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of Medical Research: I

Surgeries and Cardiovascular System



Radial Dome Osteotomy

Cardiac Lymphoma Revealed

Highlights

Complicating Lung Cancer

Papillary Muscle Hypertrophy

Discovering Thoughts, Inventing Future

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Contents of the Issue

i. Copyright Notice
ii. Editorial Board Members
iii. Chief Author and Dean
iv. Contents of the Issue

1. Severe Iatrogenic Lutembacher Syndrome in a Young Male: Case Report and Literature Review. *1-6*
2. Madelung Deformity: Surgical Correction with Radial Dome Osteotomy. *7-13*
3. Cardiac Lymphoma Revealed by a Pulmonary Embolism Case Report and Literature Review. *15-18*
4. Papillary Muscle Hypertrophy as an Variant of HOCM- A Case Report. *19-21*
5. Pulmonary Vein Thrombosis Complicating Lung Cancer, Case Report and Literature Review. *23-28*

v. Fellows
vi. Auxiliary Memberships
vii. Preferred Author Guidelines
viii. Index

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Severe Iatrogenic Lutembacher Syndrome in a Young Male: Case Report and Literature Review

By Zeine El Abasse, Rime Benmalek, Soukaina Zahri, Salim Arous, Mohamed El Ghali Benouna & Rachida Habbal

IBN Orchd University Hospital Centre

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Here we discuss the case of a 46 years old male with MS who had undergone successful PMC seven years ago, who complained from gradually progressive exertional dyspnea, fatigue and palpitations. On detailed examination and investigation, he was found to be having Lutembacher's syndrome confirmed by echocardiography. After concertation with the heart team, the patient was managed by valvular surgery.

Keeping in mind this syndrome rare occurrence, we are presenting an overview of this syndrome, through a literature review, including its various aspects and the challenges faced by the patients and the physicians in the context of developped countries.

Keywords: atrial septal defect, mitral stenosis, lutembacher syndrome, iatrogenic, surgical treatment.

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Severe Iatrogenic Lutembacher Syndrome in a Young Male: Case Report and Literature Review

Zeine El Abasse [α], Rime Benmalek [σ], Soukaina Zahri [ρ], Salim Arous [ω], Mohamed El Ghali Benouna [¥] & Rachida Habbal [§]

Abstract- Iatrogenic Lutembacher's syndrome is a rare combination of acquired Atrial Septal Defect (ASD) after percutaneous mitral commissurotomy (PMC), and Mitral stenosis (MS) (usually of rheumatic nature).

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Keywords: atrial septal defect, mitral stenosis, lutembacher syndrome, iatrogenic, surgical treatment.

I. Introduction

Lutembacher Syndrome (LS) is a rare cardiac clinical entity characterized by the unusual combination of atrial septal defect (ASD) and Mitral stenosis (MS) (commonly of rheumatic nature) [1].

In the first description of LS by the frensh physician Rene Lutembacher in 1916, after whom this syndrome was eventually named [2], both lesions were thought to be congenital in origin, but since then, the definition has been broadened.

In a typical LS case, the ASD is usually more than 15 mm in size, which can cause progressive pulmonary hypertension (PH) [3], however, in the current era of percutaneous balloon mitral valvuloplasty (BMV) for acquired MS, ASD can be secondary to trans-septal puncture, which defines a new entity called iatrogenic LS [4].Thus, the current consensus defines LS as any combination of ASD (congenital or iatrogenic) and MS (congenital or acquired) [5].

Clinically the syndrome may resemble either isolated ASD or MS, depending upon the dominant lesion [6], but can also present in unusual forms.

LS is generally associated with long-term unfavorable natural course [7] depending on the evolution of PH and the occurance of heart failure (HF).

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Surgical and percutaneous trans-catheter therapies have proven to be beneficial in patients with LS [8].

We here report the case of a young male who was diagnosed with Iatrogenic LS based on relevant clinical findings and investigations, stressing the major role of echocardiography in the contemporary diagnostic and therapeutic modalities for LS, which can be challenging in the context of developped countries.

II. Case Presentation

We report the case of a 46-year-old male with history of rheumatic mitral stenosis (MS) for which he successfully underwent percutaneous transvenous mitral commissurotomy (PTMC) 7 years ago, who presented to the emergency department (ED) with gradually progressive exertional dyspnea, fatigue and palpitations.

Physical examination found a blood pressure of 99/64 mmHg, an irregular pulse rate of 61/min in addition to a tapping apex impulse, jugular vein distention, bilateral ankle oedema and abdominal distention suggesting ascites.

On cardiac auscultation, the first heart sound was loud, the second heart sound was widely split with a prominent pulmonary component. He also had a grade 4/6 diastolic rumble increasing with expiration in the apex, associated with a grade 2/6 holosystolic murmur and a grade 3/6 pansystolic murmur in the tricuspid area. Respiratory examination revealed bilaterally equal normal breath sounds with end-inspiratory fine crackles at the base of both lung. Electrocardiogram (ECG) showed right-axis deviation and atrial fibrillation (AF) (Figure 1). Chest radiograph showed cardiomegaly with a cardiothoracic ratio of 0.61, pulmonary plethora with biatrial and right ventricular enlargement.

Transthoracic echocardiography (TTE) revealed calcified and thickened mitral valve leaflets with bicommissural calcification and a "hockey stick" appearance of anterior mitral leaflet and immobility of the posterior leaflet with severe mitral stenosis (MS)(Mitral Valve Area (MVA) was 0.5 cm^2 by planimetry and mean pressure gradient (MPG) was 18 mmHg) associated with amoderatemitral regurgitation (MR) (Figure 2). The Aortic cusps were also thick and calcified with a moderate aortic stenosis (AS) with a MPG of 18

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mmHg and an aortic valve area of 1,1 cm2, associated to a mild aortic regurgitation (AR). Left ventricular (LV) function was normal, with a Left ventricular ejection fraction (LVEF) of 57% and a global longitudinal strain (GLS) of -19.1%. LV dimension during systole (LVDs = 26cm/m^2) and diastole (LVDd = 31cm/m^2) were normal, Interventricular septal thickness (IVST) and Posterior wall thickness (PWT) were also normal (7 and 8 mm respectively). The right ventricule (RV) was dilated (RV basal diameter of 45 mm) with a preserved function and a severe functional tricuspid regurgitation (TR) with an estimated Pulmonary artery pressure of 75 mmHg and a dilated inferior vena cava. In addition to these findings, we assessed bi-atrial enlargement with a left atrial spontaneous echo contrast and a non-restrictive Atrial Septal Defect (ASD) of the ostiumsecundum variety measuring 11mm with bidirectional shunt (Figure 3). The ASD was not found in the last TTE after his percutaneous intervention, thus, the diagnosis of iatrogenic Lutembacher syndrome (LS) was suspected. The Transesophageal Echocardiography (TEE) confirmed the diagnosis by showing left atrial spontaneous echo contrast and a non-restrictive ASD with bidirectional shunting in addition to the severe MS(Figure4).

We initiated medical therapy in our patient with high dose furosemide (250mg/Day), Acenocoumarol 4mg and Digoxin 0.25mg/Day. The case was then discussed by the heart-team who decided to perform a double valve replacement and a tricuspid annuloplasty given the associated aortic disease and the severe TR, in addition to a surgical closure of his ASD using a pericardial patch.

In time of submission, the patient successfully underwent surgery with good clinical evolution.

III. Discussion

The association of ASD with various abnormalities of the mitral valve (MV) such as MV systolic prolapse with MR are frequently reported, however, MS coexisting with ASD remains rare and represents a distinctive syndrome going by the name of its inventor Lutembacher [2]. At first, the ASD and MS in LS were both thought to be congenital, but since then, the definition has changed several times [5]. The ASD may be congenital (ostiumsecundum or sinus venosus type) or iatrogenic, secondary to cardiac interventional procedures like mitral valvuloplasty [9], as it was the case of our patient. The MS is often acquired in this syndrome as a consequence of rheumatic heart disease (RHD), especially in developped countries, but may also be congenital in rare cases (accounting for only 0.6% of congenital heart disease) [5,7].

The exact prevalence of LS is not well known [10], since it is more prevalent in developped countries with RHD, however, it is a rare syndrome occurring in 4-7% of cases of ASD and 0.6-1.2% of cases of MS [6,7]. LS can present at any age but is usually more common in young adults in the third decade with a predilection for females [7] unlike our patient who was a male which was described only once in the available literature [11].

LS is particular due to its unique haemodynamic consequences that result from the interplay between the ASD and MS. The clinical scenario of this syndrome depends upon the MS severity, the ASD size, the RV compliance and the pulmonary vascular resistance [7].

The first clinical scenario is when the MS is severe and ASD is restrictive, the shunt across the defect will be less, and hence, the patient will follow the course of isolated MS. In the same way, in the second scenario, if the ASD is non-restrictive and MS is not severe, the symptoms are those of ASD alone.

The third scenario is our patient's case with both severe MS and non-restrictive ASD. Due to the RV better compliance, the blood shunts through the ASD instead of backing up into the pulmonary veins, allowing the Left atrium (LA) to decompress in the right atrium (RA), thus avoiding the rise of left a trial pressure in proportion to the MS severity. Therefore, LS is well tolerated by patients for a long time and pulmonary congestion usually doesn't occur until late in the disease. Thus, symptoms of MS such as exertional dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea are less severe and delayed due to LA decompression [12].

However, this happens at the cost of an increased pulmonary vascular resistance and left to right shunt across the ASD with progressive dilatation of both the RA and the RV leading ultimately to RV failure and decreased systemic cardiac output. Thus, LS patients usually complain from fatigability on ordinary physical exertion and palpitations [13].

Our patient had signs of RV failure and severe tricuspid regurgitation, which is indicative of right ventricular dysfunction. He also complained from exertional dyspnea, and had end-inspiratory lung crackles, with signs of pulmonary vascular congestion in his chest x-ray, further suggesting that the RV compliance has diminished considerably enough to reduce the amount of shunting via the ASD. The natural course of our patient's disease without treatment would be an irreversible pulmonary vascular disease and the development of an Eisenmenger syndrome, which is usually very uncommon and delayed due to the MS. Another instance of development of a right-to-left shunt is a rare entity called Reverse Lutembacher syndrome, in which a severe tricuspid stenosis is associated to the classic LS thus precipitating central cyanosis, digital clubbing and hepatomegaly [14].

Among other complications of LS, the risk of infective endocarditis (IE) is increased by the presence of MS unlike in isolated ASD. However, the Mitral valve is

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less calcified in LS, because of the LA decompression, resulting in less turbulent flow across the mitral valve [10]. Furthermore, LA enlargement in LS predisposes the patients to develop AF, which explains the palpitations in our patient.

Preoperative diagnosis of LS is sometimes difficult, Steinbrunn et al [6] have emphasized the dangers of incomplete diagnosis before surgery. They reported 3 patients with LS who underwent surgery for closure of "isolated" ASD, resulting in death by severe pulmonary edema in one case and reopening of the shunt in the others during the early postoperative period. The diagnosis of LS is confirmed by Two-dimensional echocardiography [15]. The severity of MS and the size and type of ASD are accurately estimated, transmitral gradient is less despite severe MS, planimetry is the more reliable method to assess the MVA in LS as compared to Doppler half-time which tends to overestimate the calculations [16]. TEE outlines the site and size of ASD with its flow pattern and is usually a superior imaging diagnostic modality than TTE, which is why we performed both in our patient. Cardiac catheterization is rarely required for the diagnosis of LS, except for the assessement of pulmonary artery hypertension reversibility in Eisenmenger Syndrome, measurement of the MVA if the echocardiography lacks precision, and the evaluation of the coronary anatomy in high-risk patients.

As for the treatment, traditionally LS has been treated by open heart surgery by Open mitral valvotomy (OMV) or mitral valve replacement (MVR) with surgical repair of the ASD). But recently, with the advancement of percutaneous interventional techniques, percutaneous trans-catheter therapy in the form of balloon mitral valvuloplasty for MS and Amplatzer atrial septal occlude for ASD, has gained preference over surgery due to its faster recovery time and decreased length of hospital stay [17,18].

However, contraindications of percutaneous intervention include presence of left atrial thrombi, inadequate rim tissue surrounding the atrial septal defect and anomalous pulmonary drainage [4]. Another contraindication and the reason why our patient could not undergo such procedures is the presence of bicommissural calcification. Moreover, our patient also had a previous PTMC in addition to an associated aortic disease and a severe TR, which made the heart team decide to perform a double valve replacement and a tricuspid annuloplasty.

A symptomatic treatment with diuretics to relieve the symptoms of right-sided HF and/or pulmonary venous congestion in addition to Beta-blockers and calcium channel blockers for rate control in AF, as well as IE prophylaxis, is strongly recommended.

IV. Conclusion

Persistent iatrogenic ASDs have become an increasingly common finding after invasive procedures requiring trans-septal puncture. Iatrogenic LS is a rare yet challenging disease that needs to be diagnosed correctly via transthoracic and transesophageal echocardiograms in order to provide early and adequate medical and surgical therapies, in order to prevent the onset of PH and HF, thus improving survival rates.

Appropriate surgical procedures, be it transcatheter procedures or open heart surgery should be carefully discussed by the heart team, after an overall assessment of disease progression and cardiac anatomy.

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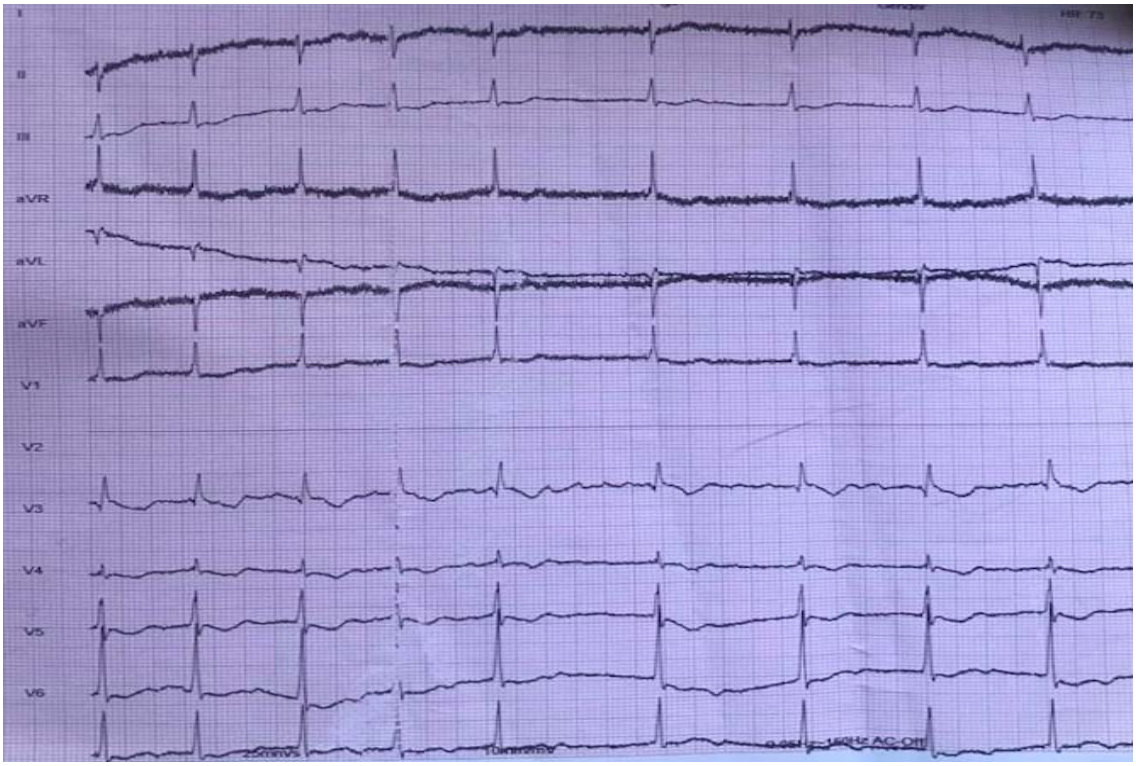


Figure 1: Electrocardiogram (ECG) showing Atrial fibrillation and right-axis deviation

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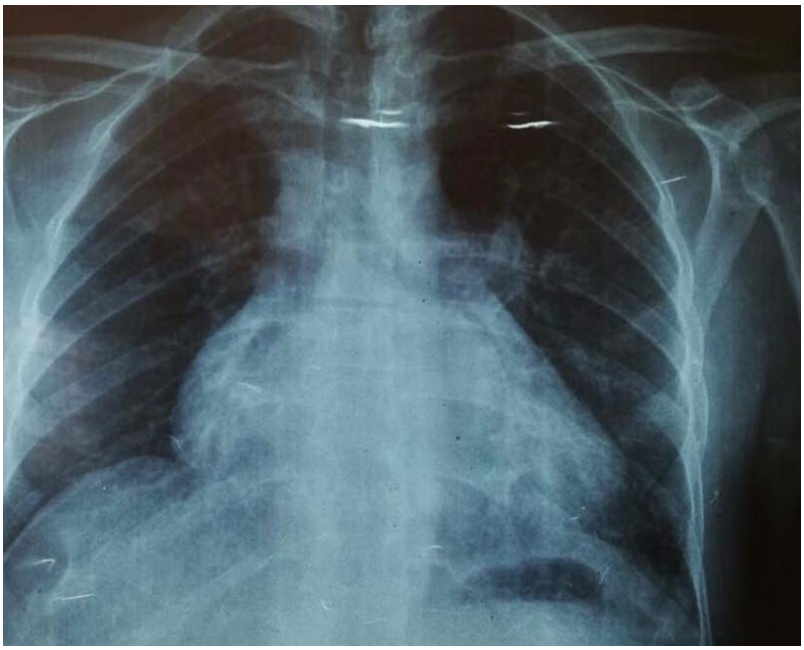


Figure 2: Chest radiograph showed cardiomegaly with a cardiothoracic ratio of 0.61, pulmonary plethora with biatrial and right ventricular enlargement.

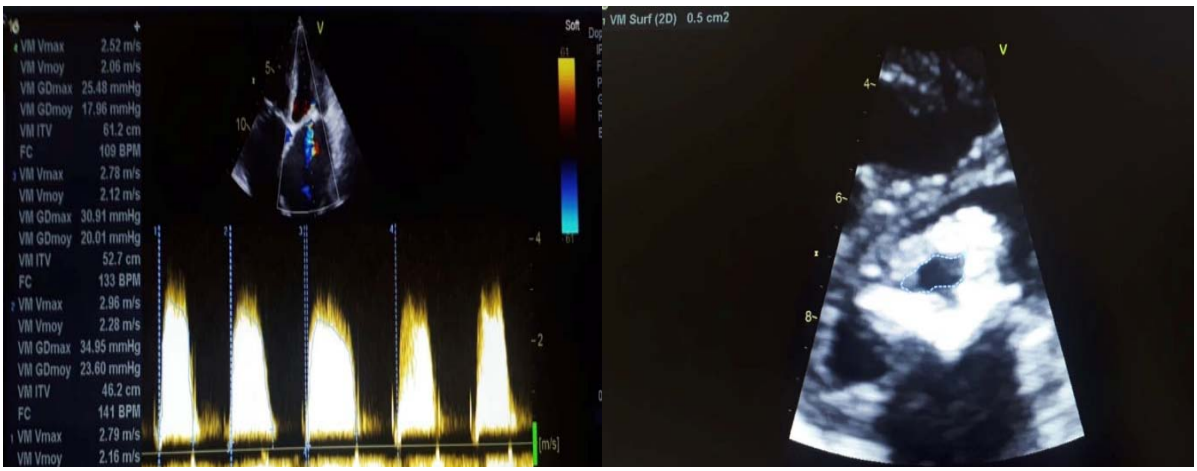


Figure 3: a- TTE Short-axis view showing a mitral valve area of 0.5 cm2 measured by planimetry, b- Apical 4 chamber view revealing a mean pressure gradient (MPG) of 18 mmHg associated with a moderate mitral regurgitation.

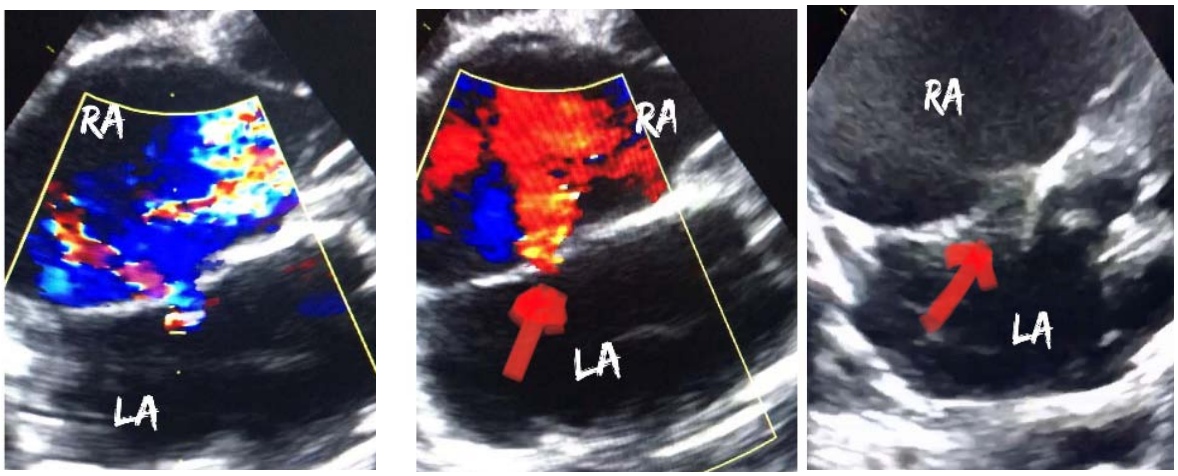


Figure 4: TTE sub-costal view showing left atrial spontaneous echo contrast and a non-restrictive ostiumsecondum ASD measuring 11mm with bidirectional shunt.

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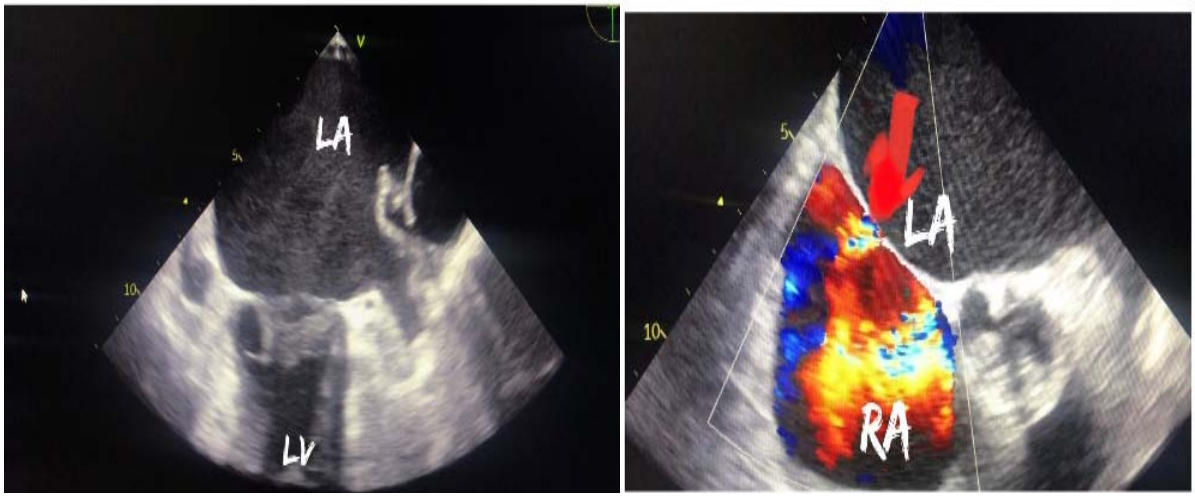


Figure 5: TEE showing a non-restrictive ASD in addition to a severe MS and a spontaneous contrast in the LA.

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Madelung Deformity: Surgical Correction with Radial Dome Osteotomy

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Between 2017 and 2019, in our clinic, 4patients were treated usingthis technique. Post-operative pain was evaluated using the NRS. Functional outcomes were assessed through evaluation of ROM, grip strength via Jamar dynamometer and using DASH questionnaire. The aesthetic defects were estimated using a section of the Michigan Hand Outcome Questionnaire. Correction of deformities was evaluated on post-operative RX using McCarrol Criteria. Were also analyzed the accuracy and tolerance of the plates used.

Keywords: madelung, deformity, radial dome osteotomy, piezosurgery, wrist 3D.

GJMR-I Classification: NLMC Code: WE 312

MADELUNGDEFORMITYSURGICALCORRECTIONWITHRADIALDOMEOSTEOTOMY

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Madelung Deformity: Surgical Correction with Radial Dome Osteotomy

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The data analysis suggested that, in all the cases of this study, the technique described led to pain relief, better wrist function and better aesthetics. The Radiographic outcome showed an improvement in all pathologic criteria.

This study showed promising results for the application of this specific surgical technique.

Keywords: madelung, deformity, radial dome osteotomy, piezosurgery, wrist 3D.

I. Introduction

Madelung deformity is a rare wrist malformation consisting of excessive radial and palmar angulation of the distal radius caused by a growth disorder of the palmar and ulnar part of the distal radius physis. This leads to shortening and angulation (recurvatio) of the epiphysis, causing a pathognomonic inclination of articular surface palmarly and ulnarly.

Shortening of the radius may cause the development of progressive incongruency at the DRUJ and a positive ulnar variance. This carries to a dorsal subluxation of the distal ulna, causing the altered aspect of the wrist with the prominent ulna.

Step-by-step alterations of the distal radius and the DRUJ are important contributor factors of the so-called carpus pyramidalization, a progressive increase of carpus convexity, and of the volar carpus subluxation.

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Other anomalies described are:

- Vickers ligament or radio-lunate ligament. It is an accessory volar ligament hypertrophic connecting distal radius metaphysis, TFCC, and volar surface of lunate. It is responsible for a compressive injury at the ulnarside of the distal radius physis and a proximal traction of the lunate, determining its collapse between radius and ulna[1-2].
- Volar radio-triquetral ligament. It may be present dorsally to Vickers ligament. It is characterized by greater dimension and distal insertion to triquetrum[3-5].
- Anomal insertion of pronator quadratus muscle [6]and presence of accessory muscles.

Madelung deformity is a rare condition, the incidence is unknown, and there is no described racial predominance.

Usually, patients come to medical observation between 8 and 14 years old, although there are in literature cases of Madelung deformity at birth or childhood. It is more common in females, the M:F rate of the disorder is 1:4. Madelung deformity can be bilateral about in a third of patients. It may be associated with Léri-Weill dischondrosteosys[7].

There are several types of Madelung deformity[8]:

- Dysplasic type is the more common. It is associated to Léri-Weill dischondrosteosys;
- Idiopathic;
- Genetic or chromosomic. It is associated to genetic pathologies such as Turner syndrome, Hurler syndrome (mucopolysaccharidosis type 1H), mucopolysaccharidosis type IV and VI, achondroplasia, hereditary multiple exostoses syndrome, hereditary osteo-onychodysplasia, pseudohypoparathyroidism type 1a-1b and Ollier disease[7].
- Post-traumatic. It is a rare form described in gymnastics athletes, and in persons subjected to repetitive microtraumatisms to the wrist. It may be secondary to infections and osteomyelitis of the distal radial physis.

These types are then subclassified according to the presence or absence of Vickers ligament. The idiopathic and dysplasic may occur in association or not with V. ligament. Therefore, we can classify Madelung deformity as true Madelung deformity in the presence of

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Vickers ligament and Madelung-like deformity if Vickers ligament is absent.

Clinically, patients complain of movement-related pain, altered aspect of the wrist, functional limitations especially in prono-supination.

In literature several therapeutic options are reported[9-11], but because of the limited number of cases, due to low incidence, it is difficult to determine the efficacy of proposed treatment strategies.

- Wait & see for asymptomatic or paucisymptomatic cases;
- Surgery:
  - Surgical correction of the radius: physiolysis with or without release of Vickers ligament; opening or closed wedge osteotomies; reverse wedge osteotomy;
  - Surgical correction of the ulna: epiphysiodesis; shortening osteotomy, Darrach's technique; Sauvè-Kapandji technique;
  - Combined surgery on radius and ulna;
  - Release of Vickers ligament

Harley et al.[12-13] in 2006 and then in 2013 described a surgical correction with dome osteotomy of the distal radius. The technique described provides a dome osteotomy with distal concavity for biplanar correction of deformity, release of Vickers ligament and arthrodesis o shortening osteotomy of the ulna.

In the present study, we report our experience in surgical correction of Madelung deformity with the release of Vickers ligament, distal convexity dome osteotomy of the distal radius, and reverse wedge.

II. Materials and Methods

Between 2017 and 2019, four patients (2 males and 2 females) affected by Madelung deformity were treated in our clinic.

Three adolescents aged between 12-14 yearsold and a 30-year-old adult. Three patients had a bilateral Madelung deformity with a mild deformity in the untreated limb (tab.1).

For preoperative planning (fig. 1) we acquired plain radiographs (AP, LL, and oblique projections), MRI to evaluate surrounding soft tissue (in particular presence/absence of Vickers ligament), and CT scans for better analysis of the deformity.

CT scan data were sent to a workstation in standard DICOM format (PACS, Carestream) to create 3D models of the affected wrist. For all patients polyamide templates of the deformed wrists (scale 1:1) were manufactured using a 3D-printer.

Table 1: Sample

Pz	Age	Bilateral	Treated limb	Follow-up (months)	Form	Vickers ligament
1	3	yes	right	15	Idiopatic	Yes
2	12	yes	right	26	Genetic: Léri-Weill	Yes
3	13	yes	left	16	Idiopatic	No
4	13	no	right	28	Idiopatic	No

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Picture 1: Pre-op planning. A: X-ray; B: MRI with Vickers Ligament (*). C: CT selection; D: 3D printing model for pre-op planning

Use of 3D-printed polyamide template in preoperative planning allowed to evaluate rotational center of deformity, plan the osteotomy, evaluate needed degrees for triplanar correction, choose implants and simulate preoperatively surgical procedure.

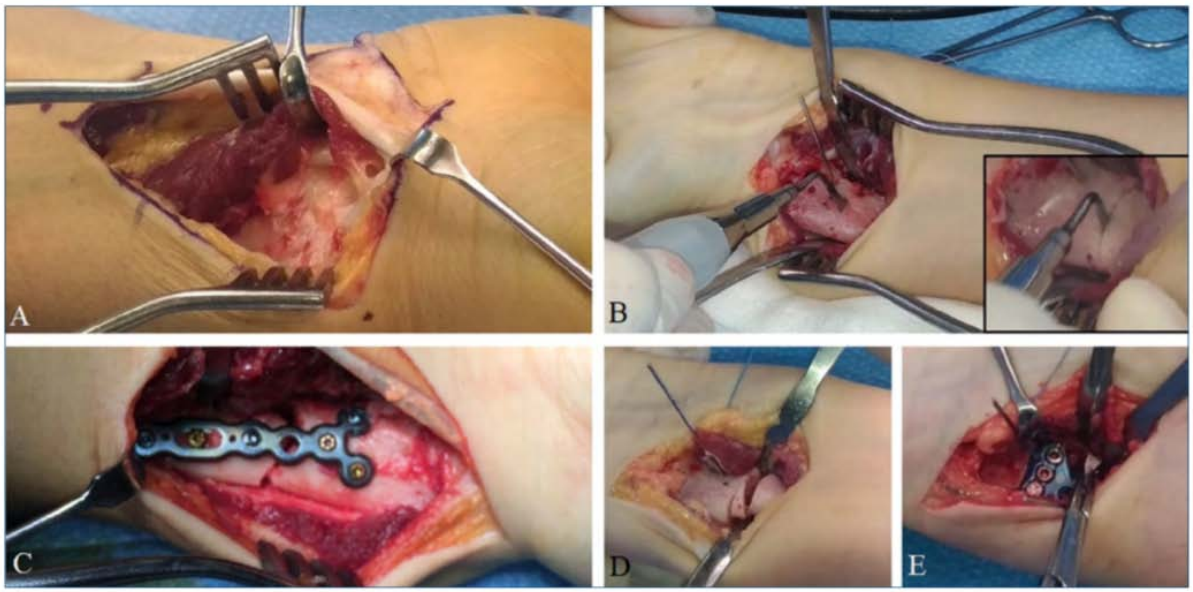
Preoperative planning using 3D virtual planning and 3D-printed templates improves accuracy of surgical correction with dome osteotomy and reduces surgical duration.

It was used a Henry approach, modified according to Orbay, for all the patients in this serie. Release of Vickers ligament in cases where it was present. After identification of rotational center and level of osteotomy, such as preoperative planning, using a piezoelectric saw (piezosurgery), it was possible to improve the accuracy of the osteotomy and the biplanar correction of the deformity with no risk of thermal necrosis.

In 2 cases it was also needed a bone wedge excision of the radial column for better correction of the deformity on the frontal plane. Then the excised bone fragment was used as auto-graft to fill the gap of dome osteotomy (reverse wedge), allowing faster consolidation and improving stability.

Low-profile mouldable plates were used for fixation because of better adaptability rather than anatomical pre-contoured. At least three screws proximal and three distal to osteotomy were used to fix the plate, avoiding physis and DRUJ.

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Picture 2: Sequel of the surgical steps (different patients) A: Release of the V.L. B: Intra-op osteotomy with piezosurgery. C: Correction and synthesis with a volar plate. D: Cut and remove of the bony wedge from the lateral part of the radius. E: Reverse and positioning of the bony wedge in the ulnar side of the osteotomy site.

In one case it was noted the absence of the pronator quadratus associated with the presence of palmaris profundus muscle. The latter anomaly was already described in literature[14].

After surgery, immobilization of the wrist for 3 weeks in a thermoplastic splint was applied to all patients. After this period of immobilization, the wrist and forearm rehabilitation protocol were started for about 6 weeks under the supervision of a hand therapist.

Between 8 and 12 months postoperative plates were removed in order to low the incidence of complication such as tendinitis, infections, and loosening.

The mean follow-up duration was 21 months (range 15-28 months). During this period patients were evaluated like proposed by Peymani et al.[15] in his review. Radiographically McCarroll criteria[16] were used to judge correction, we evaluated ROM clinically, grip strength via Jamar dynamometer. DASH questionnaires were administered to evaluate functional outcomes, NRS to quantify subjective pain. Finally, the aesthetic defects were estimated using a section of the Michigan Hand Outcome Questionnaire.

These data were related to collected data before surgery.

III. Results

Post-operative radiographs, according to McCarroll criteria, showed a reduction of ulnar tilt, lunate fossa angle, and palmar tilt; whereas no significant difference of palmar carpal displacement was found (Table2).

Table 2: Radiographic McCarrollcriteria

Pz	UT	LS	LFA	PCD	PT
1	*48.7°	*21 mm	*50,8°	*27 mm	*27,8°
	**38°	**9 mm	**33,1°	**27 mm	**18,1°
2	*60,3°	*3 mm	*56,6°	*20,5 mm	*21,3°
	**41,7°	**4,95 mm	**45°	**24 mm	**16,6°
3	*45°	*6,82 mm	*48,3°	*22 mm	*52,6°
	**31°	**6,63 mm	**33,9°	**22 mm	**41,1°
4	*43,6°	*12 mm	*51,3°	*31 mm	*21,5°
	**41°	**6 mm	**47à°	**27mm	**17,4°

UT = ulnar tilt; LS = lunate subsidence; LFA = lunate fossa angle; PCD = palmar Carpal displacement; PT = palmar tilt.
*preoperative; ** postoperative.

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Overall pain reduction after surgery was 4,75/10. Post-operative aROM and pROM were improved near to normal values. There was no significant difference in postoperative grip strength values comparing the mean value of 3 consecutive tests (Table3).

Table 3: Grip stength, Pain, ROM

Pz	Grip strength	Pain (NRS)	Range of motion					
			F	E	UD	RD	S	P
1	*38 kg	*7/10	*80°	*40°	*30°	*15°	*70°	*70°
	**37 kg	**1/10	**90°	**70°	**40°	**>20°	**>90°	**70°
2	*27 kg	*7/10	*90°	*65°	*40°	*5°	*55°	*80°
	**27 kg	**5/10	**>90°	**80°	**40°	**>20°	**>90°	**90°
3	*21 kg	*5/10	*90°	*80°	*40°	*10°	*70°	*85°
	**19 kg	**0/10	**>90°	**80°	**40°	**>20°	**90°	**90°
4	*34 kg	*6/10	*90°	*70°	*>40°	*10°	*65°	*90°
	**37 kg	**0/10	**>90	**75°	**>40°	**>20°	**>90°	**>90°

F=FLEXION; E=EXTENSION; UD=ULNAR DEVIATION ; RD=RADIAL DEVIATION ; S=SUPINATION ; P=PRONATION
*preoperative; ** postoperative.

There were significant improvements in functional outcomes after surgical correction as suggested by DASH scores, mean score 13/100. It has to be considered that only the adult patient answered the work-related section of the DASH questionnaire. The MHOQ scores were improved in all patients on average 30/100 (Table 4).

Table 4: DASH and Michigan Hand Outcome Questionnaire (MHOQ)

Pz	DASH	MHOQ
1	*19,4/100	*50/100
	**11,1/100	**75/100
2	*51,62/100	*62,5/100
	**45/100	**50/100
3	*22,4/100	*68,75/100
	**7,76/100	**93,75/100
4	*34,17/100	*18,75/100
	**11,16/100	**43,75/100

IV. Discussion

The purpose of surgical correction in patients affected by Madelung deformity is to relieve pain and improve wrist functions supporting the lunate.

The pain was relieved in all patients, 2 of them were totally pain-free. Analysis of postoperative ROM, grip strength via Jamar dynamometer, and the DASH scores showed good results. Improved MHOQ scores suggested good aesthetic correction.

Corrective dome osteotomy was evaluated on post-operative radiographs, analyzing the degree of correction according to McCarroll criteria. There was a reduction of pathological angles obtaining in some cases a value close to physiological. However, the aim of surgery is not to restore radius but is to improve wrist functions, avoiding lunate collapse. Therefore, it is important that surgery leads to correction of ulnar tilt, lunate fossa angle, and palmar tilt. In our study, we obtained correction of all these parameters.

There was no difference in palmar carpal displacement values between pre and post-surgical correction. PCD measures the palmar-directed displacement of the carpus (represented by the lunate and capitate) relative to the longitudinal axis of the ulna in the lateral view. There was no correction of PCD values because it was not made any surgical correction of the ulna in contrast with the surgical procedure described by Harley et al. The choice not to modify the deviation of the ulnar longitudinal axis surgically was made so as not to interfere with prono-supination movements since patients showed good ROM pre-operatively. The range of motion may worsen if we modify the delicate equilibrium made by the gradual dysmorphic growth of articular surfaces aiming tophysiological anatomy that is not present in DRUJ.

Eventual surgical correction of the ulna may be considered at the end of the skeletal growth (predominantly for aesthetic reasons).

Some limits to the McCarrol criteria, since x-ray projections are operator dependent, is to always obtain perfect AP and LL x-ray projections. This limit may be overcome using a preoperative CT scan that are improved with 3D reconstructions and/or MRI.

Compared to other techniques reported in the literature for the surgical correction of Madelung

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deformities, our technique appeared to be less invasive with shorter recovery time and improved patient's quality of life.

McCarroll and James[17] described a combined technique that includes osteotomies of radius and ulna (very distal radius osteotomy) through a dorsal approach. The same authors highlight an important limitation of the surgical technique in pediatric patients because fixation has to be made very close to the physeal growth plate.

Our technique allows to extend the indications to very young patients because the dome osteotomy and the fixation are made more distant to the physeal growth plate.

Another limit showed by McCarrol and James is the development of a post-surgical DRUJ instability, often asymptomatic. This is another contributing factor for our choice not to correct surgically the ulna.

Dome osteotomy showed some common points with cylindrical corrective osteotomy for Madelung deformity proposed by Imai et al.[18]. Both techniques agree to the fundamental use of CT scan and the case report used a dedicated software for the preoperatory planning, for the line of osteotomy, and to create custom-made cutting guides.

Custom-made cutting guide improve surgical accuracy to the preoperative planning, but they limit ulnar column lengthening, using bone wedge excised from radial column osteotomy (reverse wedge).

The use of a pre-op 3D printed model improves surgeons' evaluation of patient-specific anatomy and pathology by way of tactile and visual experience [19]. Furthermore, this technology supports the surgeon by selecting the most adequate device for osteosynthesis and helps patients and parents to understand the surgical procedure they will undergo.[20]

V. Conclusions

The experience with our patients (even if not statistically significant) showed encouraging results in the use of radial dome osteotomy for the correction of Madelung's deformity.

Our experience suggests that the use of 3D printed bone models in preoperative planning improves accuracy on surgical procedure and on the choice of implants.

In the near future, thanks to a larger sample, our effort will be to standardize this technique as much as possible, providing precise and adaptable indications to each individual case in order to improve the preoperative planning aiming to achieve even better surgical results.

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Cardiac Lymphoma Revealed by a Pulmonary Embolism Case Report and Literature Review

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Abstract- *Background:* Cardiac lymphoma is one of the rarest tumors involving the heart that have polymorphous and non-specific clinical symptomatology (1); it's preferential location is rather the right-sided cardiac chambers. Echocardiography and MRI are the diagnosis methods of choice for this location. The tumor is rapidly fatal unless diagnosed and treated in time.

Case Presentation: A 65 year old woman was referred for worsening exertional dyspnea , primary examination revealed a stable patient, Unilateral edema was noted with decreased sloshing of the left calf. The electrocardiogram showed a sinus tachycardia and the transthoracic echo revealed the presence of multiple intra cardiac masses (RA, RV, LV), with pericardial thickening and minimal pericardial effusion. Cardiac MRI showed a pericardial thickening extending to the basal vessels associated with several masses at the expense of the lateral wall of the RA, RV, LV. An attempted echo-guided biopsy of the mediastinal lymphadenopathy was performed but the patient did not support the procedure.

GJMR-I Classification: *NLMC Code: WG 210*



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Abstract- Background: Cardiac lymphoma is one of the rarest tumors involving the heart that have polymorphous and non-specific clinical symptomatology (1); it's preferential location is rather the right-sided cardiac chambers. Echocardiography and MRI are the diagnosis methods of choice for this location. The tumor is rapidly fatal unless diagnosed and treated in time.

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Discussion: Primary cardiac tumors are extremely rare. Examining the 22 series autopsy data, Lam KY(4)found 0,2% as a prevalence of primary cardiac tumors. The median age of the patients is 63 years. The symptoms of PCL are non specific. Diagnosis is delayed due to varied clinical presentations and limitations in diagnostic tools. Echocardiography shows uneven hypo echoic masses, and CT provides excellent anatomic assessment of cardiac lymphoma involvement. Cardiac MRI is the preferred imaging modality for cardiac masses because of its superior soft tissue characterization. The definitive diagnosis is based on the cytology of pericardial effusion or taking a trans-thoracic biopsy. Treatments of cardiac lymphomas can include chemotherapy, radiotherapy, surgery and even autologous stem cell transplantation, Subsequently our patient developed a sudden hemodynamic instability suspecting a serious pulmonary embolism despite the thrombolysis as well as the resuscitation measures, the patient died.

Conclusion: Although cardiac lymphoma is very rare, it should be considered in patients with an intracardiac mass. Its early diagnosis is essential because of the aggressive nature of these tumors.

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I. Introduction

Cardiac lymphoma is one of the rarest tumors involving the heart. Cardiac lymphomas are infiltrative, intramural, and epicardial lesions that may be singular or multiple, and the pericardium is involved in about a third of cases(1).

It is a subset of non-Hodgkin's lymphoma, characterized by poor outcomes and very often unrecognized entity due to its polymorphous and non-specific clinical symptomatology.

The preferential location of the cardiac lymphoma is rather the right-sided cardiac chambers, especially the right atrium, and it is often multifocal(2). The superior vena cava (SVC) is involved in up to 25% of cases(3), extracardiac lymphoma sites should be looked for during diagnostic evaluation, namely the mediastinum and bone marrow, in order to distinguish between primary and secondary cardiac lymphoma.

Echocardiography and MRI are the diagnosis methods of choice for this location. Histological confirmation is sometimes difficult to obtain in most living patients.

The tumor is rapidly fatal unless diagnosed and treated in time but the prognosis of this localization is often reserved in the short term and the response to chemotherapy is rarely complete and lasting.

II. Case Presentation

A 65 year old woman without cardiovascular risk factors or specific pathological history, was referred in the cardiology department of CHU Ibn Rochd Casablanca, for worsening exertional dyspnea and orthopnea. The examination on admission revealed a stable patient, the blood pressure was 122/67 mm Hg, the oxygen saturation was 95%, the pulse rate was 100 beats per minute, the respiratory rate was 19 per minute and the body temperature was 36.5C.

The cardiovascular examination was unremarkable. The peripheral ganglionic areas were free. Unilateral edema was noted with decreased sloshing of the left calf.

Biochemical investigations were within normal range, however a thrombocytopenia in the completed blood count was noted at 145000.

The electrocardiogram showed a sinus tachycardia and the transthoracic echo revealed the

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presence of multiple intracardiac masses (RA, RV, LV), with pericardial thickening and minimal pericardial effusion (Fig 1).

Thoraco-abdominal-pelvic CT showed a massive pulmonary embolism and a very strong suspicion of lymphoma with lymph node and cardiac location(Fig2).

Cardiac MRI showed a pericardial thickening extending to the basal vessels associated with several masses at the expense of the lateral wall of the RA, RV, LV. The MI venous Doppler ultrasound shows extensive deep vein thrombosis to the iliac vein.

An attempted echo-guided biopsy of the mediastinal lymphadenopathy was performed but the patient did not support the procedure, subsequently our patient developed a sudden hemodynamic instability suspecting a serious pulmonary embolism despite the thrombolysis as well as the resuscitation measures, the patient died.

III. Discussion

Primary cardiac tumors are extremelyrare. Examining the 22 series autopsy data, Lam KY(4)found 0,2% as a prevalence of primary cardiac tumors. Primary cardiac lymphoma(PCL), on the other hand, accounts for less than 2% of all resected primary cardiac tumors and 0.5% of extranodal lymphomas atautopsy(2)

There are two definitions of primary cardiac lymphomasome authors consider the absence of lymphoma apart from pericardial sac, confirmed by a complete autopsy (5)while the others accept lymphoma as primaryto the heart if the tumor is in the pericardium(6)

Many authors reported that this disease is more common in the elderly age. The medianage of the patients is 63 years(3). The reported male to female ratio is 3/1(7), our patient was 64 years old.

The symptoms of PCL are nonspecific. It can manifest as a heart rhythm disturbance, episodic syncope, vena cava superior syndrome, respiratory distress (7)or even as a restrictive cardiomyopathy (7–9) However, the most common symptoms are dyspnea, constitutional complaints(fever, chills, sweats and weight loss), chest pain, heart failure and pericardial effusion (7). In our case, the patient presented with dyspnea and worsening orthopnea.

Diagnosis is therefore often a great challenge as patients present with a wide spectrum of clinical presentation, generally depending on the site of involvement of the heart. They may involve the right chambers of the heart, with RA being the most commonly affected. These patients can develop pulmonary embolism, A-V block and disturbances in hemodynamics, but there are cases were only the left heart was involved(10). It can concern all three layers of the pericardium, myocardium, and endocardium(11), If PCL involves the pericardium, effusion or constrictive pericarditis may develop.(11) PCL-related cardiac wall rupture has also been described(12). The cardiac MRI of our patient showed a pericardial thickening extending to the basal vessels associated with several masses at the expense of the lateral wall of the RA, RV, LV, associated to a massive pulmonary embolism.

Diagnosis is delayed due to varied clinical presentations and limitations in diagnostic tools. In this regard, several diagnostic techniques can be used: ECG can objectify Several electrocardiographic abnormalities, typically an atrial arrhythmia or atrioventricular block ranging from first to third degree.(3),(13) More unusual cases show left or right bundle branch block or ventricular arrhythmia(3). Sudden death due to cardiac arrhythmia may even be the initial presentation of cardiac lymphoma.(3)(14)

Erythrocyte sedimentation rates and / or C-reactive protein (CRP) may be elevated (15)Information regarding lactate dehydrogenase levels is controversial because some authors have detected increased serum levels, Leukocytosis and neutrophilia can be found in some cases of cardiac lymphoma (16) ; our patient had only a minimal thrombocytopenia at 145 000 .

Regarding imaging, Chest radiography remains limited for the detection of heart tumor, it can sometimes show non-specific signs such as pleural effusions or cardiomegaly. Transthoracicechocardiography remains a ring stone for evaluation of heart disease and in many cases it remains the first-line imaging method, it is a good noninvasive diagnostic tool that can detect pericardial effusion and the presence of the tumor(17).

Echocardiography shows uneven hypo echoic masses with poor mobility, stiffness, wide basement, irregular shape, and boundary

However, complementary assessment with cross-section a limaging is now essential for further characterization of the tumor and the extent of involvement, CT provides excellent anatomic assessment of cardiac lymphoma involvement, primarily due to high isotropic spatial and temporal resolution.

It benefits from fast acquisition time and is a primary alternative in imaging patients with known contraindications to MR, On CT, cardiac lymphoma classically presents as a variably or poorly enhancing mass typically arising in the right side of the heart with similar prevalence of right atrial and ventricular involvement(12).

Cardiac MR is the preferred imaging modality for cardiac masses because of its superiorsoft tissue characterization, high temporal resolution, multiplanar imaging capabilities, and unrestricted field of view(18). Recently, FDG-PET was used to detect the primary effusion lymphoma of the pericardium(19).

The definitive diagnosis is based on the cytology of pericardial effusion or taking a transthoracic biopsy.(7) Cardiac lymphomas are usually B-cell

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neoplasms, most frequently DLBCL, followed by follicular lymphoma and Burkitt lymphoma(20).

The management of cardiac lymphoma is varied. Treatments differ and include chemotherapy, radiotherapy, surgery and even autologous stem cell transplantation.(21,22)

Surgeryis usually palliative and there is no evidence that it improves survival because it is difficult to resect the tumor completely. Most of the authors suggest that PCL should be considered a systemic disease and this treatment should always include chemotherapy(3).

The prognosis for patients with either primary and secondary heart lymphoma is unfortunate, at recent analysis of PCL and treatment results (cohort of 128 patients treated with chemotherapy, surgery, radiotherapy or chemotherapy /combined radiation) demonstrated a median overall survival of about12 months(3)the main prognostic factors are Initial non-specific signs and symptoms, rapid progression of cardiac involvement, and late diagnosis. (23,24).

IV. Conclusion

Cardiac lymphoma manifests as singular or multiple intramural masses with infiltrative margins and a strong predilection for the right heart, Echocardiography is often the initial imaging modality, but CT and MR imaging provide superior soft tissue contrast and anatomic information. Although cardiac lymphoma is very rare, it should be considered in patients with an intracardiac mass. Its early diagnosis is essential because of the aggressive nature of these tumors.

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Figure 1: Subcostal section showing the mass lining the lateral wall of the RA and RV.

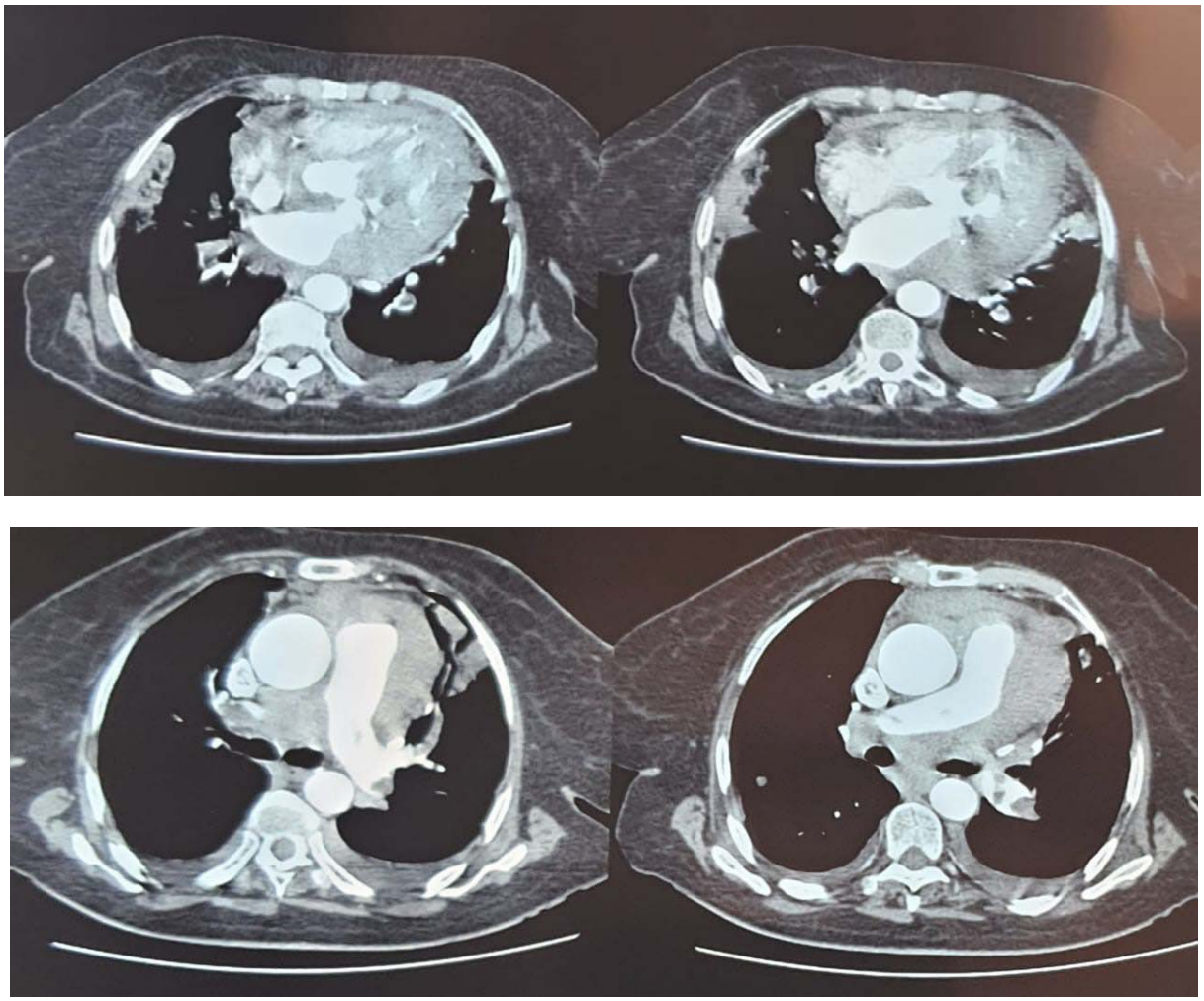


Figure 2: Thoracic CT: Massive pulmonary embolism with strong suspicion of lymphoma with ganglionic and cardiac localization.

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Papillary Muscle Hypertrophy as an Variant of HOCM- A Case Report

By Dr. Sunil Dighe, Dr. Kalyan Munde, Dr. Piyush Kalantri, Dr. Zahidullah Khan & Dr. Mahesh Bodke

Grant medical College and Sir JJ Group of Hospital

Abstract- According to the 2011, ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy (HCM), the morphologic diagnosis of HCM is based on the presence of a hypertrophied as well as non-dilated left ventricle in the absence of another cardiac or systemic disease capable of producing the magnitude of that hypertrophy (usually ≥15 mm in adults or the equivalent relative to body surface area in children). Although the papillary muscles (PMs) are an anatomic part of the left ventricular (LV) chamber, the significance and diverse morphology of these structures in HCM has not been characterized in literature. Papillary muscle (PM) hypertrophy is a rare echocardiographic finding, with very few cases reported in the literature. Therefore, solitary PM hypertrophy can have clinically important for the screening of HCM as a newly identified subtype of or an early form of HCM. We are reporting the same case in which papillary muscle hypertrophy was a culprit for HOCM.

GJMR-I Classification: NLMC Code: WG 460

PAPILLARYMUSCLEHYPERTROPHYASANVARIANTOFHOCMACASEREPORT

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Abstract- According to the 2011, ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy (HCM), the morphologic diagnosis of HCM is based on the presence of a hypertrophied as well as non-dilated left ventricle in the absence of another cardiac or systemic disease capable of producing the magnitude of that hypertrophy (usually ≥15 mm in adults or the equivalent relative to body surface area in children). Although the papillary muscles (PMs) are an anatomic part of the left ventricular (LV) chamber, the significance and diverse morphology of these structures in HCM has not been characterized in literature. Papillary muscle (PM) hypertrophy is a rare echocardiographic finding, with very few cases reported in the literature. Therefore, solitary PM hypertrophy can have clinically important for the screening of HCM as a newly identified subtype of or an early form of HCM. We are reporting the same case in which papillary muscle hypertrophy was a culprit for HOCM.

I. Introduction

Hypertrophic obstructive cardiomyopathy (HOCM) refers to those subjects with significant dynamic left ventricular outflow tract (LVOT) obstruction due to mechanical causes. Most of them presents with asymmetrical septal hypertrophy which result in Dynamic systolic anterior motion (SAM) of mitral leaflets[1]. There are also few patients of HCM who are genotypically positive but phenotypically negative in HOCM. The cardiac phenotype of HCM shows great diversity in the degree and pattern of hypertrophy (asymmetric, concentric, or apical), age of onset, and clinical course. Solitary papillary muscle (PM) hypertrophy, a form of HCM manifested as predominant PM hypertrophy sparing the rest of other LV segments, has recently gained much attention to its mechanical cause for left ventricular outflow tract (LVOT) pressure gradient formation[2,3]. The clinical diagnosis of hypertrophic cardiomyopathy (HCM) is conventionally made with cardiac imaging, at present, most frequently two-dimensional echocardiography.still, the use of cardiac magnetic resonance (CMR) imaging is increasing. Morphological papillary muscle anomalies without features of phenotypic LV hypertrophy which include isolated papillary muscle hypertrophy had gradually been recognized as variant in uncommon HCM[4,6]. The clinical features of LV mid-cavity obstruction caused by papillary muscle hypertrophy may vary from asymptomatic to dyspnea, angina, syncope, and even sudden cardiac death[4-6]. Solitary papillary muscle hypertrophy as an uncommon variant form of HCM with coexisted additional, accessory papillary muscle may develop abnormally high resting LV mid-wall pressure gradient without SAM or significant regional LV wall hypertrophy. CMR provides complete tomographic imaging of the heart with high spatial resolution images and is an excellent imaging method to assess the PMs. It is also a important tool for further investigation and assessment of the different types of cardiomyopathies, since there are also some typical findings in this exam that may suggest a particular pathology or etiology.

II. Case Report

A 33 yr old male presented to our hospital with chest discomfort-, Breathlessness at rest, palpitation, and syncope. The patient was known case Bicuspid Aortic Valve with Aortic Stenosis. He was a tobacco chewer and alcoholic addict. On admission, his heart rate was 60/min. BP 90/60 mm of hg. He has undergone Balloon Aortic Valvotomy 15 yrs back. Laboratory tests were performed, but the results were unremarkable which include complete blood count, liver and kidney function tests, serum levels of muscle and cardiac enzymes. A mid-systolic ejection murmur was heard along the left sternal border with chest X-ray showed no pulmonary congestion. ECG has LV strain Pattern. 2D Echo suggestive of severe Aortic Stenosis and Aortic Valve was showing Calcification within. We planned Coronary Angiography before sending a patient for Aortic Valve replacement.

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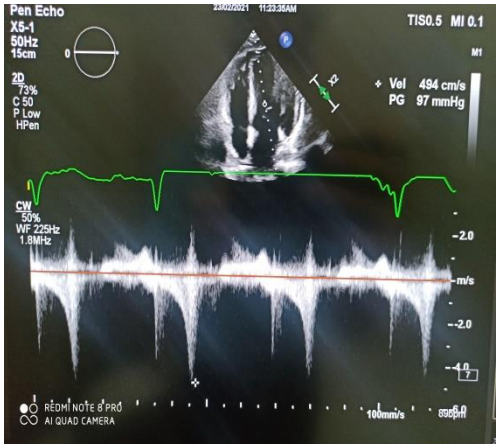


Figure 1

Figure 1: Mid Cavity Gradient in Apical 4 chamber View

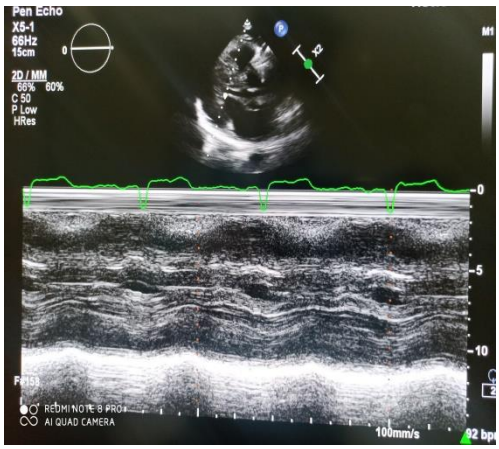


Figure 2

Figure 2: M mode of Parasternal long-axis view through Papillary muscle

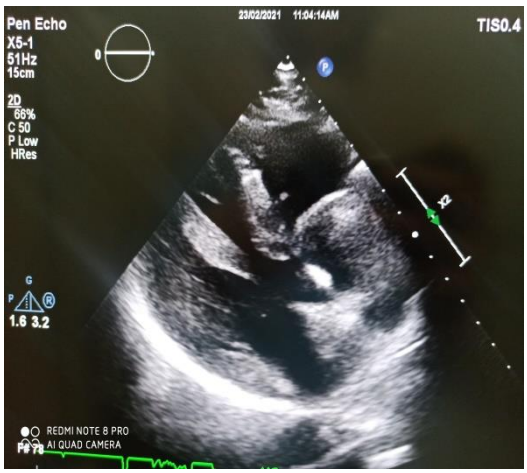


Figure 3

Figure 3: Parasternal Long Axis View showing Hypertrophy of Papillary Muscle

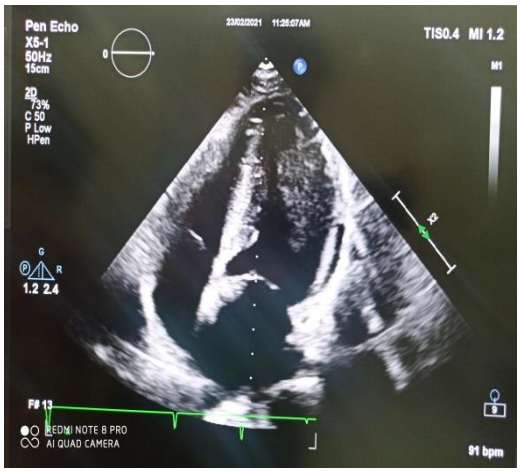


Figure 4

Figure 4: Apical 4 Chamber View showing hypertrophy of papillary muscle

2D Echo imaging of similar patient when focused on looking for aortic valve it was showing gradient at LVOT. Being no Much hypertrophy of the Interventricular septum and posterior wall, the gradient was further evaluated. it was found that Papillary muscle was thickened to a greater extent, .which was creating a pressure gradient of 97 mm of hg, as shown in figure 1. Figure 2 also demonstrates the M mode showing compromised LV cavity at end Diastole. Similarly, in figure 1 we can see the calcified aortic Valve causing restriction of leaflet motion. patient was a known case of bicuspid aortic valve and i/v/o of severe nature (Peak/Mean Trans aortic gradient 89/45 mm of hg) of aortic stenosis. Gradients across mid cavity increased on doing Valsalva maneuver by the patient, which suggested a dynamic obstruction at mid cavity and the papillary muscle hypertrophy was the etiology for it. The thickness of the papillary muscle was 14 mm in our patient, which was considered significant to cause an obstruction.

III. Discussion

The clinical significance of abnormalities and hypertrophy of the PMs is a matter of debate. This morphological finding requires further investigation, as there are few articles published, and little information on this entity. The published literature Suggests that morphological abnormalities in PMs, such as anomalous insertion, are found in the context of LV wall hypertrophy and that they are not uncommon in HCM, especially in apical hypertrophy. But they have also been reported as the only morphological abnormality in a subgroup of patients with HCM. It has also been suggested that isolated PM hypertrophy is a possible variant of HCM. These changes may evolve, along with progressive hypertrophy over time.

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The relevant abnormal findings in this patient are the hypertrophied PMs (with normal ventricular mass), hypertrophy has been associated, in some studies and case reports, with abnormal T-wave inversion and with a possible variant of HCM or an initial stage of this disease.

Clinically, HCM is usually recognized by maximum LV wall thickness ≥15 mm, with a thickness of 13–14 mm considered borderline, particularly in the presence of other compelling information (in presence of family history of HCM). Genetic testing for HCM is available as an important diagnostic tool. Still, it has limitations since about 50% of patients have an identifiable mutation in such patient, and a some proportion have variants in which the pathogenicity of the mutation is uncertain.

Despite this, the next step will be to perform a genetic test since this case may be an atypical presentation or initial stage of HCM. Stress echocardiography will also be important to determine the presence of ventricular gradients. As per the results of genetic tests and the patient's clinical course of disease, assessment and screening of patients first-degree relatives should be considered.

IV. Conclusion

Considering the available information on PM hypertrophy, the case presented may represent a gap in our knowledge of HCM. More investigation are needed in such situation, which may affect the definition of HCM and subsequently diagnosis, management and Prognosis of this HCM patients. With this case, particularly with its images, we states that HCM is a complicated disease that generally goes beyond the left ventricle walls. Such cases of HCM should not be missed, especially with availability of new imaging methods such as echocardiography and CMR. Papillary muscle hypertrophy defined as at least one of the two papillary muscles if is more than 1.1 cm in either vertical or horizontal diameter[4-5]. Echocardiography can be a useful tool for evaluating papillary muscle hypertrophy, which can cause significant gradient across LVOT. LVOT obstruction, in spite of less ventricular mass, papillary muscle hypertrophy should be considered a variant in the clinical setting.

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Pulmonary Vein Thrombosis Complicating Lung Cancer, Case Report and Literature Review

By Zeine El Abasse, Sara Abouradi, Ejjebli Samia, Salim Arous, Mohamed El Ghali Benouna & Rachida Habbal

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Abstract- *Background:* Pulmonary vein thrombosis (PVT) is a rare clinical entity. The aetiologies are very diverse ranging from malignancy, to hyper-viscosity syndromes and other etiologies. the diagnosis is difficult because the patients may present with dyspnea, cough or hemoptysis which may be confused with an etiology of the pulmonary parenchyma.

Case Report: We present a case of a 40-year-old man with a history of metastatic lung cancer diagnosed with PVT through CT scan showing the mass compressing along the right lower pulmonary vein. Transthoracic echocardiography revealed a left atrium compressed by a mediastinal tissue mass with acceleration of pulmonary venous flow on doppler, this patient was treated with low molecular weight heparin associated with palliative treatment of his cancer.

Conclusions: Patients with PVT often may often have a nonspecific clinical presentation. Anticoagulation should be considered in patients with PVT given the life-threatening complications such as peripheral embolization.

GJMR-I Classification: *NLMC Code: WG 460*

PULMONARYVEINTHROMBOSISCOMPLICATINGLUNGCANCERCASEREPORTANDLITERATUREREVIEW

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Pulmonary Vein Thrombosis Complicating Lung Cancer, Case Report and Literature Review

Zeine El Abasse [α], Sara Abouradi [σ], Ejjebli Samia [ρ], Salim Arous [ω], Mohamed El Ghali Benouna [¥] & Rachida Habbal [§]

Abstract- ***Background:*** Pulmonary vein thrombosis (PVT) is a rare clinical entity. The aetiologies are very diverse ranging from malignancy, to hyper-viscosity syndromes and other etiologies. the diagnosis is difficult because the patients may present with dyspnea, cough or hemoptysis which may be confused with an etiology of the pulmonary parenchyma.

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Conclusions: Patients with PVT often may often have a nonspecific clinical presentation. Anticoagulation should be considered in patients with PVT given the life-threatening complications such as peripheral embolization.

I. Introduction

Pulmonary vein thrombosis (PVT) is relatively rare and under diagnosed but fatal complication in clinical practice. Etiologies can be classified into surgical-related and non-surgical-related etiologies(1) Surgical PVT can occur as a complication following lung transplantation, lobectomy, and radiofrequency catheter ablation (1) Non-surgical etiologies can be due to cardiac causes such as atrial myxoma, pulmonary venous narrowing, atrial fibrillation, or non-cardiac-related causes such as primary or secondary lung malignancies, polycythemia, and hemoglobinopathies such as sickle cell disease(1–3). Only few reports have been described this lethal complication after open lobectomy(4), lung transplantation or in association with metastatic carcinoma however some cases have been described as idiopathic. Pulmonary venous thrombosis may lead to systemic organ and peripheral artery infarction such as in the brain, kidneys, spleen, and peripheral superior and inferior limbs(5) Its exact incidence is unknown and treatment depends on the etiology.

We report here a case of PVT in metastatic lung cancer and the objective of this work is to draw attention to this rare entity with its potentially fatal complications.

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II. Case Presentation

This is a 40-year-old man who presented for 1 months dry cough and large effort dyspnea He also reported weight loss of 7 kg in 10 months and hemoptysis complicated by a deterioration of the general condition. As antecedents, he smoked 1–2 packs/ day. On physical examination, the patient the patient was hemodynamically and respiratory stable with blood pressure (BP) =130/70 mmHg; heart rate (HR) =80 bpm; respiratory rate (RR) =16 rpm, cardiac auscultation was normal. Pulmonary auscultation revealed crackling rales in the lower third of the right hemithorax. The laboratory analysis revealed anaemia with a haemoglobin level of 9.8 g/dL (normal: 11. 5–15), leukocytosis of 14.7 × 103/mm3 (normal: 4. 0–10 × 103), an elevated C-reactive protein (CRP) level of 75 mg/dL (normal: 0–6)

Chest radiography showed a mass over the right lower lung, A CT scan confirmed the x-ray image with the mass compressing along the right lower pulmonary vein. Transthoracic echocardiography revealed a left atrium compressed by a mediastinal tissue mass (Fig 1) with acceleration of pulmonary venous flow on doppler (Fig 2-3) associated with a low abundance pericardial effusion

The assessment of the extension of his tumor objectified bone and hepatic metastases, after a multidisciplinary consultation including cardiologist oncologist and pulmonologist, this patient was treated with low molecular weight heparin associated with palliative treatment of his cancer. During the next 90 days the clinical conditions worsening and the patients died 5 months after the Diagnosis.

III. Discussion

The PVT is a very rare condition, since the pulmonary circulation has an extensive network of collaterals for pulmonary venous drainage (2). The PVT usually presents in relation to a known antecedent that justifies the direct venous pulmonary lesion, reason why we consider crucial to rule out this entity in the patients with presentation symptoms and signs as hemoptysis, pulmonary infiltrate and/or thoracic pain after a potentially damaging procedure for the pulmonary veins such as venous anastomosis in lung transplant patients, lobectomies and lung carcinomas, among others (4).

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Several causes of PVT have been identified, which include surgery involving veins such as lung transplantation or lobectomy; radiofrequency catheter ablation (RFCA) for atrial fibrillation(6); sclerosing mediastinitis; and certain primary or secondary tumors of the lung. Less common causes include: atrial myxoma; congenital pulmonary venous narrowing; and mitral stenosis with an obstructing left atrial clot(7) The case of this patient was discovered during a thoracoabdominal CT scan in the context of his metastatic lung cancer objectifying the PVT.

Any thoracic neoplasm can potentially extendto the pulmonary veins(8), Primary lungneoplasms can give rise to stenosis or invasionof the intrapericardial portion of the pulmonary veins and the left atrium(9)

Extension of a lung neoplasm to the left atrium through apulmonary vein can result in death caused by cardiac arrest or massive systemic tumor embolization of multiple organs(10)

The most frequent malignant cause of PVT is a primary lung neoplasm (11,12). However, PVT can also occur following a metastatic cancer, such as metastatic sarcoma (11), liposarcoma (13), small cell lung cancer(14), and mantle cell lymphoma of the small intestine(2). Our patient was diagnosed with primary lung cancer

Multiple factors probably contribute to thrombosis in pulmonary veins associated with malignancy. These include a hypercoagulable state and mechanical compression of the veins with resultant stasis and damage to the endothelium. (12)This case present a mediatinal mass that compress the pulmonary vein.

The Symptoms depend by the number and the severity of affected pulmonary veins, the collaterals and the rate of progression(15). The form of presentation varies widely and may include dyspnea, chest pain, cough or hemoptysis but these nonspecific symptoms are more frequently related to other diseases such as community-acquired pneumonia (CAP), pulmonary arterial infarction or vasculitis (6). Our patient presented with dyspnea complicated by hemoptysis

Because of the PVT is a life-threatening entity and the variability in presenting symptoms, clinicians must have heightened sensitivity to the presence of the condition, although the diagnosis is now simpler given the increase in radiological explorations performed in our patients.

CXR finding could be nonspecific such as pulmonary and interstitial opacities and pleural effusion, so the final diagnosis is usually obtained with transesophageal echocardiogram, magnetic resonance imaging, which can distinguish between tumor and thrombus, or CT after injection of intravenous contrast in the late phase to reduce flow artifacts.(11).

Echocardiography may demonstrate the extension of the thrombus into the atrium; a transesophageal echocardiogram would be preferable over a trans-thoracic echocardiogram (16)Although not all thrombi can be directly visualized with two-dimensional imaging, measurement of the blood flow velocities in the pulmonary veins can indirectly suggest this diagnosis (i.e. pulmonary vein blood flow acceleration indicates venous obstruction) (17)

Pulmonary vein thrombosis can lead togangrene of the lung and can require a repeat intervention, although development of collateral circulation through the intercostal veins may prevent this complication(18) In addition to pulmonar y infarction, PVT can often be complicated by pulmonary edema, and right ventricular failure(19) peripheral embolism can also occur and lead to limb ischemia, stroke and even a renal infarction.(20,21)

Unfortunately, there are no specific guidelines for the treatment of PVT, but anticoagulation and anti-tumor therapy are used in non resectable tumors(2,7) Other forms of treatment include thrombectomy, which is considered if medical therapy fails and also has been tried successfully for PVT after lobectomy and lung transplant; however, limited data are available in malignancy-induced PVT(22) Lobectomy is considered when PVT is complicated with massive hemoptysis or pulmonary necrosis.

Anticoagulation therapy has been considered to effectively decrease the thrombus and prevent thrombosis formation. In previous reports, approximately80% of patients with a thrombus in the PV stum preceived anticoagulation therapy which led to disappearance of the PV thrombus in most of them, without systemic embolization.(20,23) Therefore, anticoagulation therapy should be recommended in patients with athrombus in the PV stump.

This patient was treated with low molecular weight heparin associated with palliative treatment of his cancer.

IV. Conclusion

Pulmonary vein thrombosis presents in a nonspecific manner. The diagnosis is made with echocardiography. Establishing the diagnosis is crucial given the serious adverse outcomes, including peripheral embolization. The treatment remains challenging including anticoagulation in addition to treating the underlying condition

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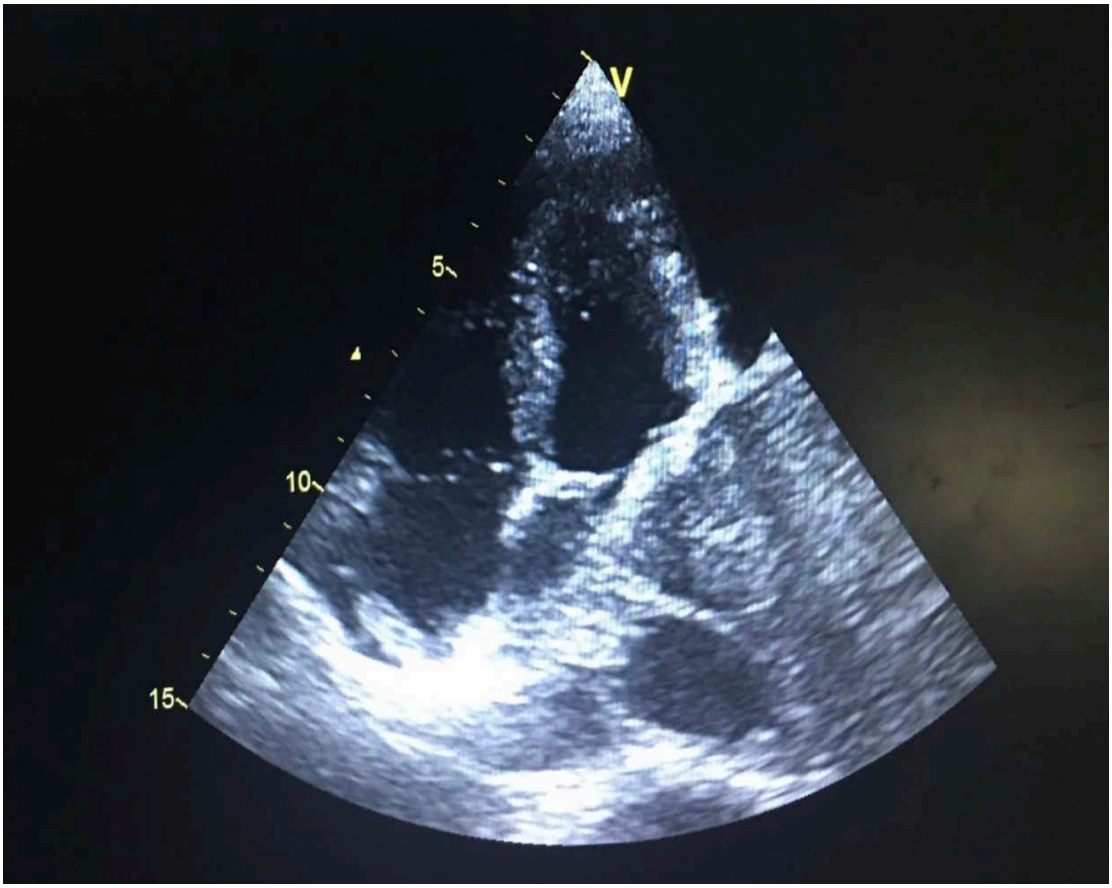


Figure 1: Left atrium compressed by mediastinal mass on transthoracic echocardiography

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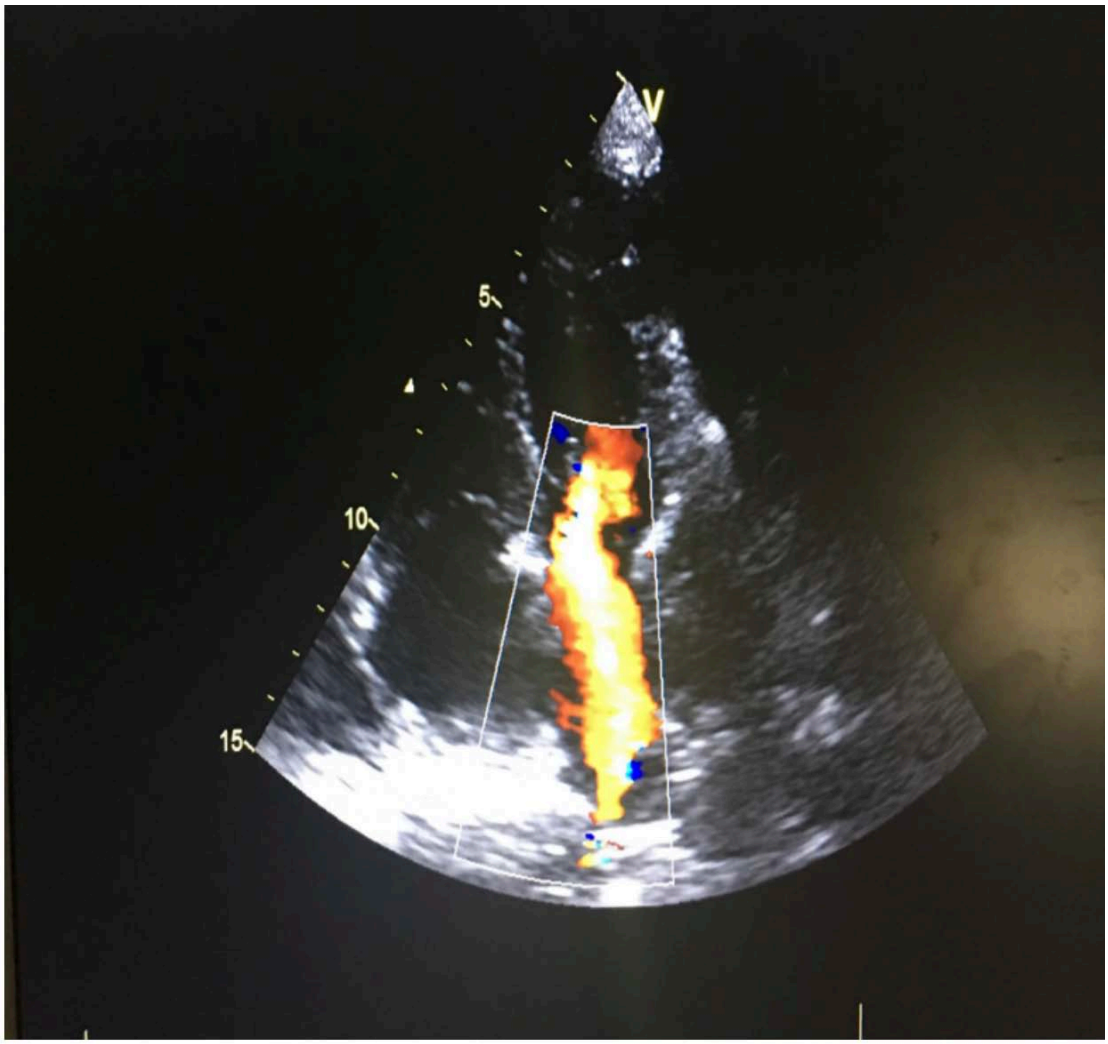


Figure 2: Acceleration of pulmonary venous flow on color Doppler

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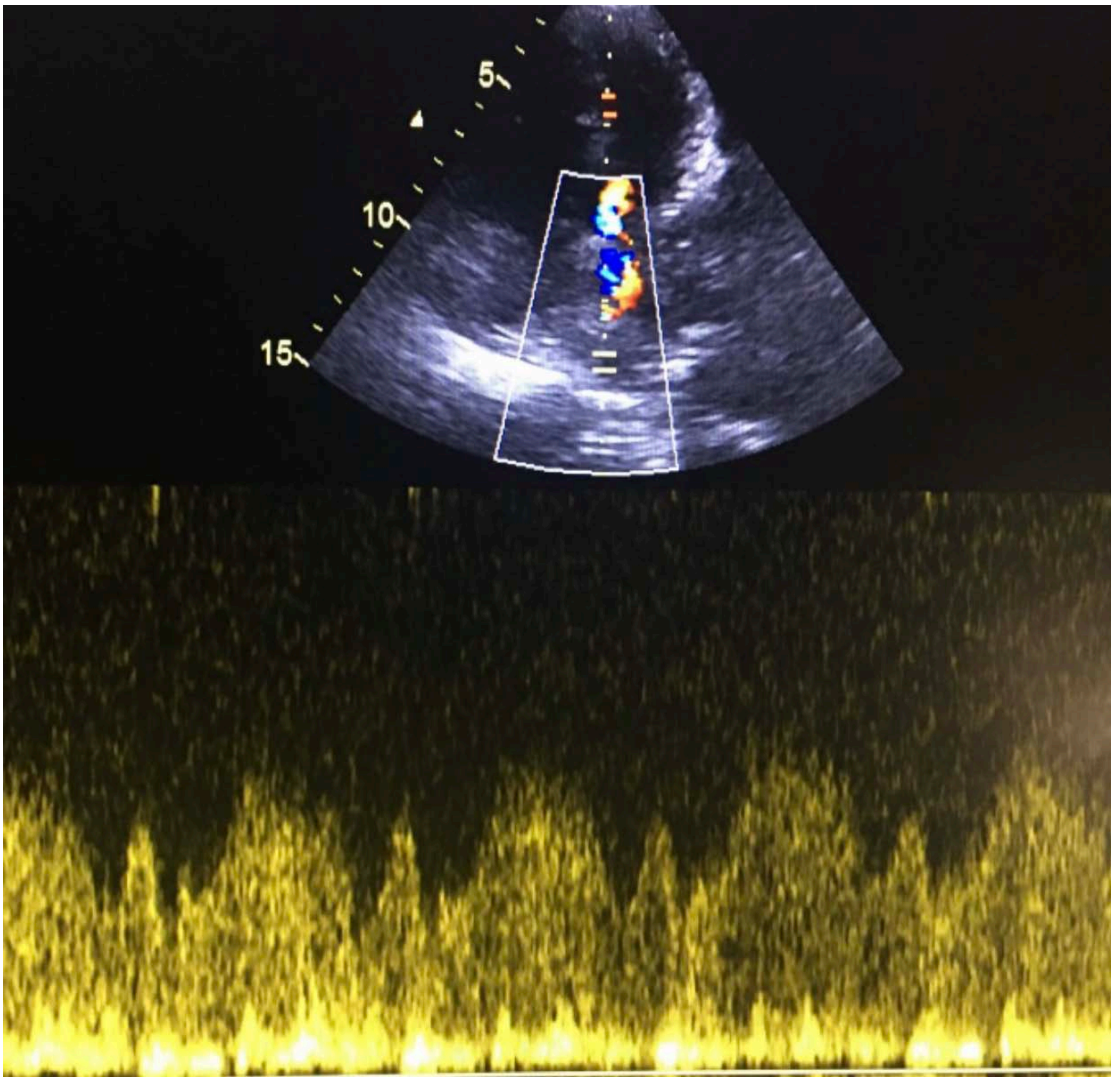


Figure 3: Acceleration of pulmonary venous flow with pulsed Doppler.

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1. Choosing the topic: In most cases, the topic is selected by the interests of the author, but it can also be suggested by the guides. You can have several topics, and then judge which you are most comfortable with. This may be done by asking several questions of yourself, like "Will I be able to carry out a search in this area? Will I find all necessary resources to accomplish the search? Will I be able to find all information in this field area?" If the answer to this type of question is "yes," then you ought to choose that topic. In most cases, you may have to conduct surveys and visit several places. Also, you might have to do a lot of work to find all the rises and falls of the various data on that subject. Sometimes, detailed information plays a vital role, instead of short information. Evaluators are human: The first thing to remember is that evaluators are also human beings. They are not only meant for rejecting a paper. They are here to evaluate your paper. So present your best aspect.

2. Think like evaluators: If you are in confusion or getting demotivated because your paper may not be accepted by the evaluators, then think, and try to evaluate your paper like an evaluator. Try to understand what an evaluator wants in your research paper, and you will automatically have your answer. Make blueprints of paper: The outline is the plan or framework that will help you to arrange your thoughts. It will make your paper logical. But remember that all points of your outline must be related to the topic you have chosen.

3. Ask your guides: If you are having any difficulty with your research, then do not hesitate to share your difficulty with your guide (if you have one). They will surely help you out and resolve your doubts. If you can't clarify what exactly you require for your work, then ask your supervisor to help you with an alternative. He or she might also provide you with a list of essential readings.

4. Use of computer is recommended: As you are doing research in the field of medical research then this point is quite obvious. Use right software: Always use good quality software packages. If you are not capable of judging good software, then you can lose the quality of your paper unknowingly. There are various programs available to help you which you can get through the internet.

5. Use the internet for help: An excellent start for your paper is using Google. It is a wondrous search engine, where you can have your doubts resolved. You may also read some answers for the frequent question of how to write your research paper or find a model research paper. You can download books from the internet. If you have all the required books, place importance on reading, selecting, and analyzing the specified information. Then sketch out your research paper. Use big pictures: You may use encyclopedias like Wikipedia to get pictures with the best resolution. At Global Journals, you should strictly follow here.



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6. Bookmarks are useful: When you read any book or magazine, you generally use bookmarks, right? It is a good habit which helps to not lose your continuity. You should always use bookmarks while searching on the internet also, which will make your search easier.

7. Revise what you wrote: When you write anything, always read it, summarize it, and then finalize it.

8. Make every effort: Make every effort to mention what you are going to write in your paper. That means always have a good start. Try to mention everything in the introduction—what is the need for a particular research paper. Polish your work with good writing skills and always give an evaluator what he wants. Make backups: When you are going to do any important thing like making a research paper, you should always have backup copies of it either on your computer or on paper. This protects you from losing any portion of your important data.

9. Produce good diagrams of your own: Always try to include good charts or diagrams in your paper to improve quality. Using several unnecessary diagrams will degrade the quality of your paper by creating a hodgepodge. So always try to include diagrams which were made by you to improve the readability of your paper. Use of direct quotes: When you do research relevant to literature, history, or current affairs, then use of quotes becomes essential, but if the study is relevant to science, use of quotes is not preferable.

10. Use proper verb tense: Use proper verb tenses in your paper. Use past tense to present those events that have happened. Use present tense to indicate events that are going on. Use future tense to indicate events that will happen in the future. Use of wrong tenses will confuse the evaluator. Avoid sentences that are incomplete.

11. Pick a good study spot: Always try to pick a spot for your research which is quiet. Not every spot is good for studying.

12. Know what you know: Always try to know what you know by making objectives, otherwise you will be confused and unable to achieve your target.

13. Use good grammar: Always use good grammar and words that will have a positive impact on the evaluator; use of good vocabulary does not mean using tough words which the evaluator has to find in a dictionary. Do not fragment sentences. Eliminate one-word sentences. Do not ever use a big word when a smaller one would suffice.

Verbs have to be in agreement with their subjects. In a research paper, do not start sentences with conjunctions or finish them with prepositions. When writing formally, it is advisable to never split an infinitive because someone will (wrongly) complain. Avoid clichés like a disease. Always shun irritating alliteration. Use language which is simple and straightforward. Put together a neat summary.

14. Arrangement of information: Each section of the main body should start with an opening sentence, and there should be a changeover at the end of the section. Give only valid and powerful arguments for your topic. You may also maintain your arguments with records.

15. Never start at the last minute: Always allow enough time for research work. Leaving everything to the last minute will degrade your paper and spoil your work.

16. Multitasking in research is not good: Doing several things at the same time is a bad habit in the case of research activity. Research is an area where everything has a particular time slot. Divide your research work into parts, and do a particular part in a particular time slot.

17. Never copy others' work: Never copy others' work and give it your name because if the evaluator has seen it anywhere, you will be in trouble. Take proper rest and food: No matter how many hours you spend on your research activity, if you are not taking care of your health, then all your efforts will have been in vain. For quality research, take proper rest and food.

18. Go to seminars: Attend seminars if the topic is relevant to your research area. Utilize all your resources.

19. Refresh your mind after intervals: Try to give your mind a rest by listening to soft music or sleeping in intervals. This will also improve your memory. Acquire colleagues: Always try to acquire colleagues. No matter how sharp you are, if you acquire colleagues, they can give you ideas which will be helpful to your research.

20. Think technically: Always think technically. If anything happens, search for its reasons, benefits, and demerits. Think and then print: When you go to print your paper, check that tables are not split, headings are not detached from their descriptions, and page sequence is maintained.

21. Adding unnecessary information: Do not add unnecessary information like "I have used MS Excel to draw graphs." Irrelevant and inappropriate material is superfluous. Foreign terminology and phrases are not apropos. One should never take a broad view. Analogy is like feathers on a snake. Use words properly, regardless of how others use them. Remove quotations. Puns are for kids, not grunt readers. Never oversimplify: When adding material to your research paper, never go for oversimplification; this will definitely irritate the evaluator. Be specific. Never use rhythmic redundancies. Contractions shouldn't be used in a research paper. Comparisons are as terrible as clichés. Give up ampersands, abbreviations, and so on. Remove commas that are not necessary. Parenthetical words should be between brackets or commas. Understatement is always the best way to put forward earth-shaking thoughts. Give a detailed literary review.

22. Report concluded results: Use concluded results. From raw data, filter the results, and then conclude your studies based on measurements and observations taken. An appropriate number of decimal places should be used. Parenthetical remarks are prohibited here. Proofread carefully at the final stage. At the end, give an outline to your arguments. Spot perspectives of further study of the subject. Justify your conclusion at the bottom sufficiently, which will probably include examples.

23. Upon conclusion: Once you have concluded your research, the next most important step is to present your findings. Presentation is extremely important as it is the definite medium though which your research is going to be in print for the rest of the crowd. Care should be taken to categorize your thoughts well and present them in a logical and neat manner. A good quality research paper format is essential because it serves to highlight your research paper and bring to light all necessary aspects of your research.

Informal Guidelines of Research Paper Writing

Key points to remember:

- Submit all work in its final form.
- Write your paper in the form which is presented in the guidelines using the template.
- Please note the criteria peer reviewers will use for grading the final paper.

Final points:

One purpose of organizing a research paper is to let people interpret your efforts selectively. The journal requires the following sections, submitted in the order listed, with each section starting on a new page:

The introduction: This will be compiled from reference matter and reflect the design processes or outline of basis that directed you to make a study. As you carry out the process of study, the method and process section will be constructed like that. The results segment will show related statistics in nearly sequential order and direct reviewers to similar intellectual paths throughout the data that you gathered to carry out your study.

The discussion section:

This will provide understanding of the data and projections as to the implications of the results. The use of good quality references throughout the paper will give the effort trustworthiness by representing an alertness to prior workings.

Writing a research paper is not an easy job, no matter how trouble-free the actual research or concept. Practice, excellent preparation, and controlled record-keeping are the only means to make straightforward progression.

General style:

Specific editorial column necessities for compliance of a manuscript will always take over from directions in these general guidelines.

To make a paper clear: Adhere to recommended page limits.



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Mistakes to avoid:

- Insertion of a title at the foot of a page with subsequent text on the next page.
- Separating a table, chart, or figure—confine each to a single page.
- Submitting a manuscript with pages out of sequence.
- In every section of your document, use standard writing style, including articles ("a" and "the").
- Keep paying attention to the topic of the paper.
- Use paragraphs to split each significant point (excluding the abstract).
- Align the primary line of each section.
- Present your points in sound order.
- Use present tense to report well-accepted matters.
- Use past tense to describe specific results.
- Do not use familiar wording; don't address the reviewer directly. Don't use slang or superlatives.
- Avoid use of extra pictures—include only those figures essential to presenting results.

Title page:

Choose a revealing title. It should be short and include the name(s) and address(es) of all authors. It should not have acronyms or abbreviations or exceed two printed lines.

Abstract: This summary should be two hundred words or less. It should clearly and briefly explain the key findings reported in the manuscript and must have precise statistics. It should not have acronyms or abbreviations. It should be logical in itself. Do not cite references at this point.

An abstract is a brief, distinct paragraph summary of finished work or work in development. In a minute or less, a reviewer can be taught the foundation behind the study, common approaches to the problem, relevant results, and significant conclusions or new questions.

Write your summary when your paper is completed because how can you write the summary of anything which is not yet written? Wealth of terminology is very essential in abstract. Use comprehensive sentences, and do not sacrifice readability for brevity; you can maintain it succinctly by phrasing sentences so that they provide more than a lone rationale. The author can at this moment go straight to shortening the outcome. Sum up the study with the subsequent elements in any summary. Try to limit the initial two items to no more than one line each.

Reason for writing the article—theory, overall issue, purpose.

- Fundamental goal.
- To-the-point depiction of the research.
- Consequences, including definite statistics—if the consequences are quantitative in nature, account for this; results of any numerical analysis should be reported. Significant conclusions or questions that emerge from the research.

Approach:

- Single section and succinct.
- An outline of the job done is always written in past tense.
- Concentrate on shortening results—limit background information to a verdict or two.
- Exact spelling, clarity of sentences and phrases, and appropriate reporting of quantities (proper units, important statistics) are just as significant in an abstract as they are anywhere else.

Introduction:

The introduction should "introduce" the manuscript. The reviewer should be presented with sufficient background information to be capable of comprehending and calculating the purpose of your study without having to refer to other works. The basis for the study should be offered. Give the most important references, but avoid making a comprehensive appraisal of the topic. Describe the problem visibly. If the problem is not acknowledged in a logical, reasonable way, the reviewer will give no attention to your results. Speak in common terms about techniques used to explain the problem, if needed, but do not present any particulars about the protocols here.

The following approach can create a valuable beginning:

- Explain the value (significance) of the study.
- Defend the model—why did you employ this particular system or method? What is its compensation? Remark upon its appropriateness from an abstract point of view as well as pointing out sensible reasons for using it.
- Present a justification. State your particular theory(-ies) or aim(s), and describe the logic that led you to choose them.
- Briefly explain the study's tentative purpose and how it meets the declared objectives.

Approach:

Use past tense except for when referring to recognized facts. After all, the manuscript will be submitted after the entire job is done. Sort out your thoughts; manufacture one key point for every section. If you make the four points listed above, you will need at least four paragraphs. Present surrounding information only when it is necessary to support a situation. The reviewer does not desire to read everything you know about a topic. Shape the theory specifically—do not take a broad view.

As always, give awareness to spelling, simplicity, and correctness of sentences and phrases.

Procedures (methods and materials):

This part is supposed to be the easiest to carve if you have good skills. A soundly written procedures segment allows a capable scientist to replicate your results. Present precise information about your supplies. The suppliers and clarity of reagents can be helpful bits of information. Present methods in sequential order, but linked methodologies can be grouped as a segment. Be concise when relating the protocols. Attempt to give the least amount of information that would permit another capable scientist to replicate your outcome, but be cautious that vital information is integrated. The use of subheadings is suggested and ought to be synchronized with the results section.

When a technique is used that has been well-described in another section, mention the specific item describing the way, but draw the basic principle while stating the situation. The purpose is to show all particular resources and broad procedures so that another person may use some or all of the methods in one more study or referee the scientific value of your work. It is not to be a step-by-step report of the whole thing you did, nor is a methods section a set of orders.

Materials:

Materials may be reported in part of a section or else they may be recognized along with your measures.

Methods:

- Report the method and not the particulars of each process that engaged the same methodology.
- Describe the method entirely.
- To be succinct, present methods under headings dedicated to specific dealings or groups of measures.
- Simplify—detail how procedures were completed, not how they were performed on a particular day.
- If well-known procedures were used, account for the procedure by name, possibly with a reference, and that's all.

Approach:

It is embarrassing to use vigorous voice when documenting methods without using first person, which would focus the reviewer's interest on the researcher rather than the job. As a result, when writing up the methods, most authors use third person passive voice.

Use standard style in this and every other part of the paper—avoid familiar lists, and use full sentences.

What to keep away from:

- Resources and methods are not a set of information.
- Skip all descriptive information and surroundings—save it for the argument.
- Leave out information that is immaterial to a third party.



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Results:

The principle of a results segment is to present and demonstrate your conclusion. Create this part as entirely objective details of the outcome, and save all understanding for the discussion.

The page length of this segment is set by the sum and types of data to be reported. Use statistics and tables, if suitable, to present consequences most efficiently.

You must clearly differentiate material which would usually be incorporated in a study editorial from any unprocessed data or additional appendix matter that would not be available. In fact, such matters should not be submitted at all except if requested by the instructor.

Content:

- Sum up your conclusions in text and demonstrate them, if suitable, with figures and tables.
- In the manuscript, explain each of your consequences, and point the reader to remarks that are most appropriate.
- Present a background, such as by describing the question that was addressed by creation of an exacting study.
- Explain results of control experiments and give remarks that are not accessible in a prescribed figure or table, if appropriate.
- Examine your data, then prepare the analyzed (transformed) data in the form of a figure (graph), table, or manuscript.

What to stay away from:

- Do not discuss or infer your outcome, report surrounding information, or try to explain anything.
- Do not include raw data or intermediate calculations in a research manuscript.
- Do not present similar data more than once.
- A manuscript should complement any figures or tables, not duplicate information.
- Never confuse figures with tables—there is a difference.

Approach:

As always, use past tense when you submit your results, and put the whole thing in a reasonable order.

Put figures and tables, appropriately numbered, in order at the end of the report.

If you desire, you may place your figures and tables properly within the text of your results section.

Figures and tables:

If you put figures and tables at the end of some details, make certain that they are visibly distinguished from any attached appendix materials, such as raw facts. Whatever the position, each table must be titled, numbered one after the other, and include a heading. All figures and tables must be divided from the text.

Discussion:

The discussion is expected to be the trickiest segment to write. A lot of papers submitted to the journal are discarded based on problems with the discussion. There is no rule for how long an argument should be.

Position your understanding of the outcome visibly to lead the reviewer through your conclusions, and then finish the paper with a summing up of the implications of the study. The purpose here is to offer an understanding of your results and support all of your conclusions, using facts from your research and generally accepted information, if suitable. The implication of results should be fully described.

Infer your data in the conversation in suitable depth. This means that when you clarify an observable fact, you must explain mechanisms that may account for the observation. If your results vary from your prospect, make clear why that may have happened. If your results agree, then explain the theory that the proof supported. It is never suitable to just state that the data approved the prospect, and let it drop at that. Make a decision as to whether each premise is supported or discarded or if you cannot make a conclusion with assurance. Do not just dismiss a study or part of a study as "uncertain."

Research papers are not acknowledged if the work is imperfect. Draw what conclusions you can based upon the results that you have, and take care of the study as a finished work.

- You may propose future guidelines, such as how an experiment might be personalized to accomplish a new idea.
- Give details of all of your remarks as much as possible, focusing on mechanisms.
- Make a decision as to whether the tentative design sufficiently addressed the theory and whether or not it was correctly restricted. Try to present substitute explanations if they are sensible alternatives.
- One piece of research will not counter an overall question, so maintain the large picture in mind. Where do you go next? The best studies unlock new avenues of study. What questions remain?
- Recommendations for detailed papers will offer supplementary suggestions.

Approach:

When you refer to information, differentiate data generated by your own studies from other available information. Present work done by specific persons (including you) in past tense.

Describe generally acknowledged facts and main beliefs in present tense.

The Administration Rules

Administration Rules to Be Strictly Followed before Submitting Your Research Paper to Global Journals Inc.

Please read the following rules and regulations carefully before submitting your research paper to Global Journals Inc. to avoid rejection.

Segment draft and final research paper: You have to strictly follow the template of a research paper, failing which your paper may get rejected. You are expected to write each part of the paper wholly on your own. The peer reviewers need to identify your own perspective of the concepts in your own terms. Please do not extract straight from any other source, and do not rephrase someone else's analysis. Do not allow anyone else to proofread your manuscript.

Written material: You may discuss this with your guides and key sources. Do not copy anyone else's paper, even if this is only imitation, otherwise it will be rejected on the grounds of plagiarism, which is illegal. Various methods to avoid plagiarism are strictly applied by us to every paper, and, if found guilty, you may be blacklisted, which could affect your career adversely. To guard yourself and others from possible illegal use, please do not permit anyone to use or even read your paper and file.

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Please note that following table is only a Grading of "Paper Compilation" and not on "Performed/Stated Research" whose grading solely depends on Individual Assigned Peer Reviewer and Editorial Board Member. These can be available only on request and after decision of Paper. This report will be the property of Global Journals.

Topics	Grades		
	A-B	**C-D**	**E-F**
Abstract	Clear and concise with appropriate content, Correct format. 200 words or below	Unclear summary and no specific data, Incorrect form Above 200 words	No specific data with ambiguous information Above 250 words
Introduction	Containing all background details with clear goal and appropriate details, flow specification, no grammar and spelling mistake, well organized sentence and paragraph, reference cited	Unclear and confusing data, appropriate format, grammar and spelling errors with unorganized matter	Out of place depth and content, hazy format
Methods and Procedures	Clear and to the point with well arranged paragraph, precision and accuracy of facts and figures, well organized subheads	Difficult to comprehend with embarrassed text, too much explanation but completed	Incorrect and unorganized structure with hazy meaning
Result	Well organized, Clear and specific, Correct units with precision, correct data, well structuring of paragraph, no grammar and spelling mistake	Complete and embarrassed text, difficult to comprehend	Irregular format with wrong facts and figures
Discussion	Well organized, meaningful specification, sound conclusion, logical and concise explanation, highly structured paragraph reference cited	Wordy, unclear conclusion, spurious	Conclusion is not cited, unorganized, difficult to comprehend
References	Complete and correct format, well organized	Beside the point, Incomplete	Wrong format and structuring

INDEX

A

Aortic · 2, 3, 5, 26
Auscultation · 2, 29

C

Congenital · 1, 3, 30

D

Deformity · 10, 11, 12, 14, 16, 17, 18
Distention · 2

E

Embolism · 20, 21, 22, 24, 31

G

Gradient · 2, 5, 8, 25, 26, 27

I

Iatrogenic · 1, 2, 5

M

Mutation · 27

R

Resonance · 25, 27, 30
Resuscitation · 20, 21
Rheumatic · 1, 2, 3, 5

S

Sedimentation · 22

V

Viscosity · 29

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