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# Dental Treatment and Quantum Mechanics

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## Abstract

In clinical practice, dentists sometimes encounter phenomena that cannot be explained by common medical concepts; for example, patients' bodily symptoms immediately improved after adjusting a denture that was not even inside their mouth, by extracting an infected tooth, and by bringing medicine close to the body. Although it seems difficult to completely elucidate the mechanism through modern medicine, it can be explained using quantum mechanics if it is assumed that symptoms appear because of the pathological waves emitted by the afflicted area of the body. The quantum, the smallest unit of matter composition, exhibits wave-particle duality. The fact that symptoms can be improved simply by bringing dentures or medicines closer to the body indicates that the waves emitted by dentures or medicine interfere with the pathological waves. Thus, the pathological waves are deformed and lead to a change in symptoms. In this way, quantum theory can explain phenomena that are difficult to elucidate in conventional medicine, which are encountered in daily clinical practice. Using biological reactions, unsolved problems in quantum mechanics, such as measurement problems, can be solved.

## Keywords

Dental Treatment, Quantum Mechanics, Wave Interference, The Bi-Digital O-Ring Test, Shoulder Impingement Syndrome

## 1. Introduction

The author has spent a long time studying the relationship between dentistry and the whole body. These two systems are interdependent, and in clinical cases, symptoms such as joint dysregulation: low back pain, stiff shoulders, hip joint dysregulation [1] [2] [3] [4], dermatitis [5] [6], and electromagnetic hypersensitivity [7] [8] [9] [10] [11]. However, because such complaints sometimes improve quickly after reforming a denture that has been removed from their

mouth, after extracting an infected tooth, or by bringing the medicine closer to the body [12], the mechanisms underlying these observations cannot be fully explained by conventional medicine that many scientists and practitioners commonly believe in. It is assumed that dentures and drugs have no effect unless they are taken into the body. And, even if an infected tooth is extracted, many bacteria existing in the body cannot be immediately removed.

For a different perspective, the author has turned to the quantum mechanical approach. Quantum mechanics mainly describes microlevel physical phenomena, such as elementary particles, atoms, molecules, or electrons [13] [14]. Because the body is also composed of these elementary particles, it can be considered to be influenced by quantum phenomena. Quanta exhibit wave-particle duality, *i.e.*, every quantum entity can be described as either a particle or a wave. When two or more waves are combined, wave interference occurs [15]. Thus, this makes it possible to explain the aforementioned phenomena if one assumes that the symptoms are caused by the pathological waves generated by the afflicted site of the body.

## 2. Case Studies

### 2.1. Case 1

Subject 1: A woman in her 70s

Chief complaint: She had difficulty in raising her left arm because of pain.

Medical history: Pain in the upper left arm was observed when raising her arm since 5 days ago and the pain gradually worsened.

Findings at initial visit: Partial dentures were located in the upper and lower jaws, and she had no discomfort or inconvenience with regard to the dentures. She had difficulty in raising her left limb because of pain (**Figure 1**).

Treatment: Her upper removable partial denture was removed and adjusted using the Bi-Digital O-Ring Test [16] [17].

Result: Although the denture was outside her body, her pain disappeared during adjustment (**Figure 2**). The denture was returned to her oral cavity and used as usual in her daily life. Her prognosis was good even after 1 week (**Figure 3**). Prognosis has been good for over a year.

Discussion of this case

It is possible that the waves emitted by the adjusted denture interfered with the pathological waves emitted by the afflicted area (*i.e.*, her left upper limb). These were thought to be the cause of pain when raising her left limb. In this case, the author assumed the pathological waves might have been deformed by interfering with the waves emitted by the denture, the pathogenicity of the pathological waves decreased, thus alleviating her symptom.

### 2.2. Case 2

Subject 2: A woman in her 70s

Chief complaint: She had difficulty in moving her head because of neck pain.



**Figure 1.** Findings at the first visit: Subject 1 had difficulty raising her left arm because of pain.



**Figure 2.** By adjusting her upper partial denture outside her body (arrow), subject 1's pain disappeared.



**Figure 3.** Findings 1 week after denture adjustment. Subject 1 had a good prognosis.

Treatment and result: When her removable partial denture was adjusted outside her body as in the first case, her neck pain disappeared. The denture was returned to her oral cavity and used as usual, and she had a good prognosis.

A YouTube video, “Denture adjustment outside the body” showing some of the details of cases 1 and 2 can be viewed at <https://www.youtube.com/watch?v=xFzv09sUWSA> (accessed on April 1, 2021).

### 2.3. Case 3

Subject 3: A 72-year-old woman

Chief complaint: She had difficulty raising her right arm because of pain (**Figure 4**).

Medical history: The woman had suffered from difficulty in raising her arm because of her pain since 1.5 months ago. She was diagnosed with shoulder impingement syndrome and had received a nerve-block injection twice. These treatments merely temporarily relieved her pain without improving the mobility of her upper limb.

Findings at the first visit: Her right upper first premolar had fractured and caused local inflammation as seen in a CT image (**Figure 5**). Antibiotics were selected according to the Bi-Digital O-Ring Test [16] [17] and brought close to the region of the infection, because of which her systemic symptoms reduced, and she could easily raise her arm (**Figure 6**). When the antibiotics were moved away from her body, the symptoms reoccurred.

Treatment: Her upper right first premolar was extracted (**Figure 7**).

Result: She could raise her arm immediately after the infected tooth was extracted and she no longer experienced shoulder impingement or pain (**Figure 8**). Three years later, she still had a good prognosis without any medical treatment.

The YouTube video “Dental treatment for shoulder impingement syndrome” shows the details for case 3. It can be viewed at <https://www.youtube.com/watch?v=mdM2eilSO4g&app=desktop> (accessed on August 1, 2020).

Discussion of this case

Shoulder impingement syndrome is a common condition involving pain and dysfunction of the afflicted shoulder. Its etiology can be multifactorial due to the interplay between intrinsic and extrinsic factors [18] [19]. In this case, it can be assumed that the pathological waves emitted by the infected first premolar and the right shoulder joint combined to cause her symptoms. Then, the symptoms improved when the antibiotics were brought close to her face because the pathological waves were interfered with those emitted by the antibiotic. Thus, the form of the pathological wave may have changed. After extracting her premolar, the original pathological waves disappeared, thus eliminating the symptoms.

This study shows that even when medicines exist outside of the body, they can have a marked effect on joint mobility. Generally, drugs and dentures are said to be ineffective in modern medicine unless they are taken into the body; however,

in this case, it was only necessary for the medications to be close to the body [12].



**Figure 4.** Subject 3 had difficulty raising her right arm because of pain.



**Figure 5.** CT image showing that Subject 3's right upper first premolar had fractured (arrow), causing local inflammation.



**Figure 6.** When antibiotics (arrow) were brought close to the area of infection, her systemic symptoms were reduced and she could raise her arm easily.



**Figure 7.** Subject 3's upper right first premolar was extracted.



**Figure 8.** Subject 3 could easily raise her arm immediately after extraction of the tooth, no longer experienced shoulder impingement, and no longer felt pain.

### 3. Discussion

Quantum mechanics forms the basis of modern physics along with the general relativity theory. It mainly describes microscopic physical phenomena, such as molecules, atoms, or the elementary particles that compose them. Quantum mechanics analyzes elementary particles, the smallest constituent units of matter [14]; naturally, if the properties of the minimum constituent units of a substance change, the properties of the human body also change. The basis of quantum mechanics is that elementary particles exhibit a wave-particle duality [15].

We may sometimes experience situations in clinical practice that cannot be completely explained by conventional modern medicine. Although it appears difficult to elucidate the mechanism behind such phenomena, it can be explained using quantum mechanics if the symptoms appear because of the pa-

thological waves generated by the afflicted site. When the waves emitted by substances such as medicine or dentures interfere with the pathological waves, the nature of the pathological waves changes and eases bodily discomfort. By considering these points, using the Bi-Digital O-Ring Test [16] [17] can be useful for determining the state of the waves.

The waves changed by the adjustment of the dentures interfered with the waves generated from the afflicted part of the body, and as a result, the symptom may have improved in case 1 and 2. The pathological waves emitted by the infected first premolar and the right shoulder joint combined to cause her symptoms in case 3. Then, the symptoms improved when the antibiotics were brought close to her face because the pathological waves were interfered with those emitted by the antibiotic. Thus, the form of the pathological wave may have changed. After extracting her premolar, the original pathological waves disappeared, thus eliminating the symptoms.

There are still many unresolved issues in quantum mechanics; for example, the phenomenology of wave function collapse can be described by the many-worlds interpretation [20] or the Copenhagen interpretation [21], but no conclusion has yet been reached. However, by applying body reactions, it may be possible to conclude which interpretation is correct, because almost all studies in physics rely on machines, not bodies.

#### 4. Conclusion

Quantum mechanics has a history going back more than 100 years; yet, it has rarely been applied in medicine. Using quantum mechanics, it becomes possible to explain some phenomena observed in clinical practice that cannot be clarified by modern medicine. Quantum mechanics will play an important role in the development of new therapies, and problems that have not been answered yet in the world in quantum mechanics may be solvable using biological reactions. Cooperation between medicine and physics is essential for progress in science.

#### Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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# Management of the Prosthetic Valve Thrombosis: This Time Is Different

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## Abstract

Prosthetic valve thrombosis (PVT) is a rare but serious entity. Its diagnosis and treatment can be challenging and mortality remains high regardless of its management. A 50-year-old man admitted with cardiac arrest, whose physical examination and echocardiography suggested PVT confirmed with fluoroscopy. Intra-aortic fibrinolysis was the treatment chosen with successful results.

## Keywords

Prosthetic Valve Thrombosis, Intra-Aortic Fibrinolysis, Low-Dose Slow Infusion

## 1. Introduction

Prosthetic valve thrombosis (PVT) is a rare and life-threatening complication with high mortality. In suspected PVT, a careful physical examination may reveal a muffling or disappearance of prosthetic sounds and the appearance of a new regurgitant or obstructive murmur [1]. Rapid confirmation of the diagnosis and prompt treatment is needed [1]. Delayed diagnosis and treatment can lead to increase morbidity and mortality. The therapeutic strategy (surgery or fibrinolysis) is mainly dependent on the hemodynamic status of the patient [2].

## 2. Case Report

A 50-year-old man received an aortic valve replacement five years earlier with a mechanical valve prosthesis with Dacron tube, due to a bicuspid aortic valve and type A aortic dissection detected incidentally by echocardiography (Figure 1).

Since then, he is receiving Vitamin K antagonist (VKA). He also has permanent AF and an episode of acute pericarditis eight years ago.

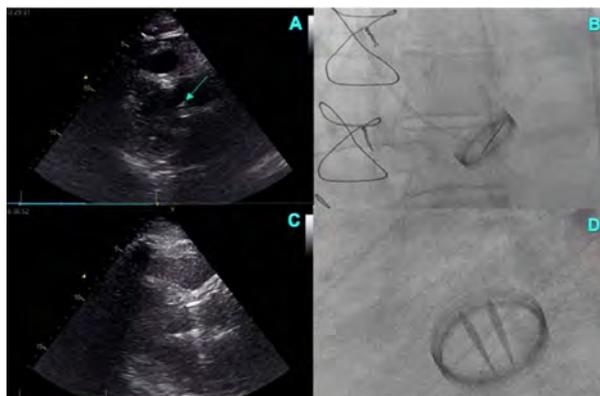
He was admitted with out-of-hospital cardiac arrest (OHCA) and ventricular fibrillation as initial rhythm. Recovered spontaneous circulation after 2 minutes of advanced cardiopulmonary resuscitation. Physical examination highlighted the absence of clicking prosthetic valve sounds. Postresuscitation ECG showed atrial fibrillation (AF) with average ventricular rate around 140 bpm with right bundle branch block (RBBB) and blood pressure of 90/60mmHg on norepinephrine infusion at 1.07 mcg/Kg/min. Analytically highlighted a metabolic acidosis with pH 7.08, lactic acid 4.4 mmol/L, bicarbonate 19.6 mmol/L, base excess  $-11.5$  mmol/L. The INR was subtherapeutic (1.4).

OHCA may be the initial manifestation of cardiac disease, particularly coronary artery disease. The physical examination and the patient's medical history make this diagnosis unlikely and make us suspect acute prosthetic valve thrombosis (PVT).

In the echocardiography, the absence of the prosthetic discs motion was striking. It also showed a suggestive image of thrombus in the left ventricular outflow tract (**Figure 2**). The left ventricular ejection fraction was mildly depressed (visual estimation 50%) and right ventricular size and function are normal.



**Figure 1.** CT angiography. Type A aortic dissection.



**Figure 2.** (A) and (B) pre-fibrinolysis. Arrow: Suggestive image of thrombus in the LVOT. (C) and (D) post-fibrinolysis.

The patient suffered rapid hemodynamic deterioration (BP 43/29 mmHg) and signs of peripheral hypoperfusion despite increasing doses of norepinephrine (2.0 mcg/kg/min).

Emerging cinefluoroscopy (CFS) was performed showing the absence of motion of one of the prosthetic leaflets. Coronary angiography showed epicardial coronary arteries without obstructive lesions and slow flow.

Due to the critical situation, intra-aortic fibrinolysis with bolus of 10 mg of rt-PA directed with the same diagnostic catheter was performed. Subsequently, the prosthetic valve was reevaluated, showing the correct motion of both leaflets, evidencing immediate hemodynamic improvement (**Figure 3**). ECG post-fibrinolysis showed disappearance of the RBBB (**Figure 4**). Slow infusion of 15 mg of rt-PA was continued for 6 hours in addition to unfractionated heparin with target aPTT of 50 - 60 seconds. At 20 hours after admission, due to several episodes of asystole and hypotension, as well as echocardiographic data of prosthetic valve restenosis, fibrinolysis was repeated with rt-PA (slow infusion of 25 mg over 6 hours), with favorable results within a few hours. The patient evolved favorably. Serial transthoracic echocardiography (TTE) showed a normally functioning prosthetic valve. Transesophageal echocardiography (TEE) showed severe spontaneous echo contrast in the left atrium with no thrombus seen.

CT angiography was performed and ruled out related abnormalities in the Dacron aortic tube. Cardiac MRI showed no thrombus or valvular pathology, unveiling changes secondary to previous aortic surgery and septal perfusion defects.

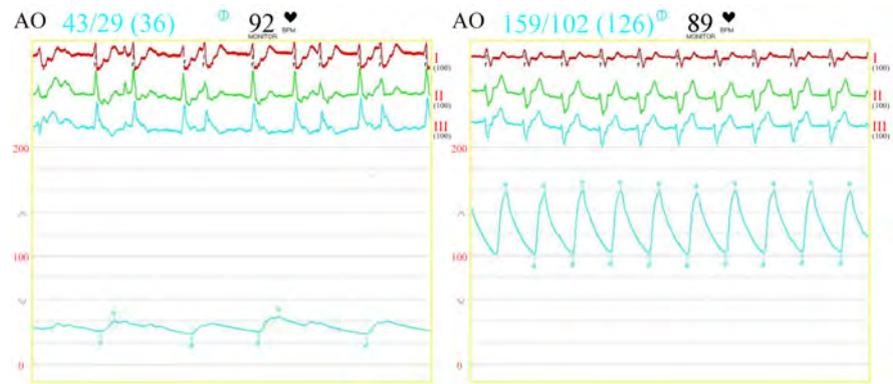
No systemic complications secondary to fibrinolysis were observed. The patient presented complete recovery of anoxo-ischemic encephalopathy, without neurological sequelae. At discharge he was on VKA.

At the 6-month follow up, our patient remained asymptomatic and optimally anticoagulated with VKA (time in therapeutic range 100%).

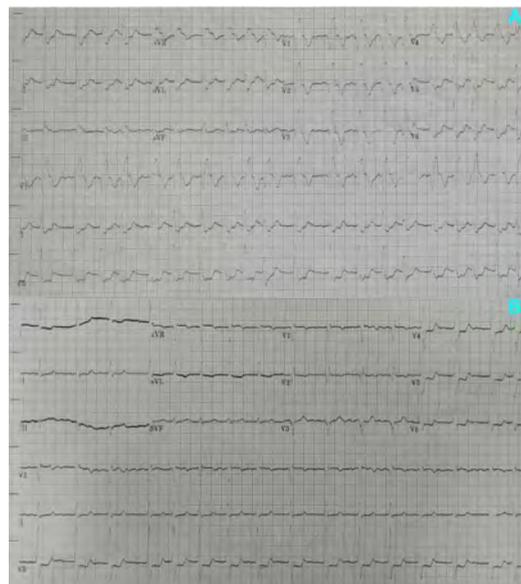
### 3. Discussion

The annual rate of prosthetic valve thrombosis with mechanical valves ranges from 0.1% to 5.7% [1], a figure that rises up to 6% in patients with infra-therapeutic anticoagulation ranges [1], as in our case presenting the patient with INR 1.4. Tricuspid PVT is 20 times more frequent than the left-sided prostheses, and mitral PVT is 2 to 3 times more frequent than aortic position [2]. Mortality associated with PVT is high, between 10% and 33% in the different studies.

The pathophysiology of PVT attends to the Virchow triad [2] and although PVT can manifest acutely, it is most often a subacute or chronic phenomenon [2]. The clinical presentation is variable and depends on the presence or absence of obstruction. A severe obstruction is associated with overt heart failure; however, a non-obstructive PVT is an accidental finding or may manifest as an embolic episode [2] [3].



**Figure 3.** Left: Pre-fibrinolysis intra-aortic pressure curve. Right: Intra-aortic pressure curve immediately after intra-aortic fibrinolysis.



**Figure 4.** ECG. (A) After fibrinolysis. Atrial fibrillation and complete RBBB. (B) After Intra-aortic fibrinolysis. AF and disappearance of the RBBB.

In the case of our patient, he began with cardiorespiratory arrest, rare form of PVT presentation.

When PVT is suspected, a thorough physical examination should be performed with special attention to prosthetic sounds (decrease or disappearance) and the appearance of new murmurs (regurgitation or obstruction).

The urgent evaluation is indicated with TTE, TEE, fluoroscopy, and/or multi-detector CT imaging to assess valve function, leaflets motion, and the presence of thrombus (Class of Recommendation (COR) 1, Level of Evidence (LOE) B) [3]. As for us, the first diagnostic test performed was a TTE, later confirmed by CFS. TEE can be performed when suspicion for PVT remains high after inconclusive TTE, but in hemodynamically unstable patients it is rarely necessary. The TEE was performed after stabilization of the patient to rule out intracardiac thrombogenic foci, visualizing severe spontaneous echo contrast in the left

atrium, which made us suspect that the origin of the thrombus leading to valve obstruction could have been the left atrium and especially the left atrial appendage, due to the acute presentation.

The differential diagnosis is with pannus or fibrous tissue [2] [3]. In our case, acute presentation with hemodynamic instability and immediate improvement after fibrinolysis, made it unlikely.

Obstructive PVT requires urgent and aggressive treatment. Although the treatment recommendations in the literature are limited, the two available therapeutic possibilities are surgery and fibrinolysis. According to 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease, urgent initial treatment with either slow-infusion, low-dose fibrinolytic therapy or emergency surgery is recommended (COR 1, LOE B) [3]. European point of view 4 is different, where surgery is recommended for obstructive thrombosis in critically ill patients without serious comorbidity (COR I, LOE C) and fibrinolysis (using rt-PA 10 mg bolus + 90 mg in 90 min with UFH or streptokinase 1,500,000 U in 60 min without UFH) should be considered when surgery is not available or is very high risk or for thrombosis of right-sided prostheses (COR IIa, LOE C) [4].

The decision between surgery and systemic fibrinolysis for symptomatic left-sided PVT should be individualized, depending on the type of prosthesis and its location, but mainly on the hemodynamic status of the patient.

Surgery is also reasonable in the presence of large or mobile thrombi and fibrinolysis is reserved for patients with a worse functional class and for those in centers that do not have cardiac surgery [1].

As far as we are concerned, fibrinolysis was the chosen treatment since the patient debuted with cardiac arrest, and was in NYHA functional class IV in a hospital without on-site cardiac surgery.

The fibrinolysis strategy we used was the slow-infusion (6 hours) with low-dose (25 mg) which we have modified, adding initial intra-aortic bolus (10 mg), given the critical initial situation of the patient, and the remaining dose (15 mg) in slow-infusion. In the TROIA trial [5], the regimen of low-dose slow infusion of r-tPA repeated as needed provides effective and safe thrombolysis in patients with prosthetic valve thrombosis. Recent studies using an echocardiogram-guided, slow-infusion, low-dose fibrinolytic protocol have shown hemodynamic success rates >90%, with embolic event rates <2% and major bleeding rates <2% [6].

#### 4. Conclusion

Mortality associated with PVT is high, regardless of the treatment used. The success of the treatment will depend firstly on the high clinical suspicion based on the physical examination and the hemodynamic status, and secondly on the rapid confirmation of the diagnosis through CFS or echocardiography. The therapeutic strategy is conditioned by the type of prosthesis and its location, but mainly by the hemodynamic status of the patient.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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# Expect the Unexpected: Bowel Obstruction Caused by *Taenia saginata*: A Case Report of Unusual Complications

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## Abstract

**Background:** Small bowel obstruction (SBO) is a common gastrointestinal condition often warranting acute surgical intervention. In human, tapeworm species can cause a parasitic infection by ingestion of raw or under cooked beef (*T. saginata*) or pork (*T. solium* and *T. asiatica*). Taeniasis usually presents with vague symptoms or mild abdominal pain and discomfort. The intestinal complications are not commonly mentioned in the literatures. **Case presentation:** We present 65-year-old Egyptian woman who lives in Oman, who is known to have type two diabetes mellitus and hypertension. She presented to Emergency department with abdominal pain and constipation for a period of three days. On examination, she was dehydrated with tachycardia (pulse rate: 110/minute) blood pressure of 100/60 mmHg and patient was afebrile. Abdominal examination revealed, mildly distended abdomen and tense, no guarding or rigidity no palpable mass, exaggerated bowel sound and rectum is empty on per rectal examination. Emergency laparotomy was performed with intra-operative findings of large foreign body impacted in the distal jejunum about 8 × 3 cm, cylinder in shape completely obliterating the lumen, distal loops was collapsed. **Conclusion:** Although Taeniasis is a very rare infection, index of suspicion such as a potential in the differential diagnosis as cause of an acute abdomen with unusual surgical complications should be considered. The diagnosis of these rare circumstances is usually made intraoperatively, and surgery is recommended only for the treatment of complications.

## Keywords

Tapeworm, Cysticercus, *Taenia saginata*, Intestinal Obstruction, Proglottids

## 1. Introduction

Small bowel obstruction (SBO) is a common gastrointestinal condition often warranting acute surgical intervention. In human, tapeworm species can cause a parasitic infection by ingestion of raw or under cooked beef (*T. saginata*) or pork (*T. solium* and *T. asiatica*). Although *Taenia saginata* can be found in many areas worldwide however, it is more prominent in tropical and underdeveloped countries [1]. It is associated with poverty, poor hygiene, and poor sanitation, therefore, health education, improvement in sanitation and hygiene play important part in prevention of such condition.

Taeniasis usually presents with vague symptoms or mild abdominal pain or discomfort. Symptoms can vary from less common symptoms like nausea, change in appetite, weakness or weight loss to more serious rare complications of intestinal obstruction and perforation of the gut [2]. The intestinal complications are not commonly mentioned in the literatures [2].

We are presenting a case of female patient presented with acute mechanical small bowel obstruction caused *Taenia saginata* infection as one of the rare causes of bowel obstruction.

## 2. Case Presentation

We present 65-year-old Egyptian woman who lives in Oman, who is known to have type two diabetes mellitus and hypertension. She presented to emergency department with abdominal pain and constipation for a period of three days. Pain was colicky in nature, sever started as a localized pain in the center of the abdomen then became diffuse to all over the abdomen. This was associated with nausea and constipation, but no vomiting or abdominal distention. She gave a history of attack of passing long white streaks like materials with her stool for one week before the development of her pain. However, no complains of bleeding per rectum or melena. No history of previous surgery, similar conditions nor weight loss.

On examination, she was dehydrated with tachycardia (pulse rate: 110/minute) blood pressure of 100/60 mmHg and patient was afebrile. Abdominal examination revealed, mildly distended abdomen and tense, no guarding or rigidity, no palpable mass, exaggerated bowel sound and rectum is empty on per rectal examination. Laboratory investigations showed WBCs of 11.70, neutrophil of 8.36, CRP: 329 mg/L, and other blood investigations were normal. Abdomen X-ray showed dilated loops of small intestines with multiple air fluid levels. CT scan of the abdomen showed large segmental mildly enhancing circumferential thickening involve distal jejunum causing moderate narrowing of lumen and dilatation of proximal small bowel, feature suggestive of sub-acute Small bowel obstruction secondary to foreign body.

Patient was reviewed by anesthetic team and was consented for laparotomy. Emergency laparotomy was performed with intra-operative findings of large foreign body impacted in the distal jejunum about 8 × 3 cm, cylinder in shape

completely obliterating the lumen, distal loops was collapsed. Resection of segment of affected small bowel was performed with primary anastomosis. No other intra-operative abnormalities were identified.

Post-operatively in recovery room, patient vomited large volume of bile containing large number of worms that were alive and moving. Patient was managed in recovery and was transferred to the ward. She recovered uneventfully and discharge home after 5 days of admission. She was then seen in outpatient clinic and was progressing well. Histopathology finding showed proglottid segments with eggs of taenia and adult worm.

### 3. Discussion

Taenia is a tapeworm acquired by ingesting undercooked beef or pork. Taeniasis is more common in Africa, Eastern Europe, Latin America and the Middle East. Most people with taeniasis will have no initial symptoms, however, as times goes, they will be aware of the infection as they pass proglottids in stool. Such gastric obstructive presentations are very uncommon in tapeworm infection, although enteric obstruction with subsequent gastric blockage has been previously reported. In contrast, bowel obstruction is a well-recognized complication of ascariasis [1].

Taenia species are common parasites that can infect humans. The two important species include the pork tapeworm or *Taenia solium* and the beef tapeworm or *Taenia saginata*. Taenia species found in Asia is a subspecies of *Taenia saginata* and it has been renamed as *Taenia saginata asiatica*. Taeniasis is endemic in Southeast Asia [2].

Humans are the definitive host. Adult tapeworms live in the human small intestine. Humans pass gravid eggs in feces; these mature eggs contaminate pastures and barnyards, where cattle and pigs ingest them. Upon reaching the alimentary canal of infected animals, the embryos are released, penetrate the gut wall, and enter the circulation. The embryos filter from the circulation and encyst in muscular tissue. Larvae (*i.e.*, cysticerci) become infectious within 2 - 3 months. Humans develop a tapeworm infection by eating raw or undercooked beef or pork containing cysticerci. The cysticercus becomes activated, attaches to the wall of the small intestine by the scolex, and becomes a mature tapeworm. This maturation process takes 10 - 12 weeks for *T. saginata* and 5 - 12 weeks for *T. solium*. A single tapeworm produces an average of 50,000 eggs per day and may live up to 25 years [3].

Examining the gravid proglottids helps identify the species; count the main uterine branches (7 - 16 branches for *T. solium*, 14 - 32 branches for *T. saginata* and 11 - 32 branches for *T. asiatica*) [4]. Examining the scolex helps differentiate the species because a *T. solium* scolex has 4 suckers and an armed rostellum and hooks but *T. saginata* scolex does not have rostellum and hook. *T. asiatica* has rostellum without a hook [4].

Histologic findings are mature cysticerci are ellipsoidal, translucent, flu-

id-filled cysts, 1 - 2 cm in diameter. Younger cysticerci are smaller. A single dense white body can be seen through the membrane. The spiral canal of the cyst wall, which has a wavy appearance in most tissue preparations, is most frequently observed in biopsy specimens. The wall, which is 100 - 200 micrometers wide, is characterized by an internal parenchymal layer of longitudinal and circular muscle, a middle layer of pseudoepithelial cells, and an outer cuticular layer composed of a dentate membrane with a microvillus projection that interfaces with host tissues. The scolex region is thickened and more organized. Cross sections of the scolex appear as several layers of folded smooth muscles, which may contain parts of the suckers or hooklets. The parasite is surrounded by an adventitia of host tissue reaction. A scant local cellular reaction that consists of some eosinophils and macrophages surrounds live cysticerci; dead cysticerci are surrounded by a dense inflammatory infiltrate that consists of the entire spectrum of inflammatory cells, including multinucleated giant macrophages [3].

*Taenia saginata* causes intestinal taeniasis, manifesting as abdominal discomfort, nausea, vomiting and weight loss. Some patients complain of passage of proglottids in the stool. Adult parasite may rarely be present in the stomach and the gastric secretions [3]. However, migrating proglottids have the tendency of causing bile duct inflammation, cholecystitis and appendicitis. Cases of intestinal small bowel perforation secondary to taeniasis have been reported in the literature.

Cysticercus attaches to the wall of the small intestine by means of scoleces and becomes a mature tapeworm. Intestinal taeniasis manifests as abdominal discomfort, indigestion, nausea, diarrhea, and weight loss [4]. Other complications include obstruction of bile ducts or pancreatic ducts, leading to cholecystitis, acute pancreatitis and granulomatous gastritis. The diagnosis of intestinal taeniasis depends on demonstration of the typical taenia eggs in the stool [5] [6].

Mechanical obstruction of small bowel is a very rare especially in unaffected communities. This indicates that taeniasis of the gastrointestinal tract can be the cause of the most unusual complications.

The diagnosis of these rare circumstances is usually made intraoperatively, and surgery is recommended only for the treatment of complications [6].

Individuals with intestinal *Taenia* infection might be asymptomatic or present with mild symptoms. Once tapeworms are detected in faeces of infected individuals, anthelmintic therapy with praziquantel or niclosamide is usually sufficient. Such treatment could prevent cases of cysticercosis [7]. Asymptomatic cysticercosis requires no treatment. Cases with infection might be asymptomatic due to the long incubation period which could extend up to 16 years prior to symptoms become evident [8]. Asymptomatic cases with cysticercosis that are undiagnosed can have adverse outcomes if they were given albendazole or praziquantel as a treatment for other conditions. This is mainly caused by an inflammatory response from the dying parasite [7].

Treatment of such condition includes taking care of personal hygiene and ensuring meat and beef are cooked to avoid complications of teniasis. Along such

lifestyle changes, medical treatment such as praziquantel or niclosamide can also be given [6]. Kalkan *et al.* 2013 have success removed *Taenia* endoscopically from the stomach [7]. Other endoscopic approaches that have been used are the injection of medication into small bowel wall allowing detachment of the tapeworms from the intestinal wall and excreted in stool.

#### 4. Conclusion

Although Taeniasis is a very rare infection, index of suspicion such as a potential in the differential diagnosis as the cause of an acute abdomen with unusual surgical complications should be considered. The diagnosis of these rare circumstances is usually made intraoperatively, and surgery is recommended only for the treatment of complications. The possibility of parasitic infection should be kept in mind as a very rare possible cause of bowel obstruction even in non-endemic country and detailed history could helpful. Medical treatment of taeniasis includes oral medicine such as praziquantel or niclosamide, although prevention is the best to avoid serious complications of infection. However, in some cases endoscopic or surgical interventions might be needed.

#### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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# The Effect of Sacubitril/Valsartan in a Dialysis Patient with Severe Heart Failure\*

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## Abstract

Heart failure (HF) is a major comorbidity in patients with end-stage renal disease (ESRD). The pathogenesis of HF in patients on renal replacement therapy represents the confluence of several traditional and nontraditional vascular risk factors, unique to the milieu of chronic kidney disease and the dialysis modality [1]. The purpose of this report is to describe the efficacy and safety of sacubitril/valsartan for an ESRD patient on hemodialysis therapy combined with heart failure with reduced ejection fraction (HFrEF). A 35-year-old woman was undergoing hemodialysis due to ESRD and suffering from heart failure with reduced ejection fraction. Because of worsening heart failure and hypertension, she was prescribed with sacubitril/valsartan at a dose of 50 mg twice a day, spironolactone at a dose of 20 mg three times a day and metoprolol at a dose of 23.75 mg once daily. There was a symptomatic improvement with the heart failure and reduction in NT-proBNP level, accompanied by a decrease of blood pressure after using sacubitril/valsartan. In conclusion, it is safe and effective to take sacubitril/valsartan in this hemodialysis patient with severe heart failure.

## Keywords

End Stage Renal Disease, Sacubitril/Valsartan, Hemodialysis, Heart Failure, Reduced Ejection Fraction

## 1. Introduction

Cardiovascular disease is the main cause of death in maintenance hemodialysis patients. Many studies have shown that the proportion of dialysis patients complicated with heart failure is as high as 45% [2] [3] [4]. Heart failure is the most

\*An informed consent was obtained from the patient.

#Corresponding author.

common and serious cardiovascular complication of dialysis patients, with a very high incidence and poor prognosis, which is an important reason for the progress and death of patients [5] [6]. The treatment of maintenance hemodialysis complicated with heart failure is inhibition of RAAS system (Angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs) and aldosterone antagonist), control of hypertension, Beta-blockers, statins, etc [7]. Sacubitril/valsartan, which combines an angiotensin receptor blocker (valsartan) with a NEPI (sacubitril), was the first angiotensin receptor–neprilysin inhibitor to be developed. Natriuretic peptide system (NPS) is a kind of neuroendocrine system. It has many beneficial functions, including natriuretic, diuretic, vasodilator and anti regulation of RASS. Neprilysin (NEP or neutral endopeptidase) is the key enzyme responsible for degrading natriuretic peptides. Sacubitril/valsartan consists of the neprilysin inhibitor sacubitril and the ARB, which strengthens the protective neuroendocrine system of the heart while inhibiting the renin-angiotensin-aldosterone system, was superior to enalapril in reducing the risks of death and of hospitalization for heart failure. The PARADIGM-HF trial showed that sacubitril/valsartan reduced the risk of cardiovascular mortality among patients with heart failure with reduced ejection fraction when compared with the angiotensin-converting enzyme inhibitor enalapril, several trials in populations with heart failure. The UK HARP-III trial (United Kingdom Heart and Renal Protection-III) has demonstrated that, compared with irbesartan, sacubitril/valsartan further reduces both blood pressure and biomarkers of cardiovascular risk (troponin I and N-terminal pro-B-type natriuretic peptide), and in a wide range of people with proteinuric chronic kidney disease, adding neprilysin inhibition to angiotensin II receptor blockade has no additional effect on kidney function or albuminuria [8] [9] [10]. However, there were less reports on tolerability and outcome of patients with hemodialysis patients. The purpose of this case is to report the efficacy and safety of sacubitril/valsartan in the treatment of ESRD patients with HFrEF.

## 2. Case Report

A 35-year-old hemodialysis female patient was evaluated for heart failure with reduced ejection fraction (EF). Six years ago, she suffered from pre-eclampsia at 33 weeks of pregnancy. The patient was diagnosed with nephrotic syndrome and creatinine level reached 595  $\mu\text{mol/L}$ . After the termination of a pregnancy, she received the renal biopsy. The pathological results of renal biopsy showed focal proliferative sclerosis IgA nephropathy with pre-eclampsia pregnancy-induced renal damage. The patient was given the CCB (calcium channel blocker) to control the blood pressure. However, she didn't go to the hospital for regular review. Two years ago, the patient had severe chest distress and she couldn't climb the stairs. Her blood pressure was 160/100mmHg and BNP level had reached 70,000 pg/ml. At that time, severe renal impairment appeared with high creatinine value of 714.5  $\mu\text{mol/l}$  and low glomerular filtration rate of 5.89  $\text{mL}/\text{min}^{-1}/1.73 \text{ m}^2$ . The patient began to receive hemodialysis and anti-hypertension medications

with CCB (calcium channel blocker) and Beta blockers. One year ago, the patient suffered severe chest tightness and inability to lie down at night, she was hospitalized in the department of cardiology. Despite medical therapy, she was 3rd times hospitalized because of decompensated heart failure. Physical examination revealed blood pressure of 140/100 and mild pitting edema in both lower extremities. The serum creatinine was 642  $\mu\text{mol/L}$  and an estimated glomerular filtration rate of  $6.65 \text{ mL/min}^{-1}/1.73 \text{ m}^2$ . She also had renal anemia and secondary hyperparathyroidism. The patient was assessed as NYHA (New York Heart Association) class 4 heart failure. The parathyroid hormone was 827.5  $\text{pg/ml}$ , albumin was 33.7  $\text{g/L}$  and the hemoglobin was 118  $\text{g/L}$  at that time. The NT-proBNP level was 385,000  $\text{pg/ml}$ . The echocardiography showed the Left ventricular function was depressed with an ejection fraction 28% and severe mitral regurgitation with a regurgitant area of  $15.1 \text{ cm}^2$  (**Table 1**). She also had severe pulmonary hypertension that pulmonary artery pressure was estimated to be 56  $\text{mmHg}$  and tricuspid incompetence with a regurgitant area of  $12.7 \text{ cm}^2$ . She was treated with 1.5  $\mu\text{g}$  of calcitriol twice a week. She received the dose of 50  $\text{mg}$  sacubitril/valsartan twice a day, 20  $\text{mg}$  of spironolactone once a day and 23.75  $\text{mg}$  of metoprolol once a day. The dialysis protocol of the patient was as follows: hemodialysis (HD) 5 times every two weeks and hemodiafiltration (HDF) once every month. She received high-throughput dialysis with a blood flow of 250  $\text{ml/min}$  and a dialysate flow rate of 500  $\text{ml/min}$ . After 1 year of active treatment, the hypertension was well controlled and there was no symptomatic hypotension. The parathyroid hormone value of the patient decreased from 827.5  $\text{pg/ml}$  before treatment to 605.1  $\text{pg/ml}$  at the 6<sup>th</sup> month after treatment and 366.4  $\text{pg/ml}$  at the 12<sup>th</sup> month of treatment (**Table 2**). The albumin value also increased to the normal level. Moreover, the functional class improved from NYHA 4 to NYHA 1. Both left and right ventricular diameters estimated by echocardiography were decreased. The left ventricular diameter decreased from 23  $\text{mm}$  to 12  $\text{mm}$  and the right ventricular diameter decreased from 59  $\text{mm}$  to 46  $\text{mm}$  (**Table 3**). Severe tricuspid regurgitation and pulmonary hypertension also got improved and pulmonary artery pressure decreased from 56  $\text{mmHg}$  to 14  $\text{mmHg}$ . The ejection fraction increased from 28% to 66% and the NT-proBNP value decreased from 385,000  $\text{pg/ml}$  to 1092  $\text{pg/ml}$ . Now, the patient's heart failure symptoms have improved significantly, and the echocardiography indicators have improved compared to before. Because patient has been insisting on taking sacubitril/valsartan, the frequency of hospitalizations due to cardiovascular complications is also decreasing. We will continue to follow-up the condition of this patient.

### 3. Discussion

Cardiovascular events are the leading causes of death in patients with chronic kidney disease. Patients with chronic kidney disease receiving hemodialysis are often complicated with heart failure, and cardiac dysfunction is closely related to renal dysfunction.

**Table 1.** Initial echocardiography report of this patient.

Right ventricular diameter 23 mm	Ventricular septal thickness 14 mm	Left ventricular diameter 59 mm	Left ventricular posterior wall 13 mm	Aortic ring diameter 21 mm
Left atrial diameter 43 mm	Ascending aorta diameter 41 mm	Pulmonary artery ring diameter 24 mm	Pulmonary valve 0.73 m/s	Right atrium 49 × 65 mm
E peak 0.99 m/s	A peak 0.37 m/s	Aortic valve 0.97 m/s	Tricuspid regurgitation 3.1 m/s	Pulmonary artery pressure 56 mmHg
EDV 295 ml	ESV 212 ml	LVEF 28%	Mitral valve orifice regurgitation area 15.1 cm <sup>2</sup>	Tricuspid regurgitation area 12.7 cm <sup>2</sup>

EDV: End Diastolic Volume; ESV: End Systolic Volume; LVEF: Left Ventricular Ejection Fraction.

**Table 2.** Comparison before and after taking sacabitril/valsartan.

	2019.1	2019.6	2019.12
BP (mmHg)	159/100	145/100	122 - 145/72 - 96
ALB (g/L)	32.2	44.7	41.7
Blood phosphorus	0.94	0.79	0.6
PTH (pg/ml)	827.5	605.1	366.4
URR (%)	65.8	74.6	75.4
KT/V	1.3	1.64	1.63

BP: Blood pressure. ALB: albumin. PTH: parathyroid hormone. URR: Urea Reduction Ratio. KT/V: Urea clearance index.

**Table 3.** Cardiac parameters before and after taking sacabitril/valsartan.

	Before	After
Right ventricular diameter	23 mm	12 mm
Ventricular septal thickness	14 mm	11 mm
Left ventricular diameter	59 mm	46 mm
Pulmonary artery pressure	56 mmHg	14 mmHg
LVEF	28%	66%
NT-proBNP	385,000 pg/ml	1092 pg/ml

LVEF: Left Ventricular Ejection Fraction. NYHA: New York Heart Association.

The case we describe here is an ESRD patient undergoing regular hemodialysis with severe heart failure. The patient underwent echocardiography showing a series of heart failure symptoms including decreased ejection fraction, pulmonary hypertension, severe mitral and tricuspid regurgitation, etc. She also had hypertension. She received the dose of 50 mg sacubitril/valsartan twice a day, 23.75 mg of metoprolol once a day and 20 mg of spironolactone three times a day. After a period of active treatment, the symptoms of heart failure of this ESRD patient with hemodialysis have been significantly improved. High blood pressure was effectively controlled without symptomatic hypotension and

NT-proBNP level decreased from 385,000 pg/ml to 1092 pg/ml. In terms of safety, during the use of sacubitril/valsartan, the patient did not experience hyperkalemia and other side effects. Previous studies had also reported the efficacy of sacubitril/valsartan in patients with ESRD, a 67-year-old man with heart failure with reduced ejection fraction due to an ischemic cardiomyopathy and renal insufficiency undergoing hemodialysis. Because of worsening heart failure with no other therapeutic options, a treatment with sacubitril/valsartan was started. After initiation of sacubitril/valsartan, there was a symptomatic improvement with a clear reduction NT-proBNP, accompanied by a decrease in filling pressures [11] A retrospective study analysed the clinical and laboratory data of 23 HFrEF patients, found that sacubitril/valsartan could reduce the hsTnT (high-sensitive troponin) and sST2 (soluble ST2) levels and improve LVEF in HFrEF patients with ESRD, which is the first study to show the effectiveness and safety of sacubitril/valsartan in ESRD patients with HFrEF [10].

In conclusion, we found that sacubitril/valsartan could effectively and safely improve the symptoms of heart failure in this patient with ESRD on regular hemodialysis.

### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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# Palinopsia as a Rare Presenting Symptom of Occipital Stroke “Case Report”

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## Abstract

Palinopsia is the recurrence or persistence of visual images after cessation of the stimulus. Palinopsia has been associated with a wide variety of etiologies and mechanisms such as drug induced, seizures, migraine, psychiatric conditions, head trauma and structural lesions in the brain. We report a case of occipital stroke who presented with oscillating palinopsia. Sudden-onset palinopsia is a very rare symptom of stroke, but it must be recognized early as it is a highly time dependent, and potentially treatable condition. A 57-year-old woman with a history of poorly controlled type 2 diabetes, hyperlipidemia, and hypertension presented with sudden onset right sided palinopsia with images of her face and right forearm with hand, occurring several times in a day, lasting for a few minutes each time, and appearing in the same location each time. There are few case reports in literature secondary to stroke and what makes it interesting also is that she reported persistence of image even when her eyes are closed. Moreover, the sudden onset is not common in literature. The Magnetic Resonance Imaging (MRI) of the brain showed evidence of left Posterior Cerebral Artery (PCA) territorial (Fusiform gyrus) subacute ischemic infarction. Visual field assessment showed a right homonymous hemianopia. She was treated with aspirin and clopidogrel. The frequency and intensity of palinopsia reduced subsequently. The face image disappeared in the first week while the forearm image disintegrated gradually over the next few weeks. Palinopsia may be a rare presenting symptom of stroke. It is important for the clinician or emergency room doctors to be alert to the possibility of undiagnosed structural neurological lesions like occipital stroke and differentiate it from non-organic causes, as prompt management may reduce the risk of visual or motor disability.

## Keywords

Palinopsia, Oscillating, Occipital Stroke, Hemianopia, Case Report

## 1. Introduction

Visual perseveration or palinopsia is a term that describes multiple types of visual symptoms characterized by recurrence or persistence of visual images after cessation of the stimulus. This category of palinopsia represents a dysfunction in visual memory and is caused by posterior cortical lesions or seizures. It can be classified as hallucinatory and illusory palinopsia which can last for various durations. Sudden-onset palinopsia is a very rare symptom of stroke, but must be recognized as stroke, as it is a treatable condition, and the treatment is highly time dependent. This patient is unique in her sudden onset symptoms, vascular aetiology and persistence of image when eyes are closed.

## 2. Case Presentation

A 57-year-old, right-handed lady was referred to the neurology outpatient department (OPD) for evaluation with sudden onset visual symptoms. She had a history of poorly controlled diabetes mellitus, hypertension and dyslipidaemia. There was family history of diabetes mellitus, hypertension and dyslipidaemia. She had a stable psycho-social history. Four days prior to presentation at the neurology OPD, the patient had attended the emergency room for raised blood pressure (200/94mmHg). She had no symptoms at that point and her blood pressure was brought under control with medications. Computed tomography of the brain at that time did not reveal any pathological findings. She complained of blurring of vision the next morning. She underwent an ophthalmological examination which was unremarkable and patient was reassured and sent home.

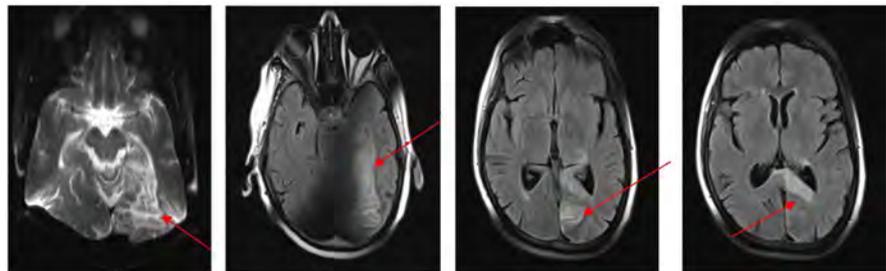
However, on day 3, she noted that the blurring of vision had worsened. She further stated that she specifically saw her right hand and forearm on the left upper quadrant of the temporal field while her right upper limb was actually on her knee or over her chest. This occurred several times in a day, lasted for few seconds and the image was static, vivid and had clear contours. The image was visible even when her eyes were closed. Occasionally, she saw her face in the same area of the visual field. This was short lasting and the image was not distorted. She also noted that she was unable to see people standing in her right temporal field. The patient did not report any visual distortions or alterations in colours or micropsia, macropsia or metamorphosia. She also did not report any associated headaches, eye pain, impaired consciousness, or awareness and she was not on any new medications. The patient was very distressed by her symptoms and sought medical attention.

Her ophthalmological assessment showed that the best corrected visual acuity using the Snellen chart was 6/15 in the right eye and 6/30 in the left eye. The intra ocular pressure was normal in both eyes. Colour vision in both eyes was normal. Anterior segment examination revealed that the right pupil was mid-dilated with grade one relative afferent pupillary defect. There was no papilloedema. Both fundi showed moderate non-proliferative diabetic retinopathy with macular edema. The ocular coherence tomography of the macula in both eyes showed

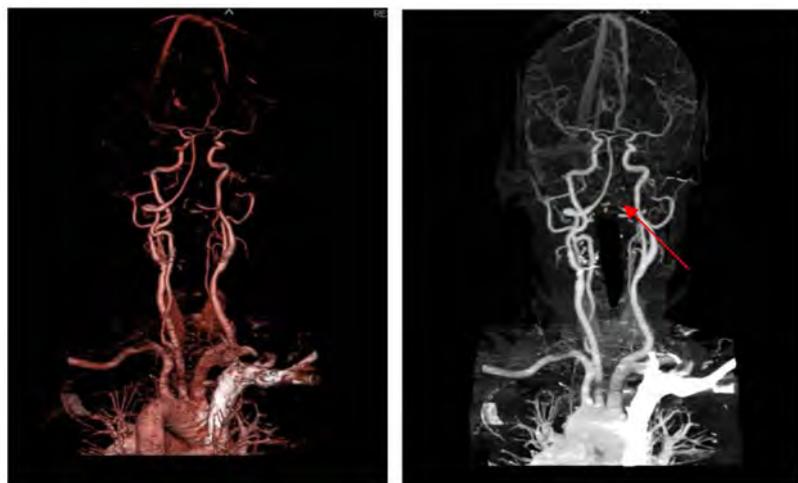
mild cystoid spaces corresponding to the macular edema with a left vitreo-macular traction. She was referred to the neurologist for evaluation of her symptoms.

Neurological examination revealed the presence of an isolated right homonymous hemianopia and absent tendon reflexes. The Magnetic Resonance Imaging (MRI) of the brain (**Figure 1**) showed an evidence of left Posterior Cerebral Artery (PCA) territorial (Fusiform gyrus) subacute ischemic infarction with evidence of minute haemorrhagic transformation. There was a secondary tonsillar herniation plus the sequelae of chronic lacunar infarcts and involuntional brain changes.

**Figure 2** shows the findings on the Computed Tomographic Angiography (CTA). There was evident proximal narrowing of the P2 & P3 segments of the left posterior cerebral artery with total occlusion of its continuation as the P4 segment. There were mild to moderate atherosclerotic changes of the aortic arch with bilateral carotid bulb calcified atheromatous plaques which were more prominent on the left side. The anterior communicating and left posterior cortical artery were hypoplastic. There was a slight narrowing of the M3 segment of the left middle cerebral artery (MCA).



**Figure 1.** Brain MRI in different sequences showing hyperacute infarction showed by the red arrows on the left occipital lobe.

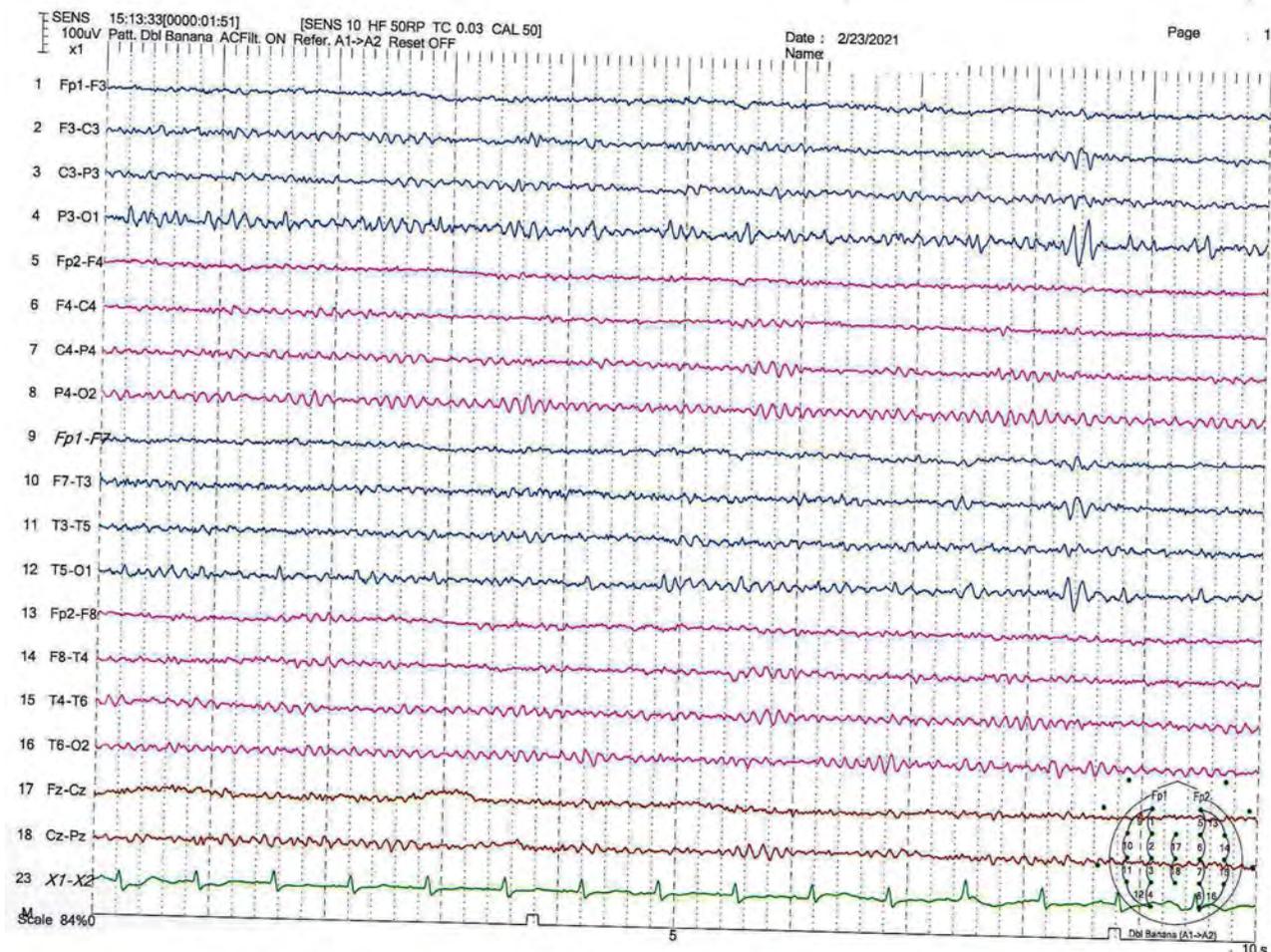


**Figure 2.** Computed tomography angiography of cerebral vessels showing total occlusion of its continuation as the P4 segment shown as absent artery compared to the right side as shown by red arrows.

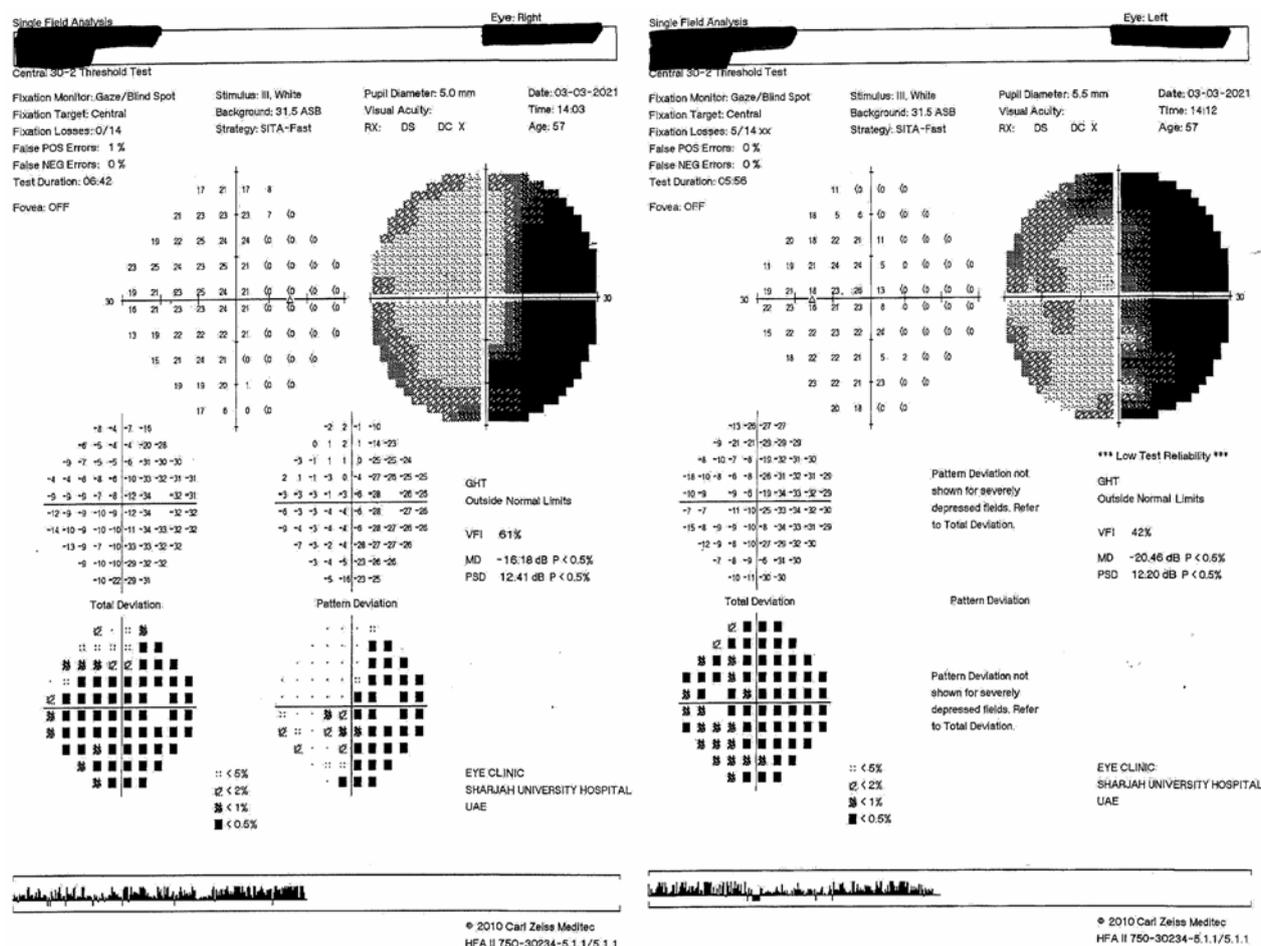
Laboratory investigations including complete blood counts, C-reactive peptide, Vitamin B12, liver, renal, thyroid function tests and coagulation profile were within normal limits. Serum homocysteine levels were marginally high. Lipid profile revealed high triglycerides and low HDL values. Urine analysis revealed microalbuminuria. The antinuclear antibody (ANA) test and the Anti-neutrophil cytoplasmic antibodies (ANCA) (both cytoplasmic (c-ANCA) or perinuclear (p-ANCA)) were negative.

**Figure 3** demonstrates the electroencephalogram (EEG) record which showed a left posterior occipital and temporal sharp contour wave form discharges, sharp wave discharges and slow waves which were centered at the posterior electrode. The run of discharges disrupted the background alpha rhythm and its field extended to left posterior electrodes in the temporo-occipito-parietal lobes (Low frequency time constant LFT1Hz & High frequency filter HFF 70 Hz). The echocardiogram was unremarkable with no significant arrhythmias or Atrio-ventricular (AV) block.

Her Visual field assessment showed a right homonymous hemianopia (**Figure 4**). Visual evoked potentials were within normal limits.



**Figure 3.** EEG.



**Figure 4.** Visual field assessment.

Following completion of this extensive, multi-disciplinary work up, she was diagnosed as a case of palinopsia due to occipital ischemic stroke secondary to left posterior cerebral artery (P4) thrombotic occlusion. Her medical background was the main risk factor.

The patient was started on high dose aspirin (300 mg) daily for one week along with statins, rehydration, Omega 3, Citicholine and NeuroAid capsules (proprietary blend of 9 herbal ingredients). The patient remained on regular follow up with the neurology and ophthalmology OPD. One week later, she reported that the afterimages of her hand and forearm were less frequent and less intense. At times, the image also appeared to disintegrate. The afterimages of her face disappeared after a week. Her hemianopia, however did not improve. She was reassured and started on Clopidogrel 75 mg and low dose aspirin 100 mg along with her other medications. Clopidogrel was discontinued after a few days as she developed sores/ulcers in the mouth. The patient was followed up for 4 months in the neurology/Ophthalmology OPDs. Her palinopsia did not relapse, had no hemiplegia, but her hemianopia improved mildly depending on the objective visual field assessment. The diabetic, lipid and hypertensive levels were well controlled.

### 3. Discussion

We presented a patient with long standing risk factors for ischaemic stroke having repeated episodes of palinopsia as a major presenting symptom of stroke. Palinopsia is always indicative of some systemic dysfunction. It is important for the clinician to be alert to the possibility of undiagnosed neurologic disease, as it could be life-saving.

This case is unique in that palinopsia was a presenting feature of stroke. The well-coordinated assessments lead to critical diagnoses that met the patient's concerns. Our patient was clearly satisfied with the management of palinopsia and we outlined stroke prognosis depending on the risk factors and size of infarction on the MRI brain. This affected her visual field capacity. When palinopsia is due to structural disease, palinopsia usually occurs in patients with impaired vision who are not entirely -blind and usually tends to occur transiently during the progression or resolution of visual field defect [1]. Sun Yuan-Ting *et al* reported a case with a sequential appearance and disappearance within two days of homonymous hemianopsia, palinopsia and metamorphopsia in the defective visual field, nine months after the stroke [2]. They speculated that sequential hemodynamic changes in the right occipital area, from hypoperfusion to transient hyperperfusion and finally to normal perfusion could have precipitated these symptoms. Weber *et al* described a case of a 77-year-old man with ischaemic risk factors who presented with isolated intermittent palinopsia lasting for a few hours [3]. There were no other focal neurological or associated symptoms and his neurological examination was unremarkable. He was managed as a case of acute occipital lobe stroke.

The palinopsia in our patient was not precipitated by the presence of an obvious visual stimulus and was not associated with migraine. Migraine is the most common single cause of visual hallucinations and illusions, and palinopsia can be present either before or after a migraine attack. Kalita *et al.* reported that around 10% of migraineurs had palinopsia and they tend to be more frequent and persistent than in normal individuals [4]. The increased frequency of palinopsia in migraineurs may suggest an enhanced physiological function of cortical neurons in the migraine. Takayama *et al* reported a case of a 55-year-old male patient with long-standing migraine history who developed an abscess in the right occipital lobe [5]. The patient developed episodes of palinopsia accompanied with migraine attacks after healing of the occipital lesion. The authors hypothesized that migraine potentiates the effect of cortical lesions in generation of palinopsia and palinopsia occurred only when the neocortical excitability exceeded a certain limit by severe migraine attack.

Palinopsia can occur as manifestation of epilepsy and seizures of the visual processing and memory centres of the brain might be the underlying cause of palinopsia [6]. Palinopsia may occur during a seizure aura, the ictal phase, and postictally and anticonvulsive therapy has also been found to reduce palinopsia in symptomatic patients [7]. Occasionally palinopsia may be the only symptom

of the seizure, without any motor or sensory symptoms [7]. Our patient did not have any clinical symptoms of epilepsy and her EEG changes may be related to the structural damage over the occipital cortex. However, the cortical irritation or hyperexcitability due to stroke may also have provoked the epileptic phenomenon and the two cannot be easily distinguished. However, the isolated palinopsia, the duration of the event and relapses during the day without other features of epilepsy and reduction of symptoms without anti-convulsant therapy makes it less likely to be a manifestation of epilepsy. Systemic disease can result in epilepsy, as reported by Kondziella and Maetzel, wherein a patient experienced palinopsia associated with epileptic seizures caused by an occipital lobe glioblastoma [8].

Episodes of palinopsia may also occur due to disturbances of peripheral vestibular function. Stafuzza *et al* reported a patient with episodes of palinopsia after the functional loss of the 3 semicircular canals of the right ear with preservation of the otolithic function [9]. The abnormal temporal integration of the visual and vestibular inputs may result in palinopsia if there is a delay in the vestibular inputs [9]. The absence of vestibular symptoms and normal hearing function in our patient makes it unlikely to be a causal factor.

Palinopsia has generally been described in reference to static afterimages. In our case too, the patient reported static images of her forearm and hand. Lahiri *et al* reported a case of kinetic palinopsia with images of a car moving backwards in his left visual field [10]. Similar to our case, their patient also had an associated field defect (left homonymous hemianopia) due to right occipital arteriovenous malformation (AVM) affecting the lingual gyrus and right occipital lobe. Palinopsia commonly occurs in the setting of posterior visual pathway deafferentation causing homonymous visual field deficits and it is most often hallucinatory palinopsia. This palinoptic mechanism is thought to be similar to visual release hallucinations (Charles-Bonnet syndrome), which are due to neuronal hyperexcitability, often from ocular vision loss [7]. Whereas hallucinatory palinopsia occurs due to focal cortical hyperexcitability from cortical visual loss. Visual release hallucinations can be distinguished from palinopsia by the content of the images (whether the formed image or scene actually occurred).

Palinopsia has been reported in Creutzfeldt-Jakob disease (CJD), Leber's Hereditary optic neuropathy, optic neuritis, perilesional hyperperfusion, multiple sclerosis, ceroid lipofuscinosis, Glycine receptor antibody syndrome, nonketotic hyperglycemia, tumors and leukemias [6] [7] [10] [11]. There were no associated neurodegenerative processes or other systemic diseases which may be a likely etiology for the palinopsia in our patient. There was no cognitive decline or ataxia as seen in palinopsia in ceroid lipofuscinosis [11]. Palinopsia usually results from a lesion in the dominant hemisphere in 25% of cases [9], as seen in our patient. Authors have reported a case of a glioblastoma tumor in the non-dominant occipito-temporal region, wherein the pattern of palinopsias was a complex scene of the family members and room contents [12]. In contrast, our patient had static palinopsia with afterimages of his own forearm and hand.

Palinopsia has been found to be brought on by a variety of prescribed medications (Topiramate, clomiphen citrate, Zosuquidar, Nefazodone, Interleukin 2 therapy, trazodone) and illicit drugs (marijuana, mescaline, lysergic acid diethylamide, 3,4-methylenedioxymethamphetamine [6] [7] [13] [14] [15] and most are illusory palinopsia. These symptoms typically occur during drug initiation or dose increase and resolve after drug discontinuation. Clomiphen citrate an infertility drug can result in palinopsia that may persist for years after use of the drug [13] [14]. The illusory palinopsia induced by drugs is mainly caused by its action on the serotonergic system and serotonin depletion leads to cortical hyperexcitability. In our case, a thorough history taking and inventory of medications, both past and present was done to rule out any influence of drug induced palinopsia. Also, the treatment for ischaemic stroke reduced the frequency and intensity of palinopsia. The face image disappeared in the first week while the arm image disintegrated gradually over the next few weeks.

Palinopsia in our patient was also not associated with visual snow which is characterized by the perception of TV static-like “snow” in the entire visual field [16]. The absence of vestibular symptoms and normal hearing function of this patient makes it unlikely to be associated with vestibular dysfunction which may provoke palinopsia in few cases [9]. Selective intra cerebral stimulation of the right lateral fusiform gyrus may result in partial or superimposed palinopsia on other images (facial palinopsia) [17]. Involvement of fusiform gyrus in stroke may result in facial palinopsia, *i.e.* an individual face percept superimposed on the non-face stimulus. This might explain the face image observed by our patient.

#### **4. Conclusion**

Palinopsia may be a presenting symptom of stroke. It is important for the clinician or Emergency room doctors to be alert to the possibility of undiagnosed structural neurological lesions like occipital stroke and differentiate it from non-organic causes, as prompt management may reduce the risk of visual or motor disability. Palinopsia is an unusual symptom with which many clinicians outside neurology may be unfamiliar and often requires interaction from multiple disciplines including neurology, eye care, and psychiatry. Those experiencing palinopsia often may be distressed and reluctant to describe their rather disturbing symptoms clearly to the attending physician. It is imperative for doctors to be aware of this condition, recognize associated factors and elicit the information by asking correct questions to the patient. In addition, it is important to address their concerns and reassure them that what they experience is a well-documented phenomenon and may be more common than it is perceived to be.

#### **Ethical Approval and Consent to Participate**

This work follows the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

Written informed consent was obtained from the patient for reporting and publication of this case report and any accompanying images and the relevant approval was obtained from the ethical committee of the University Hospital Sharjah.

### Consent for Publication

The consent for publication was obtained from both the patient and the University Hospital Sharjah ethical committee.

### Availability of Supporting Data

The data is stored in the electronic database of the University Hospital Sharjah under the confidentiality act.

### Authors Contributions

Dr. M.E was responsible for the literature review, neurological assessment of the patient, writing of discussion and references. Dr. M.A had evaluated the patient and did the ophthalmological work up personally. He contributed to manuscript writing in his area of expertise. Mrs. S.S and Mrs. N.S shared the effort of electrophysiology studies while all authors edited the English language of the manuscript and have approved the final draft of the manuscript before sending for publication.

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### Conflicts of Interest

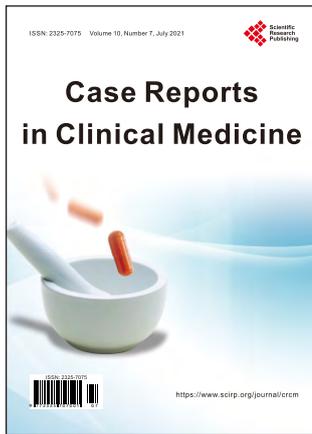
The authors have no competing interest to declare.

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