# نحوه اپروچ به خانم ۵۹ ساله با سرگیجه وضعیتی در درمانگاه پزشکی خانواده

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# شکایت اصلی (CC)

سرگیجه و سردرد وضعیتی به دنبال درازکشیدن

# شرح بیماری فعلی (HPI)

بیمار خانم ۵۹ ساله کیس شناخته شده T2DM و RAبا شکایت از سرگیجه و متعاقب آن سردرد به درمانگاه پزشکی خانواده مراجعه کرده است. سرگیجه بیمار از ۱ ماه پیش شروع شده و به این صورت است که پس از دراز کشیدن به پشت شروع شده و بیمار به صورت چرخش فضای اتاق دور سر، آن را توصیف می‌کند. سرگیجه در ابتدا همراه با تهوع (بدون استفراغ) بوده اما بعد از مدتی تهوع دیگر تکرار نشده است. بعد از مدت کوتاهی از شروع سرگیجه، سردرد شروع می‌شود. با تغییر وضعیت از خوابیده به نشسته، سرگیجه رفته رفته بهبود می‌یابد اما سردرد مدت طولانی‌تری باقی می‌ماند. سردرد بیمار از ناحیه پیشانی شروع شده و سپس به بالای سر و پشت گردن انتشار می‌یابد. بیمار سردرد خود را شدید و به صورت فشار از درون توصیف می‌کند. (بیشترین دردی که تا به حال تجربه کرده است) بیمار در اکثر اوقات سردرد را تنها با مصرف مسکن (استامینوفن کدئین) کنترل می‌کند. بیمار تنها محرک این حملات سرگیجه و سردرد را دراز کشیدن ذکر می‌کند. شروع سرگیجه بیمار در خوابیدن به پشت، راست یا چپ تفاوتی نمی‌کند. بیمار سابقه ضربه اخیر به سر را هم ذکر نمی‌کند.

# شرح بیماری فعلی (HPI)

بیمار کاهش شنوایی دو طرفه و درد گوش راست که به فک هم انتشار می‌یابد را ذکر می‌کند.

در یک سال گذشته بیمار سابقه کاهش وزن حدودا ۱۹ کیلوگرم را از ۶۵ کیلوگرم ابتدایی را هم ذکر می‌کند که اکنون مقداری افزایش یافته است.

بیمار همچنین سابقه چند ماهه «کم‌خونی» را هم ذکر می‌کند.برای آن تحت کولونوسکوپی قرار گرفته که ضایعه‌ای یافت نشده است.

# Review of Systems

General: Weight loss (65→46kg) / Night sweat ? / no known allergy

Endocrine: cold intolerance / no thirst / excessive urination

Neurologic: Headache / vertigo / tingling in feet

Eyes: low vision acuity / eye pain / cataract

Ears: right ear pain / bilateral hearing problem

Mouth: dry mouth / hoarseness

# Review of Systems

Lungs: no shortness of breath

Heart: no chest discomfort

GI: infrequent difficult swallowing of water / constipation / no black or red stool

# Past Medical History

- Rheumatoid Arthritis (از ۱۵ سال پیش)
- Type 2 Diabetes Mellitus (از؟ ۱۰ سال پیش)
- Anemia (از ۶ ماه پیش)
- Osteopenia
- ACS and PCI (۱۰ سال پیش)
- Proteinuria? (۳۰ سال پیش)
- metatarsal fracture of left foot

# Past Surgical History

- Cesarean section (۲۳ سال پیش)
- Appendectomy (۳۰+ سال پیش)
- Vulvar abscess drainage (۲ سال پیش)

# Medication History

- Prednisolone 5 mg
- Methotrexate 2.5 mg
- Alendronate 70 mg
- Aspirin 80 mg
- Folic acid 5 mg
- *Fefol plus 27 mg*
- acetaminophen codeine 300/20 mg

# Family History

سابقه سرطان پستان در خاله
سابقه دیابت در برادر و برادرزاده

# Physical Examination

Vital signs:

BP= 120/70 mmHg PR= 82/min RR=18 O2sat= 95%
T= 36.2

General: Weight= 53 kg

Cardiac: no murmur or click auscultated / S1 and S2 present

Pulmonary: clear on auscultation

# Physical Examination

## Neurologic:

Normal cranial nerves / normal gait / normal point to point movements / no pronator drift / Romberg positive / no tremor or fasciculation / normal muscle tone / normal muscle strength / no sensory problem / DTR: mostly hyporeflexia / no nuchal rigidity

HEENT: intact? tympanic membrane / no foreign body in ear canal / no sign of inflammation / pale palpebral conjunctiva

# Physical Examination

Abdomen: no tenderness / no mass or organomegaly on palpation / no distension

Skin: no petechiae

# Lab results

| CBC | 1404/3/9 0:17 | 1404/3/6 6:39 | 1404/3/5 15:14 |
|---|---|---|---|
| W.B.C | 4.6 | 5.0 | 5.8 |
| R.B.C | 3.8 | 3.6 | 3.47 |
| HGB | 10.6 | 10.1 | 10.6 |
| HCT | 34.2 | 32.9 | 32.1 |
| MCH | 27.89 | 28.06 | 30.55 |
| MCHC | 30.99 | 30.7 | 33.02 |
| MCV | 90 | 91.39 | 92.51 |
| PLT | 298 | 291 | 334 |
| Neut | 65.5 | 51.8 | 60 |
| Lymph | 29.4 | 44.2 | 38 |
| Mixed | 5.1 | 4.0 | 2 |
| RDW CV | 16.7 | 16.1 | 16.3 |
| MPV | 8.7 | 9.1 | |

| Hematology | 1404/3/6 6:39 |
|---|---|
| ESR | 125 |
| Retic count | 0.7 |

| Serology & Immunology | 1404/3/6 6:39 |
|---|---|
| cRP کمی | 2.3 |

# Lab results

| Biochemistry | 1404/3/9 0:17 | 1404/3/6 6:39 | 1404/3/5 15:14 |
|---|---|---|---|
| Urea | 31 | | 43 |
| Creatinine | 1.1 | | 1.2 |
| Na | 141 | | 143 |
| K | 4.5 | | 4.6 |
| ferritin | | 7 | |
| Ca | | 8.5 | |
| Phosphorus | | 3.8 | |
| LDH | | 400 | |
| Albumin | | 4.0 | |
| Fe | | 80 | |
| TIBC | | 353 | |

| | | | |
|---|---|---|---|
| Troponinکمی | | | <0.1 |
| SGOT | | | 15 |
| SGPT | | | 14.7 |
| ALk Phosphatase | | | 273 |
| CPK | | | 56 |
| CK MB | | | 15 |

# Lab results

| VBG | 1404/3/9 0:17 |
| --- | --- |
| PH | 7.46 |
| PCO2 | 46.4 |
| BEecf | 9.6 |
| PO2 | 31.0 |
| HCO3 | 33.5 |

# Lab results

| U/A | 1404/3/9 9:22 | | |
|---|---|---|---|
| Color | Yellow | Urobilinogen | Negative |
| Appearance | Clear | W.B.C | 0-1 |
| Specific Gravity | 1.010 | R.B.C | 0-1 |
| pH | 7 | EP Cells | 2-3 |
| Urine protein | Negative | Bacteria | Negative |
| Glucose | Negative | Mucus | Negative |
| Ketone | Negative | Crystals | Negative |
| Blood | Negative | | |
| Nitrite | Negative | | |
| Bilirubin | Negative | | |

# Imaging workups

سونوگرافي كامل شكم و لگن:

كبد با اندازه و شكل طبيعي رويت شد. اكوژنيسيته پارانشيم كبد نرمال مي باشد. مجاري صفراوي داخل و خارج كبدي و ورید پورت دیامتر نرمال دارند. كيسه صفرا فاقد اتساع با ضخامت جداري نرمال رويت شد. سنگ و اسلاج در داخل آن رويت نشد.

اندازه، اكوژنيسيته پارانشيمال و شكل طحال طبيعي مي باشد.span: 120mm

سونوگرافي از پانكراس و پاراآئورت در حد قابل بررسي ضايعه مشخصي رويت نگرديد.

اکوژنیسته پارانشیم کلیه چپ افزایش یافته رویت می شود.چک BUN/CRتوصیه می شود. کلیه چپ با سایز 90 mmو با ضخامت پارانشیم 10 mmرویت شد.(حداقل نرمال) کلیه راست با سایز 99 mmو با ضخامت پارانشیم 11mmرویت شد. تصویر کیست کورتیکال به دیامتر 11م.م در پل میانی کلیه چپ رویت شد. تصویر کیست کورتیکال به دیامتر 20م.م در پل تحتانی کلیه راست رویت شد. سنگ و هیدرونفروز و ضایعه فضاگیر Solidدرکلیه ها مشاهده نشد.

حجم و شكل مثانه طبيعي مشاهده شد. ضخامت جداري مثانه نرمال مي باشد.مثانه فاقد سنگ مشاهده شد.

رحم با نمای پوست منوپوزال رويت شد. ضخامت اندومتر thinرويت شد. هر دو تخمدان ابعاد و اكوي نرمال دارند.

تخمدانها با نمای آتروفیک رويت شدند. ضايعه پاتولوژيک در آدنكس هاي دو طرف مشاهده نشد.

مايع ازاد در حفره شكم و لگن مشاهده نشد.

# Imaging workups

سونوگرافي از هر دو برست و اگزيلاري هر دو سمت:

در بررسي از هر دو برست باپروب سطحي بافت فيبروگلاندولار برست با اكوي طبيعي مشاهده گرديد.

تصوير ضايعه هايپو اكو با حدود صاف و واضح با ابعاد 3*9 م.م در ساعت يک Far zoneبرست چپ رويت شد . (B3)

كيست ساده با ديامتر 3 م.م در ساعت دو و Near zoneبرست چپ ديده می شود. (B2)

در بررسي ناحيه retroareolar در هر دو قسمت ضايعه مشكوكي مشاهده نگرديد.

پوست و نيپل در هر دو طرف طبيعي است. شواهدي به نفع رتركشن و ديس تورشن بافتي مشاهده نشد.

در نواحي آگزيلاري دو طرف شواهدي به نفع لنفادنوپاتي پاتولوژيک ديده نشد.

# Problem List

۱- سرگیجه و سردرد

۲- آنمی

۳- آرتریت روماتویید

۴- دیابت ؟

۵- استئوپنی

۶- کاهش شنوایی

۷- رتینوپاتی دیاپتی و کاتاراکت

# Evaluation of the patient with vertigo: History

Time course — Vertigo is never a permanent, continuous symptom. Even when the vestibular lesion is permanent, the central nervous system adapts to the defect so that vertigo subsides over days or weeks. Constant vertigo lasting months is not vestibular (though constant imbalance can be). However, some patients describe constant dizziness but actually mean that they have a constant susceptibility to frequent episodic dizziness.

# Evaluation of the patient with vertigo: History

Vertigo can occur as single or recurrent episodes and may last seconds, hours, or days. This time course of symptoms provides one of the best clues to the underlying pathophysiology of vertigo.

Recurrent vertigo lasting under one minute is usually benign paroxysmal positional vertigo (BPPV).

A single episode of vertigo lasting several minutes to hours may be due to migraine or to transient ischemia of the labyrinth or brainstem.

The recurrent episodes of vertigo associated with Meniere disease or vestibular migraine also typically last hours but can be briefer.

More prolonged, severe episodes of vertigo that occur with vestibular neuritis can last for days. This is also characteristic for vertigo originating from multiple sclerosis or infarction of the brainstem or cerebellum.

# Evaluation of the patient with vertigo: History

Aggravating and provoking factors — All vertigo is made worse by moving the head. Many patients in the midst of a vertiginous attack may be extremely reluctant to move. If head motion does not worsen the symptoms, the patient is less likely to be experiencing vertigo. This feature does not distinguish among the various causes of vertigo.

Certain kinds of movements may increase suspicion of damage to the otoliths, the organs that detect linear accelerations to the head. These include imbalance provoked by stop–and–go movements of elevators or cars in traffic, as well as standing on a boat or floating dock.

Attacks of BPPV are often provoked by specific head movements or postures (eg, rolling over in bed, extending the neck).

# Evaluation of the patient with vertigo: History

When dizziness is provoked by static visual stimuli, such as patterned carpets or walls, or moving stimuli, such as sports or action movies on large screens, scrolling on the computer, or crowds at malls, this points to persistent postural perceptual dizziness (also known as visual vertigo, visuo–vestibular mismatch).

Recent hyperextension injury to the neck, usually with persistent neck pain, suggests the possibility of vertebral artery dissection with brainstem or labyrinthine ischemia.

Recent viral symptoms may suggest acute vestibular neuritis, which is believed to be produced by viral or postviral inflammation of the eighth cranial nerve. However, a history of recent viral illness is both nonspecific and insensitive; less than one–half of patients with vestibular neuritis will report this.

# Evaluation of the patient with vertigo: History

**Associated symptoms :**

- Acute vertigo due to a vertebrobasilar stroke is almost always accompanied by other evidence of brainstem ischemia such as diplopia, dysarthria, dysphagia, weakness, or numbness. However, infarction of the cerebellum may present as vertigo with no other symptoms. Focal neck pain may suggest vertebral artery dissection.

- Vertigo in patients with multiple sclerosis may also be preceded by or associated with other neurologic dysfunction, depending on the locus of demyelination.

- Deafness and tinnitus suggest a peripheral lesion of the inner ear. A sensation of aural fullness typically accompanies attacks of Meniere disease.

- Headache, photophobia, and phonophobia suggest migrainous vertigo. Many patients with migrainous vertigo will also experience visual aura in at least some of their attacks.

- Shortness of breath, palpitations, and sweating may suggest a panic attack but can occur with vertigo too. Vertigo is often so terrifying that such symptoms are not uncommon with vestibular disease.

# Evaluation of the patient with vertigo: History

**Prior medical history**

- A prior history of migraine suggests that this may be the etiology of vertigo.
- The presence of stroke risk factors such as hypertension, diabetes mellitus, smoking, and a history of vascular disease support a diagnosis of vertebrobasilar ischemia. Patients with an episode of vertigo and one or more risk factors for stroke have a substantial risk of subsequent stroke: an 8 percent two-year risk with one or two risk factors and 14 percent two-year risk with three or more risk factors.
- Past head trauma is a common antecedent to BPPV and persistent postural perceptual dizziness.
- Certain medications are associated with vestibular (eg, cisplatin, aminoglycosides) or cerebellar (eg, phenytoin) toxicity.

# Evaluation of the patient with vertigo: Examination

**Nystagmus:** In a patient with acute vertigo, nystagmus is usually visible with the patient looking straight ahead. If the lesion is peripheral, the fast phase is away from the affected side. Usually, nystagmus increases in frequency and amplitude with gaze toward the side of the fast phase, eg, leftward gaze increases left–beating nystagmus, if present (Alexander law).

Other features of nystagmus have localizing value for central versus peripheral vertigo

Type of nystagmus. A mixed horizontal–torsional jerk nystagmus results if a peripheral lesion affects all three semicircular canals or the vestibular nerve on one side. The horizontal fast phases beat toward the normal ear, as do the upper poles of the eyes for the torsional fast phases. The jerk nystagmus from peripheral disease occasionally appears purely horizontal, but it is never purely torsional or vertical. (Also, pendular nystagmus is never due to peripheral vestibular disease.) The jerk nystagmus with central lesions may have any trajectory.

Visual fixation tends to suppress nystagmus that is due to a peripheral lesion, but it does not usually suppress nystagmus from a central lesion. This can be tested with Frenzel lenses, which are large magnifiers that blur vision and prevent visual fixation. A peripheral lesion is likely if nystagmus increases when Frenzel lenses are in place.

# Evaluation of the patient with vertigo: Examination

Another way to test the effect of fixation is by covering and uncovering one eye during fundoscopy of the other. A peripheral disorder is likely if nystagmus increases on covering the fixating eye. It should be kept in mind that, in the ophthalmoscopic examination, the direction of nystagmus appears reversed because the optic nerve head is behind the center of eye rotation.

Testing nystagmus in different gaze positions can provide other localizing clues. In peripheral lesions, the predominant direction of nystagmus remains the same in all directions of gaze.

# Evaluation of the patient with vertigo: Examination

Balance and gait — The ability to stand or walk unsupported and the direction of falling may provide useful clues to the origin of vertigo, although it may be difficult to persuade a patient with severe vertigo to attempt to walk.

Unilateral peripheral disorders generally cause patients to lean or fall toward the side of the lesion. Patients may be uncomfortable and reluctant to move because of their vertigo, but they can still walk. Romberg testing will cause the patients to fall or tilt to one side.

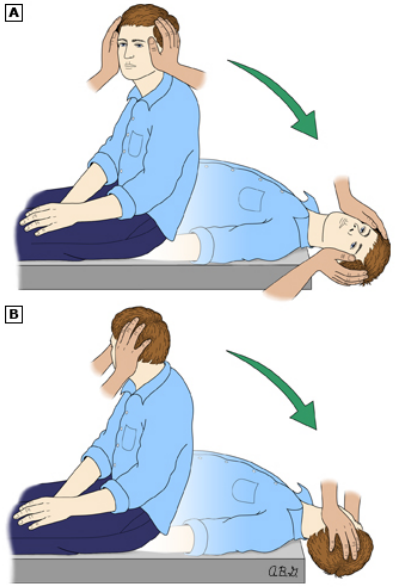
Patients with an acute cerebellar stroke are often unable to walk without falling. The direction of tilting or falling with Romberg testing may vary.

The sensitivity of balance testing may be increased by other maneuvers such as eye–closing, standing on foam, and performing head movements.

# Evaluation of the patient with vertigo: Examination

Dix–Hallpike maneuver — Positional maneuvers are designed to produce vertigo and elicit nystagmus in patients with a history of positional dizziness. These maneuvers are most useful in patients who do not have symptoms or nystagmus at rest and whose vertigo is episodic. The Dix–Hallpike maneuver tests for canalithiasis of the posterior semicircular canal, which is the most common cause of BPPV.

# Evaluation of the patient with vertigo: Examination




1. With the patient sitting, the neck is extended and turned to one side (panel A).
2. The patient is then placed supine rapidly, so that the head hangs over the edge of the bed, still turned to the side.
3. The patient is kept in this position and observed for nystagmus for 30 seconds.

*In patients with benign paroxysmal positional vertigo affecting the posterior canal, nystagmus usually appears with a latency of a few seconds and lasts less than 30 seconds. It has a typical trajectory, beating upward and torsionally, with the upper poles of the eyes beating toward the ground.*
*If the test is positive, the affected semicircular canal is the side to which the head is turned in step 1.*

4. After symptoms and nystagmus stop, the patient sits up.
5. The patient is again observed for nystagmus for 30 seconds.

*In patients with benign paroxysmal positional vertigo, the nystagmus may recur but in the opposite direction.*

6a. If nystagmus is provoked, the patient should have the maneuver repeated to the same (provoked) side; with each repetition, the intensity and duration of nystagmus will diminish. Testing of the other side is unnecessary.
6b. If nystagmus is not provoked, the maneuver is repeated with the head turned to the other side (panel B).
6c. If nystagmus is still not provoked, other subtypes of benign paroxysmal positional vertigo can be tested by appropriate maneuvers. Refer to UpToDate content for specific directives.

# Evaluation of the patient with vertigo: Diagnostic tests

Brain imaging — MRI of the brain is indicated in selected patients when the history and examination suggest either a central cause of vertigo or a vestibular schwannoma (acoustic neuroma). CT scans are significantly less sensitive for the diagnosis of cerebellar infarction and for pathologies affecting the brainstem or vestibular nerve.

In a patient with acute sustained vertigo, it is often difficult to distinguish between a vascular event involving the cerebellum and vestibular neuritis. While the latter has a benign course, the former can be acutely life threatening. Neuroimaging is indicated if the examination is not entirely consistent with a peripheral lesion, if there are prominent risk factors for stroke, if there are neurologic signs or symptoms, if the patient cannot walk, or if there is a new headache accompanying the vertigo .

# Evaluation of the patient with vertigo: Diagnostic tests

Audiometry - Audiometry is more sensitive than office testing to detect hearing loss and can quantify the loss at high and low frequencies. The audiometric battery also establishes if recruitment is present and tests for word recognition.

**Clinical features of common causes of vertigo***

| | Time course | Suggestive clinical setting | Characteristics of nystagmus¶ | Associated neurologic symptoms | Auditory symptoms | Other diagnostic features |
|---|---|---|---|---|---|---|
| **Benign paroxysmal positional vertigo** | Recurrent, brief (seconds) | Predictable head movements or positions precipitate symptoms | Peripheral characteristics | None | None | Dix-Hallpike maneuver shows characteristic findings |
| **Vestibular neuritis** | Single episode, acute onset, lasts days | Viral syndrome may accompany or precede vertigo | Peripheral characteristics | Falls toward side of lesion, no brainstem symptoms | Usually none | Head impulse test usually abnormal |
| **Meniere disease** | Recurrent episodes, last minutes to several hours | Spontaneous onset | Peripheral characteristics | None | Episodes may be preceded by ear fullness/pain, accompanied by vertigo, unilateral hearing loss, tinnitus | Audiometry shows unilateral low-frequency sensorineural hearing loss |
| **Vestibular migraine** | Recurrent episodes, last several minutes to hours | History of migraine | Central or peripheral characteristics may be present | Migraine headache and/or other migrainous symptoms either preceding, accompanying, or following vertigo | Usually none | Between episodes, tests are usually normal |
| **Vertebrobasilar TIA** | Single or recurrent episodes lasting several minutes to hours | Older patient, vascular risk factors, and/or cervical trauma | Central characteristics | Usually other brainstem symptoms | Usually none | MRI or MRA may demonstrate vascular lesion |
| **Brainstem infarction** | Sudden onset, persistent symptoms over days to weeks | As above | Central characteristics | **Usually** other brainstem symptoms, especially lateral medullary signs | Usually none; an exception is anterior inferior cerebellar artery syndrome | MRI will demonstrate lesion |
| **Cerebellar infarction or hemorrhage** | Sudden onset, persistent symptoms over days to weeks | Older patient, vascular risk factors, especially hypertension | Central characteristics | Gait impairment is prominent; headache, limb dysmetria, dysphagia **may** occur | None | Urgent MRI, CT will demonstrate lesion |

CT: computed tomography; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging; TIA: transient ischemic attack.

* For other diagnoses, refer to UpToDate topics on differential diagnosis of vertigo.

¶ Peripheral characteristics of nystagmus: horizontal or horizontal-torsional; suppresses with visual fixation; does not change direction with gaze. Central characteristics of nystagmus: may be horizontal, torsional, or vertical; does not suppress with visual fixation; may change direction with gaze.

# Evaluation of the patient with vertigo: Diagnostic approach

**Patients with acute–onset, sustained vertigo** — Common diagnoses in this setting include vestibular neuritis, demyelinating disease, and a stroke in the brainstem or cerebellum. The HINTS examination, in particular the head impulse test, can also be particularly useful in this setting to distinguish a peripheral cause of vertigo (eg, vestibular neuritis) from a central, cerebrovascular cause. In a young patient without cerebrovascular risk factors in whom a normal head impulse test suggests a localization within the central nervous system, multiple sclerosis might be more likely.

# Evaluation of the patient with vertigo: Diagnostic approach

**Patients with episodic vertigo** — Likely diagnoses in patients with episodic vertigo depend on the duration of events as well as the presence of associated features:

Very brief episodes of isolated vertigo that are precipitated by predictable movements or positions of the head are often caused by benign paroxysmal peripheral vertigo (BPPV). The Dix–Hallpike maneuver can help confirm this diagnosis.

The diagnosis of episodes with a longer duration (minutes to hours) may be further distinguished by the presence or absence of associated clinical features:

- Associated headache suggests vestibular migraine.
- Unilateral hearing loss, tinnitus, and ear fullness suggest Meniere disease.
- Other brainstem neurologic deficits suggest vertebrobasilar transient ischemia.

# سطوح پیشگیری

**Primordial Prevention**

**Primary Prevention**

**Secondary Prevention**

**Tertiary Prevention**

**Quaternary Prevention**

# Primordial prevention

- آموزش سبک زندگی فعال در سنین پایین برای پیشگیری از بی‌تحرکی مزمن
- ارتقای سلامت عمومی و تغذیه مناسب در جامعه برای پیشگیری از بیماری‌های عروقی و متابولیک (که ممکن است در سرگیجه نقش داشته باشند)
- سیاست‌های سلامت عمومی برای کاهش مصرف نمک و پیشگیری از فشارخون بالا

# Primary prevention

- آموزش افراد در معرض خطر (مثلاً افراد مسن) برای جلوگیری از سقوط یا حرکات ناگهانی گردن و سر
- ورزش‌های تعادلی برای حفظ عملکرد دهلیزی در سالمندان
- درمان زودرس بیماری‌هایی مثل فشارخون بالا یا دیابت برای جلوگیری از آسیب سیستم عصبی.

# Secondary prevention

- انجام مانور Dix–Hallpikeبرای تشخیص سریع BPPV
- ارجاع سریع به متخصص ENT یا نورولوژی برای تشخیص و مدیریت سرگیجه
- مانور Epley برای درمان زودرس BPPV و کاهش ناتوانی

# Tertiary prevention

- توانبخشی تعادلی برای بیمارانی که پس از درمان همچنان احساس ناپایداری دارند
- آموزش بیمار برای پیشگیری از افتادن و جراحات ناشی از سرگیجه
- استفاده از وسایل کمکی (مانند عصا) در موارد سرگیجه مزمن یا مکرر

# Quaternary Prevention

- جلوگیری از تصویربرداری‌های بی‌مورد مثل MRIیا CT-Scan در موارد BPPVواضح با مانور تشخیصی مثبت
- پرهیز از تجویز داروهای غیرضروری مثل بتاهیستین یا بنزودیازپین‌ها در مواردی که نیاز نیست
- آموزش بیمار درباره ماهیت خوش‌خیم بیماری برای کاهش اضطراب و تقاضای مداخلات غیرلازم

از توجه و حضور همگی شما عزیزان سپاسگزارم.