

HealthNews DIGEST

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Dr. Sherbaz Bichu

CEO & Specialist Anaesthetist
Aster Hospitals & Clinics, UAE

As we enter the new year of 2024, I want to recognize and celebrate the incredible work our doctors have done in the past year. I am pleased to welcome you to the 17th edition of the HealthNews Digest Newsletter on behalf of Aster's leadership. As always, I am amazed by your unwavering dedication and commitment to providing our patients with the finest care despite the challenges and obstacles.

The HealthNews Digest has evolved into an essential platform for Aster to maintain its commitment to clinical excellence and knowledge distribution. I am proud that Aster has successfully handled critical cases and provided clinical excellence to our patients.

Let us continue to build on our triumphs as we enter the new year and make even more significant strides in patient care. Your contributions to Aster and the healthcare industry as a whole are immeasurable; let us continue to strive for excellence in everything we do.

Happy, healthy, and successful 2024 to everyone!



Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics, UAE

As the Medical Director for Aster Hospitals and Clinics, I am honoured to welcome you to the 17th edition of the newsletter. As we begin the new year 2024, I am proud to reflect on our accomplishments in the previous year.

Our greatest achievement lies in handling critical cases with the utmost professionalism and expertise. I want to commend all of you for your tireless efforts in providing exceptional care to our patients, even in the most challenging situations. Your expertise, compassion, and tireless efforts have made all the difference in the lives of those who have entrusted us with their care.

We have also made significant progress toward maintaining our commitment to clinical excellence. I encourage you all to take the time to read through this newsletter and continue to provide your expertise and devotion in the upcoming editions to keep deepening our understanding of the medical field and to contribute to shaping healthcare's future.

I wish you all a prosperous, happy, and healthy new year!



Dr. Sandeep Janardan Tandel
General & Laparoscopic Surgery (Specialist)



Dr. Kranti Lohokare Jadhav
Obstetrics & Gynaecology (Specialist)

Adenomyosis and Endometriosis

Reviving Hope: Laparoscopic Surgery Triumphs over Uterine Adenomyosis and Endometriotic Ovarian Cysts at Aster Hospital, Sharjah

PRESENTATION

- 49 year old female
- P2L2 patient with uterine fibroid & bilateral ovarian cyst
- Medical history of type-2 Diabetes Mellitus and Hyperlipidemia
- Surgical history of Cystectomy 10 years back and Partial Thyroidectomy 4 years back
- History of 2 NVDs with last childbirth 16 years back
- Family history - mother diabetic
- Admitted with:
 - On and off severe abdominal pain
 - Negative tumour markers

FINDINGS

During Examination:

- Afebrile, conscious
- Pulse rate - 90/min
- BP - 122/86 mmHg
- Soft abdomen
- Mild vaginal spotting on periods
- BMD Test T Score - 2.0
- Pap Smear - Normal
- Bulky uterus of 10 weeks size

- Bilateral ovarian cysts densely adhered to the surroundings
- Pod dense adhesions of cysts with the bowel – 8x10 cm cyst on the right side and 6x7 cm on the left side
- Uterine fibroid 2.5x2.5 cm intramural in the anterior wall

Ultrasound Sonography (USG) showed:

- Enlarged uterus with a posterior wall leiomyoma
- Large bilateral ovarian multilocular cystic lesions with internal echoes extending into the Pouch of Douglas.

MRI showed:

- Bilateral large complex adnexal lesions with left one showing one haemorrhagic/ endometriotic component displacing the urinary bladder cranially – Differential Diagnosis, including complex cysts with left-sided hydrosalpinx.
- Bulky uterus with small subserosal fibroids with chronic cervicitis.

Mammography showed:

- Dense breasts – BI-RADS category 2

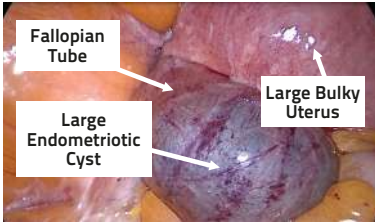
DURING PROCEDURE

After obtaining informed consent, the patient was admitted for Laparoscopic Abdominal Hysterectomy with Bilateral Salpingo-Oophorectomy and dense Adhesiolysis:

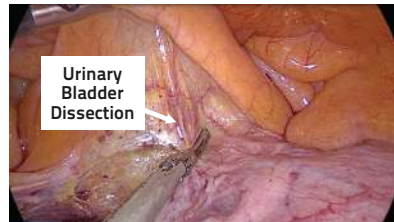
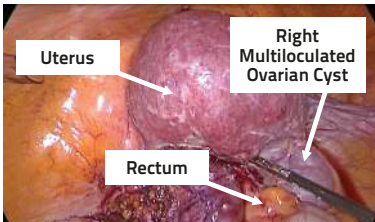
- Patient was placed in a semi-lithotomy position, and parts were painted and draped.
- The main port was inserted in the supraumbilical area with an open technique under vision.
- Uterus was found 10 weeks bulky with densely adhering cysts bilaterally. Right side - 10x8 cm simple cyst with 4x4 cm endometriotic cyst; left side – 6x7 cm simple cysts densely adhered to the pod and bowel.
- Dense adhesions were found in the pelvis where Endometriotic and other multiloculated cysts were densely adherent to the rectum posteriorly and laterally to the lateral abdominal wall, obscuring the ureter and iliac vessels. Anteriorly dense adhesions to the uterus were present. The bladder was pushed superiorly.
- Bilateral ovarian cysts were punctured, and adhesiolysis was done.
- Adhesiolysis started from the lateral pelvic wall, identifying the ureters and iliac vessels. The rectum was separated from the mass posteriorly.
- Bilateral round ligaments and infundibulopelvic ligaments were coagulated and cut with harmonic. Two leaves of broad ligament were separated, and bilateral uterine arteries were identified. The bladder was separated from the uterus and pushed down after separating.
- Bilateral uterine arteries were coagulated with bipolar cautery.
- Vaginal vault was opened anteriorly with monopolar cautery, and the uterus with cervix cut from the vagina with monopolar cautery.
- Specimen was cut as it was large and then removed from the vaginal vault.
- The vaginal vault was sutured with V-loc sutures.

- Haemostasis attained. The mop count was confirmed with an adequate 800 mL clear urine.
- Drain was kept at the left port, the main port was closed under vision, and the side ports were removed.
- Skin was sutured, and dressing was done.

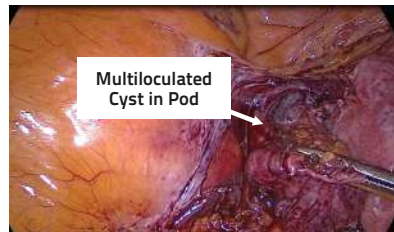
The specimen weighed 260 grams.



Dissection of Adnexal Cyst on Right side



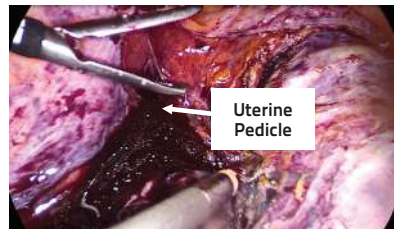
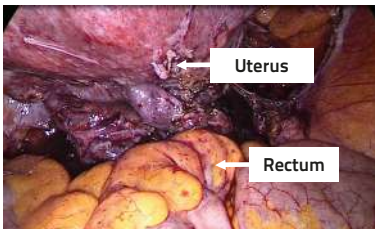
Urinary Bladder Dissection



Multiloculated Cyst in Pod

Dissection of Adherent Rectum from the Uterus

Left side Endometriotic and Multiloculated Cysts under Dissection



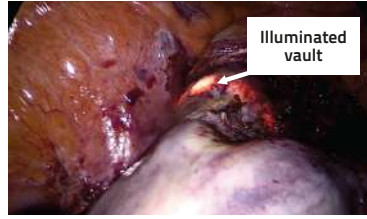
Uterine Pedicle

Uterus after releasing all adhesions from Lateral Pelvic Wall, Cysts and Rectum

Uterine Pedicle Identification and Coagulation



Uterus after Adhesiolysis - ready for Vault Opening



Vaginal Vault illuminated with Illuminator



Intra-corporeal cutting for Uterus Removal through Vault



Intra-corporeal Vault Closure with V-loc Suture

POST PROCEDURE

The patient withstood the procedure well and was discharged in a stable condition.

DISCUSSION

Ovarian endometriosis (endometrioma cysts) and adenomyosis are best treated with excision techniques in order to obtain effective pain relief. Laparoscopic excision of endometriosis may be accomplished by utilising different techniques, including sharp dissection and ultrasound scalpel.

With appropriate expertise and setting, a laparoscopic approach can manage moderate to severe endometriosis. Severe endometriosis involving the bowel, rectum and bladder may require a multidisciplinary team involving gynaecologists and general surgeons requiring significant expertise.

The removal of moderate to severe endometriosis can result in significant improvement in pain and quality of life.

CONCLUSION

With good expertise, teamwork, and communication with patients, moderate to severe endometriosis and adenomyosis can be done with a laparoscopic approach for better pain management and overall patient satisfaction.

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HFpEF

Insights into Diagnosing and Managing Heart Failure with Preserved Ejection Fraction (HFpEF)



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INTRODUCTION

Heart failure with preserved ejection fraction (HFpEF) is characterized by a left ventricular ejection fraction of 50% or higher along with clinical indicators and/or symptoms of heart failure, diastolic dysfunction, or structural anomalies in the left ventricle (LV) (1). HFpEF is highly prevalent and affects up to 50% of all HF patients and is associated with considerable morbidity and mortality (2). Rather than an isolated abnormality in left ventricular diastolic function, individuals with HFpEF exhibit a complex array of limitations in cardiac, vascular, and peripheral function (3). The current management strategies for HFpEF align with those for general HF management, involving the use of diuretics to reduce congestion and alleviate symptoms (2). Additionally, the specific causes should be identified and addressed for effectively managing contributing comorbidities (2). The complex interaction between ageing and comorbidities underscores the HFpEF as a significant public health burden (4).

This article discusses the diagnosis and management strategies for HFpEF, emphasizing the importance of addressing comorbidities and utilizing specific therapies for symptom relief and improved outcomes.

RISK FACTORS AND COMORBIDITIES

The clinical presentation of HFpEF results from a complex interplay of multiple risk factors, which leads to organ dysfunction and ultimately give rise to clinical symptoms (5). Cardiac comorbidities play a pivotal role in the pathogenesis of HFpEF through inflammatory endothelial activation (5). Beyond factors like gender and age, HFpEF is linked to a range of other comorbidities, as illustrated in Figure 1 (5). Collectively, these contributory elements have the capacity to induce a systemic state of inflammation (5).

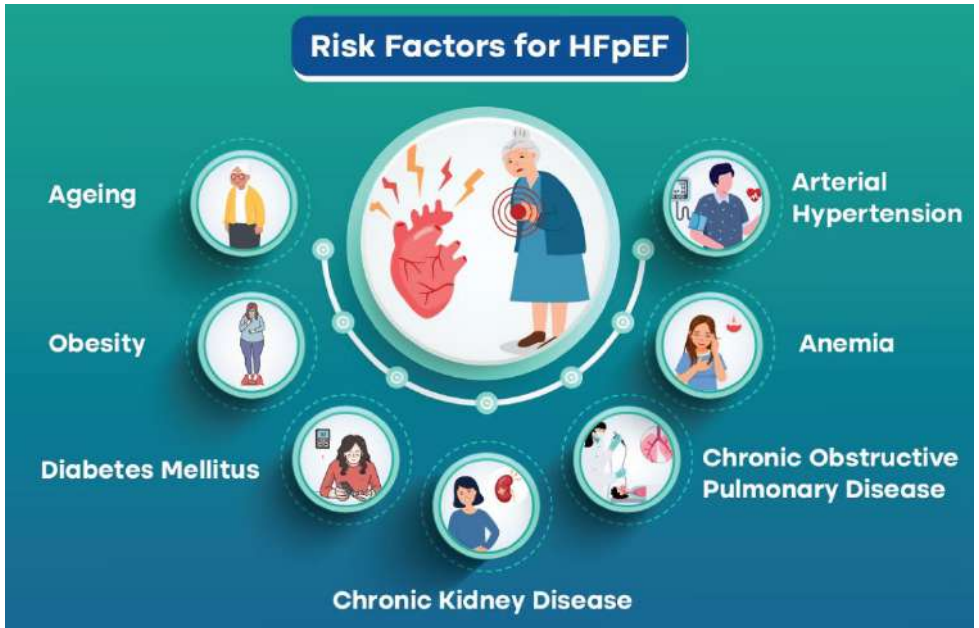


Figure 1. Risk factors contributing to the development of HFpEF (5)

DIAGNOSIS OF HFpEF

Several diagnostic criteria have been developed, however their sensitivities and specificities for diagnosing HFpEF vary greatly (6). More recently, two score-based algorithms, (H2FPEF and Heart Failure Association Pretest assessment, Echocardiography, and natriuretic peptide, Functional testing, Final etiology (HFA-PEFF), have been introduced to assist in the diagnostic process (6). Both scoring systems classify a significant portion of suspected HFpEF patients as having an intermediate likelihood, necessitating further diagnostic evaluations (6).

In order to facilitate widespread clinical application, European Society of Cardiology (ESC) guidelines recommend a simplified diagnostic approach starts with the evaluation of pre-test probability (6). The diagnosis should include the following components (6):

- Signs and symptoms associated with heart failure (6).
- LVEF >50% (6).
- Cardiac structural and/or functional abnormalities consistent with LV diastolic dysfunction or elevated LV filling pressures, including increased levels of Natriuretic Peptides (NPs) (6).

In the presence of Atrial fibrillation (AF), the left atrial (LA) volume index must be greater than 40 mL/m² (6). Regarding exercise stress testing, abnormal results are indicated by an E/e' ratio at

peak stress ≥ 15 or tricuspid regurgitation (TR) velocity at peak stress >3.4 m/s (6). In cases of diagnostic uncertainty, additional confirmatory tests such as cardiopulmonary exercise testing, exercise stress testing, and invasive haemodynamic testing should be used (6). Hemodynamic exercise testing involves measuring factors such as pulmonary capillary wedge pressure (PCWP) and left ventricular end-diastolic pressure (LVEDP) (6).

TREATMENT OF PATIENT WITH HFpEF

The approach to managing HFpEF involves the utilization of diuretics, the identification and addressing of specific underlying causes of HFpEF, and the management and treatment of associated comorbidities (2,7). Given the complex nature of HFpEF, a one-method-fits-all treatment strategy is ineffective (4). Therefore, a variety of therapeutic methods are essential, as illustrated in Figure 2 (4).

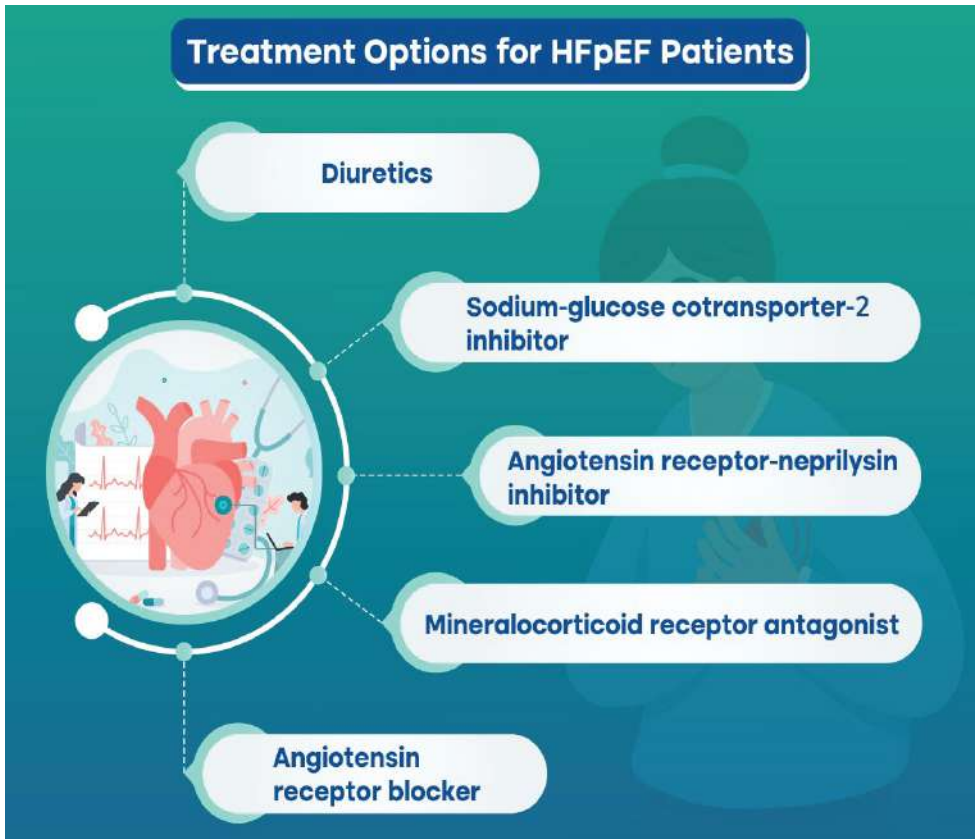


Figure 2. Treatment Methods for HFpEF (2)

DIURETICS

Diuretics are well-established medications for treating fluid overload and are regarded as a cornerstone in the symptomatic treatment of HFpEF (4). They effectively relieve HF symptoms and are frequently used regardless of LVEF (8). Patients with HFpEF are extremely sensitive to volume changes and have a small window between volume overload, which causes congestive symptoms, and hypovolemia (8).

The CHAMPION trial suggested the beneficial effect of diuretics (9). In this trial, the treatment group received daily pulmonary artery (PA) pressure data for HF management, resulting in a 46% lower HF hospitalization rate over 6 months compared to the control group (9,10). After 17.6 months, the rate was 50% lower (9). These findings indirectly substantiate the effectiveness of diuretics in mitigating morbidity in individuals with HFpEF (8).

SODIUM-GLUCOSE COTRANSPORTER 2 INHIBITOR (SGLT2i)

SGLT2 inhibitors hold substantial promise in addressing various pathophysiological abnormalities identified in HFpEF (11). Clinical trials have provided evidence that SGLT2 inhibitors can effectively reduce epicardial adipose tissue, irrespective of their impact on a patient's body weight (11). Additionally, these inhibitors exhibit the capacity to mitigate inflammation within the adipose tissue surrounding the heart and major blood vessels, while also addressing issues associated with cardiac filling and aortic distensibility in HFpEF cases (11).

In the EMPEROR-Preserved trial, which investigated the effects of empagliflozin in symptomatic HFpEF patients, regardless of their diabetic status (12). When compared to a placebo, empagliflozin therapy led to a significant reduction in the primary endpoint, which was a composite measure of cardiovascular death or hospitalization due to HF (12). Notably, these therapeutic benefits remained consistent across HFpEF patients with and without diabetes (12).

ANGIOTENSIN RECEPTOR-NEPRILYSIN INHIBITOR (ARNi)

The addition of neprilysin inhibition to Angiotensin receptor blockers, has shown promising results in improving atrial and ventricular myopathy in patients with HFpEF (4). The PARAMOUNT-HF trial investigated the effectiveness and safety of ARNi in which patients were randomly assigned to ARNi or valsartan (13). Sacubitril-valsartan demonstrated superior results in reducing NT-proBNP levels after 12 weeks of treatment compared to valsartan, and it was well-tolerated (13).

In addition, a post hoc analysis revealed that individuals who had experienced a more recent episode of HF hospitalization experienced greater advantages from sacubitril-valsartan, while there was no benefit observed among patients with no prior hospitalization (10).

ANGIOTENSIN RECEPTOR BLOCKERS (ARBs)

ARBs have significance in treating adipose tissue inflammation as well as mitigating myocardial fibrosis and remodeling (4). In the CHARM Preserved trial, patients with LVEF more than 40% were randomly assigned to candesartan or placebo (14). The primary objective was to identify the occurrence of cardiovascular mortality or CHF hospitalisation (14). Candesartan considerably reduced CHF-related hospital admissions in patients with HF and LVEF higher than 40% (14).

MINERALOCORTICOID RECEPTOR ANTAGONISTS (MRAs)

MRAs have shown promise in enhancing diastolic function in HFpEF patients (2). The impact of an MRA, spironolactone was studied in TOPCAT study for HFpEF patients (2). The results revealed a modest reduction (HR, 0.89) in the combined occurrence of death, aborted cardiac death, and HF hospitalization (2). However, there was a statistically significant reduction in HF hospitalization (HR, 0.83) was observed in the treatment group (2). Furthermore, another post hoc analysis indicated that spironolactone's effectiveness may be most pronounced among patients with lower LVEF values (2).

GUIDELINES FOR THE MANAGEMENT OF HFpEF

Despite its increasing prevalence and economic burden, treatment options for HFpEF are limited, and because patients are older, highly symptomatic, and have a reduced quality of life, the goal of therapy is primarily symptom relief and improve their quality of life (15). Figure 3 summarizes the guidelines by American Heart Association (AHA)/American College of Cardiology (ACC)/Heart Failure Society of America (HFSA) and European Society of Cardiology (ESC) for managing HFpEF (2).

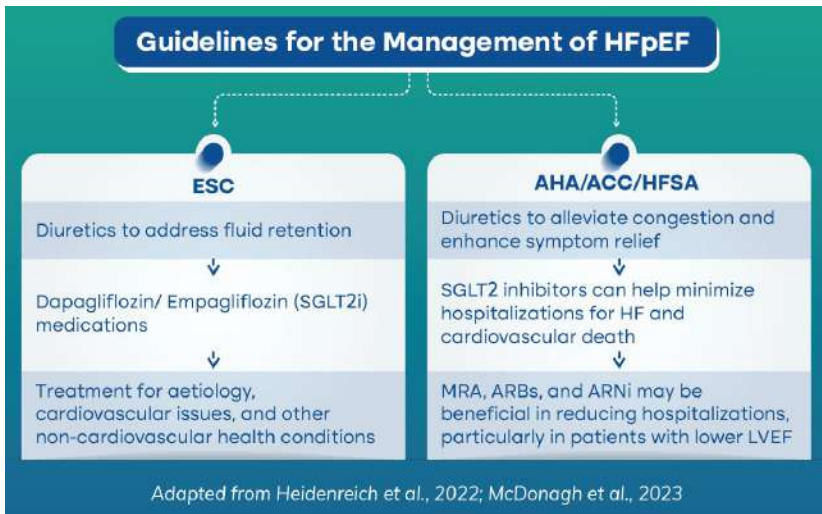


Figure 3. Medication recommendations for HFpEF (2,7)

Key Highlights

HFpEF is characterized by the preservation of left ventricular ejection fraction (LVEF \geq 50%) and involves intricate cardiac, vascular, and peripheral limitations, highlighting a substantial public health challenge (1).

The European Society of Cardiology (ESC) recommends a simplified diagnostic approach, including the assessment of signs and symptoms of heart failure, LVEF > 50%, and objective evidence of cardiac abnormalities (6).

Managing HFpEF necessitates a multifaceted approach, encompassing the management of comorbid conditions and the use of diuretics, as well as other guideline-directed medical therapies such as SGLT2 inhibitors, ARNi, ARBs, and MRAs (2,16).

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Post-Traumatic Osteoarthritis

A case of Post-Traumatic Osteoarthritis treated with Total Elbow Arthroplasty successfully at Aster Hospital, Mankhool



Dr. Alexis Jude Dominic Xavier
Orthopaedics (Specialist)

PRESENTATION

- 47 year old female
- Medical history of hypertension
- No family history of medical illness
- History of slip and fall at home, following which patient had pain and swelling in the right elbow
- Admitted with:
 - Complaints of right elbow pain and stiffness for one year
 - Severe pain for the last two weeks interfering with daily activities, even difficulty in moving hands to eat food

FINDINGS

During Examination:

- Pain and tenderness around the right elbow palpable on both sides of the olecranon.
- Range of movement - 30 - 80° only
- Supination and pronation - 0

DURING PROCEDURE

The patient underwent Total Elbow Arthroplasty under General Anaesthesia:

- Parts scrubbed, painted, and draped in the supine position in bolster under the ipsilateral shoulder.
- The skin and subcutaneous tissues were incised through the posterior incision centred between the olecranon and medial epicondyle.
- Proximally, the ulnar nerve was isolated and separated with umbilical tape, dissected free of its adhesions and branches, and transposed anteriorly.
- The triceps attachment was elevated sub-periosteal and reflected laterally.
- The collateral ligaments were released, and the elbow was dislocated.
- The humeral canal was opened, and the cuts were made.
- Sizing was done and found to be 4. The ulna was broached after opening up the canal.

- The trial reduction was done, and full flexion, extension, pronation, and supination was done and found to be full.
- Actual implants were fixed with cement, and the movement was reassessed.
- The triceps was reattached.
- Wound was closed in layers over the drain.

POST PROCEDURE

The patient tolerated the procedure well and was in a stable condition on discharge. The pain was reduced post-operatively. She was started on active elbow mobilization and the ROM (range of motion) was 10 to 100 degrees at 2 months post-op.

DISCUSSION

Total elbow replacement, or total elbow arthroplasty, is a surgical procedure designed to alleviate pain and restore function in severely damaged elbow joints. Typically performed for conditions like arthritis or fractures, the surgery involves replacing the damaged joint surfaces with artificial components made of metal and plastic. This procedure aims to improve joint movement, reduce pain, and enhance overall elbow functionality.

Recovery involves physical therapy to regain strength and mobility. While effective, complications such as infection or prosthesis loosening may occur. Patient selection and postoperative care play crucial roles in the success of total elbow replacement, with many individuals experiencing significant improvements in their quality of life.

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BPPV

Beyond Benign Paroxysmal Positional Vertigo (BPPV): Treatment and Management Approaches for Lateral and Anterior Canal BPPV



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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most prevalent form of peripheral vestibular vertigo (1). This condition is marked by abrupt vertigo episodes triggered by some postural head movements, such as upward head movement, turning over from one side to another, or standing up after sitting (2). Vertigo is triggered by the displacement of otoconia crystals into the semicircular canals of the inner ear or their attachment to the cupula (1). It is the most common cause of vertigo/ dizziness in elderly patients in their sixties, predominantly in women (1). Even though benign, BPPV significantly disrupts patients' daily routines and overall well-being (1).

This article aims to provide insights into the diagnosis and treatment approaches of lateral canal BPPV and anterior canal BPPV.

TYPES OF BENIGN PAROXYSMAL POSITIONAL VERTIGO:

Posterior canal BPPV (PC-BPPV) is the most common form of positional vertigo corresponding to 60-70% of cases of BPPV (3,4). Other forms of BPPV include lateral or horizontal canal BPPV (LC-BPPV), and anterior canal BPPV (AC-BPPV) (1). PC-BPPV and LC-BPPV share similar symptoms but LC-BPPV is characterized by more severe and intense attacks with an incidence of 5-14% (3,5). AC-BPPV is a rare type of BPPV with a frequency of 1-2% (6). Modified diagnostic and treatment maneuvers of PC-BPPV are used in AC-BPPV (7). A pictorial representation of the three types of BPPV relative to the otolith positions in the ear canal is depicted below:

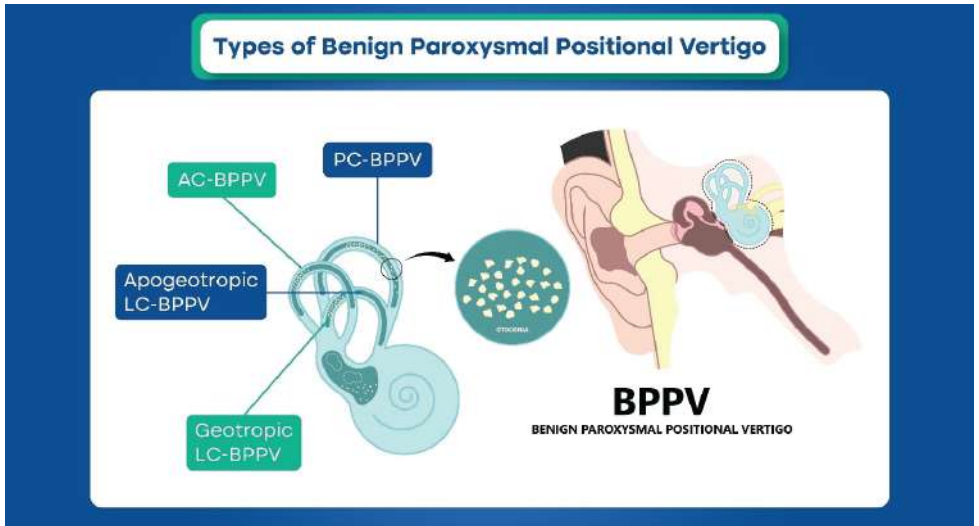


Figure 1: Types of Benign Paroxysmal Positional Vertigo (1)

1. POSTERIOR CANAL - BENIGN PAROXYSMAL POSITIONAL VERTIGO (PC-BPPV):

The posterior canal is most affected as its positioned lower than the other canals, especially in the right ear (4). There are two probable mechanisms of vertigo: canalithiasis or geotropic (otoliths in canal) and cupulolithiasis or apogeotropic (otoliths in cupula), they bring abnormal stimulation of vertigo and nystagmus relative to head movement (4).

2. LATERAL CANAL - BENIGN PAROXYSMAL POSITIONAL VERTIGO (LC-BPPV):

LC-BPPV cases occur spontaneously, it may also occur due to canal conversion or as a complication after treatment for PC-BPPV (3). There are two types of LC-BBPV:

Geotropic LC-BPPV causes the otoliths to move towards the affected ear causing an ampulletal excitatory current causing a nystagmus beating towards the affected ear (5). When the head is turned towards the unaffected ear the otoliths move away from the ampulla causing an ampulofugal inhibitory current and the nystagmus beats towards the unaffected ear (5). The nystagmus is more intense towards the affected ear (5).

The reverse phenomenon occurs in ageotropic LC-BPPV, the otoliths move away from the affected ear and show an ampulofugal inhibitory endolymphatic current, and the nystagmus beats toward the unaffected ear (5). When the head is turned towards the unaffected ear, the otoliths move towards the ampulla and shows an ampulletal excitatory endolymphatic current causing the nystagmus to beat with less intensity towards the affected ear (5).

DIAGNOSIS:

The diagnosis of LC-BPPV involves assessing the otolith's movements in the horizontal or lateral canals of the inner ear (5). There are several diagnostic maneuvers which are used for diagnosing LC-BPPV and its subtypes, these differ in the degree of head movement change from the supine to upright position (5). As the basic mechanism of LC-BPPV are common the differing features of the diagnostic tests are elaborated below:

1) McCLURE-PAGNINI TEST:

The patient is made to turn the head to each side at 90° angle in the supine position (5). It may be difficult to identify the intensity of nystagmus in this test, hence other methods are utilized (5).

2) SEATED SUPINE POSITIONING TEST:

In this test, the patient is made to go from seated to supine position with the head flexed at 30° (5). In geotropic LC-BPPV the otoliths move from the posterior towards the utricle and away from ampulla (5). It leads to an ampullofugal inhibitory endolymphatic current and causes a nystagmus beating towards the unaffected ear (5). In apogeotropic LC-BPPV, the particles are in the anterior arm of LC or adhered to the cupula and move towards the ampulla (5).

3) BOW AND LEAN TEST:

The nystagmus is noted by asking the patient to bend the head over 90° and lean backwards over 45° in the sitting position (5). In this maneuver, the otoliths move toward the ampulla while bowing and away from the ampulla when leaned backwards (5). Conversely, in apogeotropic LC-BPPV, the otoliths move away from the ampulla while bowing and towards the ampulla in the lean test (5).

4) PSEUDO-SPONTANEOUS NYSTAGMUS (PSN):

Some patients may experience PSN, which can be differentiated by the bow and lean test in the sitting position (5). The PSN increases when the head is bent over at 30° and reverses when bent over at 60° (5). The nystagmus may be triggered by head's slow rotation (5).

TREATMENT:

A) APOGEOTROPIC LC-BPPV:

The objective for treating this variant is to detach the otoliths in the cupula (in cupulolithiasis) and remove them from the anterior arm and move them towards the utricle (5). There are several repositioning treatments available for LC-BPPV:

Lateral canal - Benign paroxysmal positional vertigo (LC-BPPV):

Treatment Approaches for Apogeotropic Lateral Canal BPPV

Test	Method	Remarks
Appiani Modification of Gufoni's Maneuver	This maneuver consists of quickly moving the patient from sitting position to the affected side and swift 45° upward turn and finally returning to the sitting position (8).	The brisk movements helps to detach the otoliths and direct them towards the posterior arm (8). This method also helps in converting apogeotropic type to geotropic LC-BPPV (8). This is followed with a proper Gufoni maneuver to complete the repositioning (5).
Modified Sémont Maneuver	In this method, the patient is brought from the sitting position to the side-lying position of the affected side followed by downward head turn at 45° (8).	It combines the effect of inertia and gravitational forces to detach the otoconia (8).
Cupulolith Repositioning Maneuver (CuRM)	The patient is made to rotate the head 135° to the affected side while in the supine position, and mastoid oscillation is applied to the affected site for 30 seconds by a hand-held 60 Hz hand-held vibrator (8). The patient is turned 45° to the healthy side and again 90° on the same side (8). For the apogeotropic variant, the patient's head is turned 90° to the healthy side and oscillation is applied again and the patient is slowly brought to the sitting position (8).	This method also targets cupulolithiasis by mastoid oscillation and gravitational forces for detaching the otoliths and move them towards the canal from the utricle (8).
Zuma Maneuver	The patient is made to go quickly from sitting position to the lying down position on the affected side, and held for 3 minutes (8). Followed by rotating the head 90° towards the ceiling for another 3 minutes (8). The patient is then positioned into dorsal decubitus and the head is turned 90° towards the unaffected side and held for another 3 minutes (8). Lastly, the patient's head is tilted slightly forward and slowly returned to the sitting position (8).	This method also combines inertial and gravitational forces for detaching the otoconia (8). Zuma can be done for both geotropic and apogeotropic vertigo (8).

Figure 2: Treatment Methods for Apogeotropic LC-BPPV (5,8)

B) GEOTROPIC LC-BPPV:

Geotropic LC-BPPV is characterized by free floating otoliths in the posterior arm of the LC, hence the treatment aims to move the particles towards the utricle (5) .

Lateral canal - Benign paroxysmal positional vertigo (LC-BPPV):		
Treatment Approaches for Geotropic Lateral Canal BPPV		
Test	Method	Remarks
Forced Prolonged Position	The patient is made to turn their head/whole body to the unaffected side in the supine position (8). The position should be maintained for 12 hours to facilitate otolith movement (8).	This maneuver may be difficult to perform in elderly patients and in patients with skeletal and cardiac diseases (8).
Gufoni Maneuver	This method differs from the apogeotropic type wherein the patient is quickly turned to unaffected side from the sitting to the supine position. The patient's head is quickly downward by 45° and held for 2 minutes (8). The patient is slowly brought to the sitting position (8).	As this movement is done on the unaffected side, less intense vertigo maybe experienced (8).
Head Shake Maneuver	This maneuver is performed with the patient in the supine position with head bent at 30° (8). The physician then shakes the head at an angle of 30° to the left and right for 15 seconds at a rate of 2 Hz (8).	The rapid movements of this maneuver are intended to break the otolith crystals and detach them from the cupula (3,8).
Lempert maneuver (Log Roll/ Barbecue Maneuver)	The patient is initially in the supine position followed by 270° head rotation to the unaffected in three 90° turns at 30-s intervals (9).	If the nystagmus doesn't stop, multiple roll maneuvers may be performed (10).

Figure 3: Treatment Methods for Geotropic LC-BPPV (3,8–10)

3. ANTERIOR CANAL BENIGN PAROXYSMAL POSITIONAL VERTIGO (AC-BPPV):

AC-BPPV is the rarest form vertigo, the anterior canal is positioned higher than the posterior and horizontal canal which makes it unlikely for the otoconia's to reach the anterior canal against gravity (7). Moreover, its anatomical orientation facilitates spontaneous clearance of the otoliths (7).

DIAGNOSIS:

AC-BPPV is significant for vertical downbeat nystagmus towards the affected side when evoked by Dix Hallpike test (7). The torsional component is often less intense and unclear which makes it difficult to differentiate from posterior canal down beating BPPV (7). Central nervous system disorders can cause down beat nystagmus during positional tests and should be excluded from peripheral down-beating nystagmus (7).

1) DIX-HALLPIKE MANEUVER:

The patient is made to sit with legs hanging over the table and is quickly brought to the supine with the neck extended over the edge of the table and observed for nystagmus (2). If the otoliths are suspected in the right ear, the head is turned 45° to the right and towards the left if otoliths are suspected in the left ear, after 60-s the patient is returned to upright position (with head still turned) and checked again for nystagmus (2).

In this test, the otoliths block the canal, causing vertical down beating nystagmus towards the unaffected ear (2). No inversion of the nystagmus is observed when the patient returns to the sitting position (2).

2) SUPINE HEAD HANGING TEST:

The patient is initially in the supine position with the head extended over the table edge, he is observed for 60-s for downbeat nystagmus (11). In this test, torsion is directed towards the affected eye (11). Downward vertical nystagmus confirms the presence of AC-BPPV (11).

TREATMENT:

As the posterior and anterior canals are coplanar, reversed maneuvers of PC-BPPV are used for treating AC-BPPV (7). The reverse treatment maneuvers are started on the healthy side (7).

Anterior Canal Benign Paroxysmal Positional Vertigo (AC-BPPV):

Treatment Approaches for Anterior Canal BPPV

Test	Method	Remarks
Reverse Epley Maneuver	The patient is initially in the seated position and made to turn their head 45° to the affected side (7). The patient briskly brought to the supine position with head still turned and extended downward (7). The head is now turned 45° to the unaffected side and body is supported on the left side (7). Finally the patient is brought to the seated position with head still turned (7).	The patient's eyes should be observed for nystagmus consistent with Lempert maneuver and Dix Hallpike maneuver (7). Consistent pattern indicates insufficient movement of the otoconia (7).
Yacovino Maneuver	The maneuver begins with the patient in upright position, the head is brought to the head hanging position by 30°, the head is elevated so that the chin turns the chest and finally returned to the sitting position (7). The patient is maintained for 30-s at each step (7).	This method inverts the anterior canal which facilitates the movement of the crystals ampullofugally (7). However, there is risk of the crystals entering the posterior canal and canal switch during this maneuver (7).
Short Canal Repositioning Maneuver	The patient is seated in the upright position with head turned 45° to the affected side (7). The head is brought to 40 head hanging position (7). With the head still hanging, the head is turned to the healthy side (7). Lastly, the patient is brought back to the upright position (7).	The short CRP maneuver is a variation of Epley maneuver; it can be performed after determining the side of involvement (7).

Figure 4: Treatment Methods for AC-BPPV (2,7)

Key Highlights

- BPPV causes recurrent vertigo attacks when the head's position is changed during everyday actions (5). It may be caused by idiopathic factors or due to other concurrent risk factors (5).
- Each type of BPPV can be diagnosed by stimulating the otolith movement and observing the nystagmus movement (12). Specific nystagmus patterns are induced by the diagnostic maneuvers (12).
- There is no single correct treatment maneuver for treating each of the BPPV types, selecting a suitable strategy will depend on the physician's experience and patient's clinical presentation (5).
- The otolith repositioning methods depend on inertia and gravity that facilitate detachment and movement of the otoconia crystals (12).
- BPPV may present as an acute vestibular syndrome where symptomatic medication may be needed along with repositioning maneuvers (13).

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DVT and PE

A life-threatening case of Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE) treated successfully at Aster Cedars Hospital and Clinic, Jebel Ali



Dr. Birjis Shaikh
Internal Medicine (Specialist)

PRESENTATION

- 28 year old male
- No medical history of Diabetes Mellitus / Hypertension / Dyslipidemia
- History of chicken pox 10 days back and received symptomatic care
- No surgical history
- Admitted with:
 - Pain in both lower limbs (left>right)
 - Chest pain on the 3rd day after admission

FINDINGS

On Examination:

- Vitals: Pulse 80/min, BP 128/86 mmHg, SO2 99% on room air
- Severe tenderness on the left calf muscle (pain scale 7/10)
- Swelling of the left leg more than the right leg
- Peripheral pulses palpable
- CVS: Sinus Rhythm; normal S1, S2, no murmur
- RS: Clear bilateral air entry, no adventitious sounds, no tachypnea

On Investigations:

- CBC: within normal limits
- CRP: High (26.3; normal <5 mg/dL)
- D- dimer: High (8681; normal <500 ng/mL)

Days	D-dimer Levels
On admission	8681
Day 2	3066
Day 6	2475

- Coagulation profiles: Normal
- Protein C and S activity: within normal limits
- Cardiac enzymes: within normal limits

Doppler Scan for lower limbs:

- Bilateral popliteal and left posterior tibial veins had acute to subacute thrombosis. The right posterior tibial vein had partial thrombosis.

CT Pulmonary Angiography:

- Acute thrombus with partial filling defects involving bilateral main pulmonary arteries extending into both lower lobar arteries, predominantly posterobasal segmental arteries - suggestive of Acute Pulmonary Thromboembolism.
- Mildly dilated main pulmonary trunk
- Bilateral both pulmonary arteries, right atrium and right ventricle noted.
- Mild left ventricular thickening noted.
- Maximum diameter of the pulmonary trunk is 27 mm. The right pulmonary diameter measured 18 mm, and the left was 17.5 mm.
- Bilateral pulmonary veins appeared normal and were seen draining normally to the left atrium.

2D Echocardiography:

- No RMWA
- Good LV Systolic Function
- Normal valves
- RA/RV not dilated
- No clot/pericardial effusion

DIAGNOSIS

Deep Vein Thrombosis with Acute Pulmonary Embolism.

TREATMENT

Inj. Clexane 0.6 mL SC BID for 7 days and Tab. Xarelto 15 mg BID for 1 month, followed by 15 mg OD for 6 months.

COURSE IN HOSPITAL

The patient was admitted to the ICU for observation. He was started on Clexane 0.6 twice a day. He was having chest pain on and off during his stay. His vitals were stable throughout the admission. His 2D echocardiography was measured serially for RA/RV measurement. He was given symptomatic treatment with pain relievers. His coagulation profiles were also measured serially.

FOLLOW UP

The patient started improving, pain reduced and was discharged home. He was discharged with Tab. Xarelto (Rivaroxaban) 15 mg BID for 4 weeks, then once a day for 3 months.

DISCUSSION

Initial Resuscitation:

The initial approach to patients with **suspected** Pulmonary Embolism (PE) should focus on stabilizing the patient while clinical evaluation and definitive diagnostic testing are ongoing.

Respiratory And Haemodynamic Support:

- **Empiric Anticoagulation** – For patients with suspected PE who are haemodynamically **stable** or haemodynamically unstable and successfully resuscitated, the administration of empiric anticoagulation depends upon the risk of bleeding, the clinical suspicion for PE, and the expected timing of diagnostic tests:
 - For patients with a **low** risk of bleeding and a high clinical suspicion for PE, empiric anticoagulation rather than waiting until definitive diagnostic tests are completed.
 - Do not anticoagulate patients with **absolute contraindications** to anticoagulant therapy or those with an **unacceptably high** risk of bleeding.
 - For patients with a **moderate** risk of bleeding, empiric anticoagulant therapy may be administered on a case-by-case basis according to the assessed risk-benefit ratio.
- **Definitive therapy for suspected PE** – In patients with a high clinical suspicion of PE who are haemodynamically **unstable** and who have a definitive diagnosis by portable perfusion scanning or a presumptive diagnosis of PE by bedside echocardiography, systemic thrombolytic therapy rather than empiric anticoagulation or no therapy:
 - If bedside testing is delayed or unavailable, the use of thrombolytic therapy as a life-saving measure should be individualized; if not used, the patient should receive empiric anticoagulation.

For patients who are haemodynamically unstable and the clinical suspicion is low or moderate, empiric anticoagulation is similar to that suggested for haemodynamically stable patients; empiric thrombolysis is not justified in this population.

- **Definitive therapy for confirmed PE** – For patients in whom the diagnostic evaluation **confirms** PE, an approach that is stratified according to whether or not the patient is haemodynamically stable or unstable.
 - **Haemodynamically stable low-risk/Non-massive PE** – For most haemodynamically **stable** patients with PE that is low risk/non-massive, the following applies:
 - For those in whom the risk of bleeding is low, anticoagulant therapy be initiated or continued
 - Outpatient anticoagulation is safe and effective in selected patients at low risk of death, provided that they do not have respiratory distress, serious comorbidities, or require oxygen or narcotics and that they also have a good understanding of the risks and benefits of such an approach.
 - **Haemodynamically stable intermediate-risk/Submassive PE** – For most haemodynamically **stable** (i.e., normotensive) patients with intermediate-risk/submassive PE, anticoagulation should be administered, and patients monitored

closely for deterioration, observation with serial lower extremity ultrasonography may be appropriate.

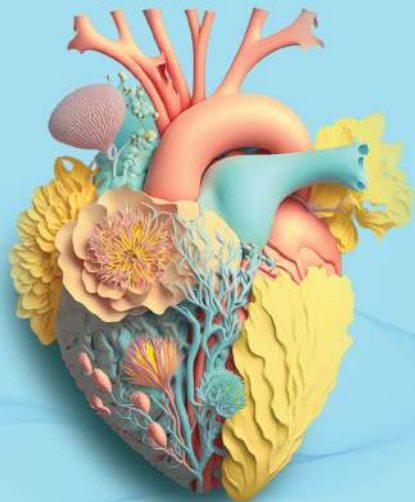
- For those who have contraindications to anticoagulation or have an unacceptably high bleeding risk, an inferior vena cava (IVC) filter be placed rather than observation.
- **Haemodynamically unstable PE** – For most patients with haemodynamically **unstable PE**, the following applies:
 - **No contraindications to thrombolysis** – For patients with refractory hypotension and without contraindications to thrombolysis, systemic thrombolytic therapy followed by anticoagulation rather than anticoagulation alone
 - **Contraindications to thrombolysis** – For those in whom thrombolysis is contraindicated, catheter or surgical embolectomy rather than observation.
- **Adjunctive therapies** – In patients with PE who are fully anticoagulated, early ambulation rather than bedrest, when feasible
- **Prognosis** – PE, left untreated, has a mortality rate of up to 30%, which is significantly reduced with anticoagulation. The highest risk occurs within the first seven days, with death most commonly due to shock.
- **Follow-up** – Patients treated with unfractionated heparin and/or warfarin should be monitored for laboratory evidence of therapeutic efficacy. Patients should also be monitored for early (e.g., recurrence) and late (e.g., chronic thromboembolic pulmonary hypertension) complications of PE and for the complications of anticoagulation and other definitive therapies. In addition, patients should be investigated for the underlying cause of PE.

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