a piperazinoazepine drug used as an antidepressant in people. It antagonizes central presynaptic α2 -receptors and blocks serotonin receptors.50 It is a weak 5-HT1 sero-tonin receptor antagonist, a potent 5-HT2 and 5-HT3 serotonin receptor antagonist, and an H1-histamine antagonist.50 It is used to control chemotherapy-associated nausea and vomiting in humans and, more recently, in small animals.

TABLE 1 Vomiting Reflex Components, Receptors, and Controlling Neurotransmitters and Medications

Receptor	Receptor Agonists	Receptor Antagonists		
Chemoreceptor trigger zone				
D_2-Dopaminergic	Dopamine	▸ Metoclopramide ▸ Trimethobenzamide ▸ Chlorpromazine	▸ Prochlorperazine ▸ Acetylpromazine	▸ Haloperidol ▸ Droperidol
M_1-Cholinergic	Acetylcholine	▸ Propantheline ▸ Isopropamide ▸ Prochlorperazine	▸ Chlorpromazine ▸ Scopolamine	▸ Methscopolamine ▸ Acetylpromazine
H_1-Histaminergic	Histamine	▸ Diphenhydramine ▸ Dimenhydrinate ▸ Meclizine	▸ Prochlorperazine ▸ Chlorpromazine	▸ Acetylpromazine ▸ Promethazine
α_2-Adrenergic	Norepinephrine	▸ Prochlorperazine ▸ Chlorpromazine	▸ Yohimbine ▸ Acetylpromazine	
5-HT_3-Serotonergic	Serotonin	▸ Ondansetron ▸ Dolasetron	▸ Mirtazapine ▸ Propofol	▸ Metoclopramide ▸ Granisetron
$ENK_{\mu,\delta}$-Enkephalinergic	Met-enkephalin Leu-enkephalin	▸ Butorphanol		
Neurokinin-1 antagonist	Substance P	▸ Maropitant		
Emetic center				
5-HT_{1A}-Serotonergic	Serotonin	▸ Diphenhydramine	▸ Dimenhydrinate	▸ Meclizine
α_2-Adrenergic	Norepinephrine	▸ Prochlorperazine	▸ Chlorpromazine	▸ Yohimbine
Glucocorticoid receptors	Dexamethasone	▸ Cyproterone	▸ Mifepristone	
Neurokinin-1 antagonist	Substance P	▸ Maropitant		
Vestibular apparatus				
M_1-Cholinergic	Acetylcholine	▸ Propantheline ▸ Isopropamide ▸ Prochlorperazine	▸ Chlorpromazine ▸ Scopolamine	▸ Methscopolamine ▸ Acetylpromazine

VI. TREATMENT OF COMMON VOMITING CONDITIONS

Box 1 lists several conditions and diseases that commonly cause vomiting. Gastritis or Gastric Ulceration Treatment to manage vomiting caused by gastritis or gastric ulceration must include proper fluid therapy and gastric mucosal protection. Many clinicians use broad-spectrum antiemetics because they cover local and peripheral receptors. Chlorpromazine, serotonin antagonists, and metoclopramide are good options. Maropitant seems to work extremely well in dogs. If vomit-ing is associated with gastrointestinal ulceration due to NSAID administration, therapy with misoprostol, a prostaglandin E1 (PGE) analog, may be effective in controlling both the ulcerative lesion and vomiting as a secondary problem. Proton pump inhibitors and H2 –histamine antagonists provide more complete inhibition of gastric acid secretion in severe cases of ulceration. If Helicobacter spp are the underlying cause of ulceration, appropriate antibiotic

therapy and antacids should relieve the clinical signs of the infection. Patients with neoplastic diseases often have gastrointestinal ulceration. Mast cell tumors of any stage, grade, and size can cause vomit- ing in dogs by increasing the plasma hista-mine concentration. Histamine acts on the CRTZ and the gastric mucosa. Mast cell tumor ulceration and its effects are treated with H 2 - histamine antagonists. Tumor size and his-tamine release in dogs are controlled with the administration of corticosteroids.

TABLE 2 Most Common Antiemetics Used in Small Animal Medicine

Drugs	Site of Action	Dosage	Side Effects
α_2-Adrenergic antagonists			
Prochlorperazine[2,13]	CRTZ and emetic center	**Dogs and cats:** 0.1–0.5 mg/kg SC or IM q6–8h	Hypotension, sedation
Chlorpromazine[2,7,13]	CRTZ and emetic center	**Dogs:** 0.1–0.5 mg/kg SC, IM, or IV q6–8h **Cats:** 0.2–0.5 mg/kg SC, IM, or IV q6–8h	Hypotension, sedation
Yohimbine[2,7]	CRTZ and emetic center	**Dogs:** 0.25–0.5 mg/kg SC or IM q12h	Hypotension, sedation
D_2-Dopaminergic antagonists			
Metoclopramide[2,7,13]	CRTZ, GI smooth muscle	**Dogs:** 0.1–0.4 mg/kg PO, SC, or IM q6h **Cats:** 0.2–0.4 mg/kg PO, or SC q6–8h **CRI:** 1–2 mg/kg/day	Extrapyramidal signs, constipation
Trimethobenzamide[2,13]	CRTZ	**Dogs:** 3 mg/kg IM q8–12h	Allergic reactions
H_1-Histaminergic antagonists			
Diphenhydramine[2,7,13]	CRTZ	**Dogs and cats:** 2–4 mg/kg PO or IM q8h	Sedation, GI effects
Dimenhydrinate[2,7,13]	CRTZ	**Dogs and cats:** 4–8 mg/kg PO q8h	Sedation, GI effects
Meclizine[2]	CRTZ	**Dogs and cats:** 4 mg/kg PO q24h	Sedation, xerostomia, tachycardia
M_1-Cholinergic antagonists			
Propantheline[2]	Parasympathetic nervous system	**Dogs and cats:** 0.25 mg/kg PO q8h	Gastric retention, ileus, tachycardia
Isopropamide[2]	Parasympathetic nervous system	**Dogs and cats:** 0.2–0.4 mg/kg PO q8–12h	Gastric retention, ileus, tachycardia
5-HT_3-Serotonergic antagonists			
Ondansetron[2,7]	CRTZ and vagal afferent neurons	**Dogs:** 0.11–0.176 mg/kg slow IV push q24h **Cats:** 0.1–0.15 mg/kg slow IV push q24h	Sedation
Dolasetron[2]	CRTZ	**Dogs:** 0.6 mg/kg IV q24h or 0.5 mg/kg PO, SC, or IV q24h **Cats:** 0.6 mg/kg IV q12h or 0.6–1 mg/kg PO q12h	Electrocardiogram changes
Mirtazapine[2]	CRTZ and vagal afferent neurons	**Dogs:** 0.6 mg/kg PO q24h, not to exceed 30 mg/day **Cats:** 3–4 mg/cat PO q72h	Sedation, ataxia, depression, vocalization
NK_1-Neurokinin antagonist			
Maropitant[40]	CRTZ and emetic center	**Dogs:** 1 mg/kg SC q24h up to 5 days or 2 mg/kg PO q24h up to 5 days	Injection site soreness, ataxia, anorexia, diarrhea
5-HT_4-Serotonergic antagonist			
Cisapride[2,7]	Myenteric neurons	**Dogs:** 0.1–0.5 mg/kg PO q8h **Cats:** 0.1–1.0 mg/kg or 5 mg (total dose) PO q8–12h	None
Motilin agonist			
Erythromycin[2,7]	GI smooth muscle	**Dogs and cats:** 0.5–1.0 mg/kg IV q8h, up to 5.0 mg/kg PO q8h	Vomiting at antimicrobial doses (15 mg/kg tid)
Opioid			
Butorphanol[4,13]	Emetic center	**Dogs:** 0.2–0.4 mg/kg IM 30 min before cisplatin infusion	Sedation, ataxia, anorexia, diarrhea
Others			
Propofol[2]	CRTZ	None reported in veterinary medicine	Apnea, hypotension, seizurelike signs
Dexamethasone[13]	Emetic center, medulla	**Dogs:** 0.1 mg/kg SC or IV before chemotherapy	GI ulceration
Diazepam[2]	Vestibular system suppression	0.1–0.2 mg/kg PO q6h	Sedation

VII. PANCREATITIS

Pancreatitis causes ileus due to intestinal inflammation, resulting in direct afferent input to the vomiting center. Metoclopramide is the most common antiemetic used in these patients because it acts centrally and peripherally. In dogs, phenothiazines, 5-HT3 – serotonergic antagonists, and maropitant can be useful if metoclopramide fails to control vomiting Motion Sickness Motion sickness, or kinetosis, is generated from the vestibular apparatus. Studies in humans have revealed that motion sickness is caused by three mechanisms: (1) conflicting inputs from the visual and vestibular systems; (2) conflict-ing inputs from the two vestibular systems (the semicircular canals and the otolith organs); or (3) comparison of input from these systems with the individual's expectations derived from previous experiences. Vomiting caused by motion sickness involves M1 –cholinergic and H1 -histaminergic receptors,2,11 and treatment should antagonize both receptors. Phenothiazines like chlorpromazine and prochlorperazine can antagonize both receptors at the same time, but diphenhydramine, dimenhydrinate, cyclizine, meclizine, and promethaz- ine are H1 -histamine blocking agents only, and they should be combined with a M1 –cholinergic receptor blocker for effective control of emetic signals originating from the vestibular apparatus. Maropitant prevents kinetosis in dogs by blocking the final common pathways of the vomiting reflex, including signals from the vestibular system.40 Scopolamine is a muscarinic M1 -cholinergic antagonist used to treat motion sickness, but results are not consistent.

Emetic center				
5-HT$_{1A}$-Serotonergic	Serotonin	▸ Diphenhydramine	▸ Dimenhydrinate	▸ Meclizine
α_2-Adrenergic	Norepinephrine	▸ Prochlorperazine	▸ Chlorpromazine	▸ Yohimbine
Glucocorticoid receptors	Dexamethasone	▸ Cyproterone	▸ Mifepristone	
Neurokinin-1 antagonist	Substance P	▸ Maropitant		
Vestibular apparatus				
M$_1$-Cholinergic	Acetylcholine	▸ Propantheline ▸ Isopropamide ▸ Prochlorperazine	▸ Chlorpromazine ▸ Scopolamine	▸ Methscopolamine ▸ Acetylpromazine
H$_1$-Histaminergic	Histamine	▸ Diphenhydramine ▸ Dimenhydrinate ▸ Meclizine	▸ Prochlorperazine ▸ Chlorpromazine ▸ Diazepam	▸ Cyclizine ▸ Promethazine
Vagal afferents				
5-HT$_3$-Serotonergic	Serotonin	▸ Ondansetron ▸ Dolasetron	▸ Mirtazapine ▸ Metoclopramide	▸ Granisetron
Neurokinin-1 antagonist	Substance P	▸ Maropitant		
Vagal efferents				
D$_2$-Dopaminergic	Dopamine	▸ Metoclopramide ▸ Trimethobenzamide ▸ Chlorpromazine	▸ Prochlorperazine ▸ Acetylpromazine	▸ Haloperidol ▸ Droperidol
5-HT$_4$-Serotonergic	Cisapride Metoclopramide Serotonin	▸ Piboserod		
M$_2$-Cholinergic	Acetylcholine	▸ Propantheline ▸ Isopropamide ▸ Prochlorperazine	▸ Chlorpromazine ▸ Scopolamine	▸ Methscopolamine ▸ Acetylpromazine
Motilin	Erythromycin Motilin	—		

VIII. UREMIA

Uremic toxins cause decreased gastrin clear-ance and irritate the gastrointestinal mucosa, resulting in ulcerative lesions and gastritis. When these toxins cross the blood–brain barrier, they stimulate central and peripheral receptors and activate D2 -dopaminergic recep-tors in the CRTZ. Dopamine antagonists like metoclopramide and chlorpromazine effectively block these receptors. Diuresis with appropriate fluid therapy and a proton pump inhibitor or H 2 –histaminergic antagonist helps relieve uremia by diminish-ing the secretion of hydrogen ions into the stomach, providing protection and promoting mucosal healing.

IX. GASTROINTESTINAL MOTILITY DISORDERS

Prokinetics—cisapride, metoclopramide, and erythromycin—should be used to control vom-iting due to nonobstructive delayed gastric emptying. These drugs exert their effects on different receptors. Cisapride, the most effective prokinetic agent available,11 lacks direct anti- emetic effects but stimulates 5-HT4 –serotonergic receptors. Metoclopramide's antagonism of D2 -dopaminergic receptors enables it to stimu-late motility in areas where these receptors are present (the higher gastrointestinal tract, lower esophageal sphincter, stomach.

Motilin agonist			
Erythromycin[a,2]	GI smooth muscle	**Dogs and cats:** 0.5–1.0 mg/kg IV q8h, up to 5.0 mg/kg PO q8h	Vomiting at antimicrobial doses (15 mg/kg tid)
Opioid			
Butorphanol[a,13]	Emetic center	**Dogs:** 0.2–0.4 mg/kg IM 30 min before cisplatin infusion	Sedation, ataxia, anorexia, diarrhea
Others			
Propofol[a]	CRTZ	None reported in veterinary medicine	Apnea, hypotension, seizurelike signs
Dexamethasone[13]	Emetic center, medulla	**Dogs:** 0.1 mg/kg SC or IV before chemotherapy	GI ulceration
Diazepam[a]	Vestibular system suppression	0.1–0.2 mg/kg PO q6h	Sedation

[a]Plumb DC. *Veterinarian Drug Handbook*. 6th ed. Ames, IA: Wiley-Blackwell; 2008.
[b]Richter KP. Treating acute vomiting in dogs and cats. *Vet Med* 1992;87(8):814-818.

X. UNDETERMINED ETIOLOGY

Patients with vomiting of undetermined etiology must be treated with the safest approach pos-sible once systemic diseases (e.g., liver disease,renal disease, endocrine disease) have been ruled out. Patients that are uncomfortable from excessive vomiting or are at high risk for aspira-tion pneumonia and have not been exposed to a toxic agent should be treated with antiemetics when available. α2 -Adrenergic antagonists and D2 -dopaminergic receptors are first-line anti-emetics. Maropitant is a good alternative not only because it seems to block impulses in the final common pathways of the vomiting reflex but also because it is administered once daily, dogs seem to tolerate it fairly well, and, so far, adverse effects are minimal. 5-HT3 –serotonergic antagonists have become very popular over the past few years and have good results. The addi-tion of other drugs to antiemetic therapy should be considered if vomiting becomes refractory in these patients

XI. CONCLUSION

1) *Dry Heaves:* Dry heaves are the type of vomit where the feeling is present, but nothing comes out. It is termed as non-productive vomit.
2) *Blood-Streaked Vomit:* Blood streaked vomiting causes a cut in the esophagus or stomach. Here, vomit comes out filled with blood, having red or dark-brown colour.
3) *Coffee-Ground Vomit:* Intestinal bleeding in the upper-gastro results in exposure of iron in the blood to the stomach acid. This acid oxides the iron and causes the vomit to resemble coffee grounds. It could be a sign of health issues like GERD, peptic ulcer, liver disease, etc.
4) *Bile-Stained Vomiting:* Bile vomiting is persistent vomiting following a meal. Moreover, bile reflux and bowel obstruction result in this vomit. Bile blends with food in the duodenum and when the small intestine is blocked, its substances expulse with the bile. Furthermore, following surgical removal of a gallbladder, gastric bypass surgery, etc. this reflux of bile takes place in the stomach and leads to this vomit.
5) *Faecal Vomit:* This type of vomiting occurs due to intestinal obstruction, or unusual connection between colon and stomach. As a result, an expulsion of fully or partially digested matter happens via the mouth.
6) *Projectile Vomiting:* Here, the contents of the stomach comes out in a massive force. Typically, it occurs following feeding.

XII. TREATMENT OF VOMITING

There are various vomiting treatments; with one-time vomiting typically considered to be quite serious. However, following an incident of vomiting, hydration is essential. Thus, individuals must consume plenty of water since they contain the necessary electrolytes to help in this situation.

Moreover, solid food can upset a sensitive stomach at this point. Thus, it is better to avoid such food at this point. Furthermore, medication can help to reduce or control vomiting. Apart from these, alternative remedies like consuming ginger, lemongrass oil, or bergamot are an effective vomiting solution. Additionally, dietary changes can also help in case of repeated vomiting.

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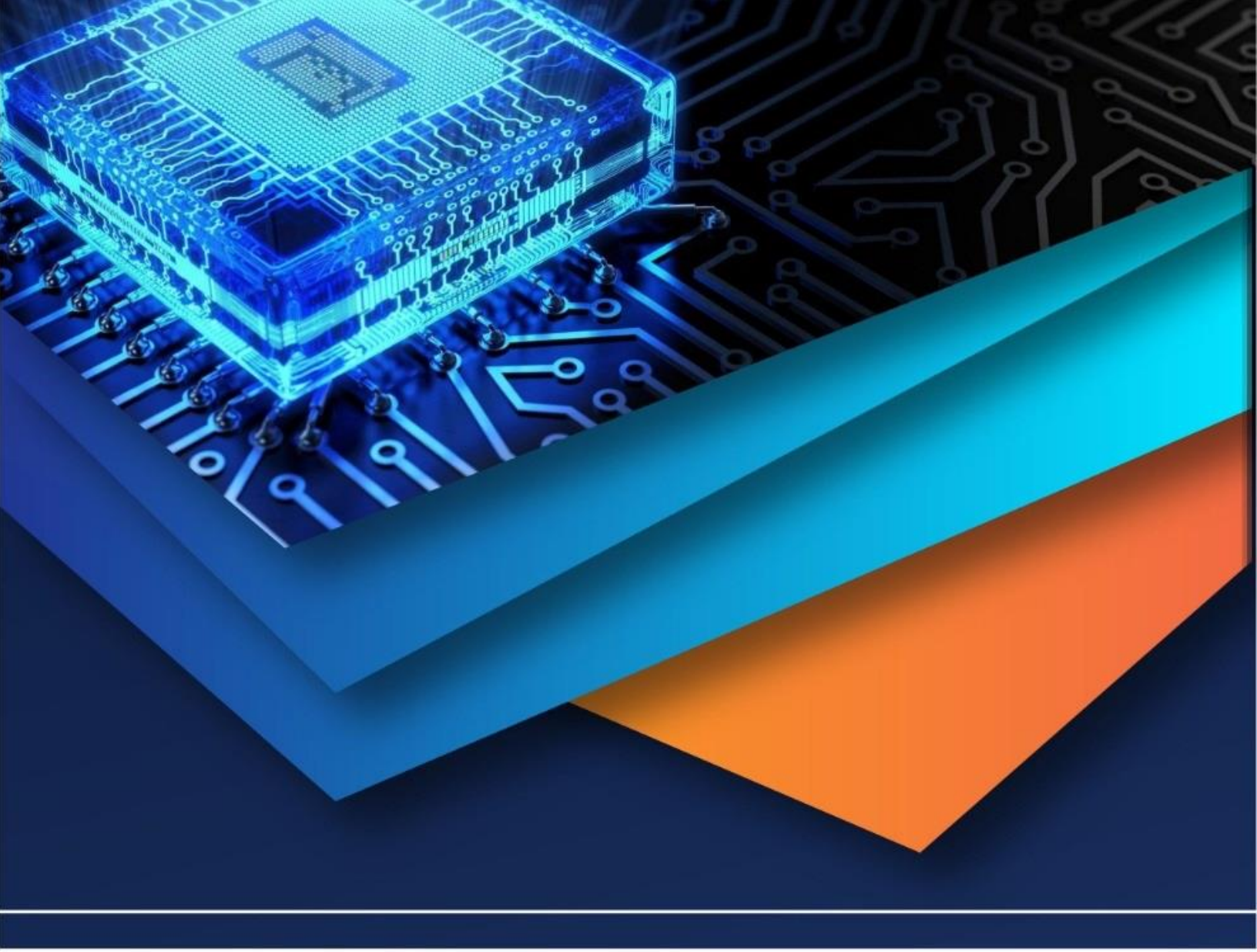

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