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ISSN Print: 2325-7075    ISSN Online: 2325-7083
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The figure on the front cover is from the article published in Case Reports in Clinical Medicine, 2016, Vol. 5, No. 9, pp. 335-341 by Yoshiro Imai, Ryo Iida, Masahiko Nitta and Akira Takasu.
Case Reports in Clinical Medicine (CRCM)

Journal Information

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Bilateral Simple Orthotopic Ureteroceles in an Adult Male Complaining of Urgency and Pain on Urination

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Abstract

We report a case of a 38-year-old man with a simple orthotopic bilateral ureterocele and pain on urination that was improved by transurethral incision. His symptoms were present for 3 years since he first visited our hospital in 2012. Cystoscopy at our hospital revealed a simple bilateral ureterocele, and he was given an alpha blocker plus anticholinergic agent. The medication did not relieve his symptoms. Three years after his initial visit in 2012, he returned for a re-evaluation in May, 2015, because his symptoms had worsened. Computed Tomography (CT) showed normal locations of the bilateral ureteric orifices in the bladder, bilateral ureterocele with a right-sided predominance and a cobra head sign, and bilateral hydroureter. We performed a transurethral incision of his ureteroceles. Intraoperatively, we found that the ureterocele on the right ureter entered the opening and expanded at the time of urine discharge. The symptoms significantly improved after the procedure.

Keywords

Simple Orthotopic Ureterocele, Transurethral Incision of Ureteroceles, Ureterocele Complications

1. Introduction

Simple ureterocele generally has few symptoms and is often treated conservatively [1] [2]. The most frequent symptoms are urinary tract infection, dysuria, palpable abdominal tumor, and vesicoureteral reflux (VUR). The majority of ureteroceles are related to duplex systems and are ectopic. Simple ureterocele usually remain asymptomatic and/or unrecognized in adults. We report a case of an adult male with simple orthotopic bilateral ureteroceles and pain on urination that was improved by transurethral
incision.

2. Case Presentation

A 38-year-old man with a history of childhood asthma visited a community hospital complaining of urgency and pain on urination present for 3 years. Cystoscopy at our hospital revealed a simple bilateral ureterocele, and he was given an alpha blocker plus anticholinergic agent (Figure 1). The medication did not relieve his symptoms, and he experienced adverse effects including dizziness. The medication was thus discontinued, and he left our hospital.

Three years after his initial visit in 2012, he returned for a re-evaluation in May, 2015, because his symptoms had worsened. Laboratory evaluation revealed white blood cells (WBC) 6300 cells/mL, hemoglobin 16.0 g/mL, hematocrit 45.1%, blood glucose 175 mg/dL, blood urea nitrogen 14.5 mg/dL, creatinine 0.73 mg/dL, and C-reactive protein 0.05 mg/dL; 0-1 WBC and 0-1 erythrocyte per high-power field were seen in his urine.

On cystoscopy, the bilateral ureterocele appeared larger than it was 3 years previously (Figure 2). Computed Tomography (CT) showed normal locations of the bilateral ureteric orifices in the bladder, bilateral ureterocele with a right-sided predominance and a cobra head sign, and bilateral hydronephrosis (Figure 3). The patient’s symptoms were slightly relieved following prescription of tadalafil 5 mg/day. However, after requesting a consultation for further symptom relief, he consented to surgical treatment, and we performed a transurethral incision of his ureteroceles. Intraoperatively, we found that the ureterocele on the right ureter entered the opening and expanded at the time of urine discharge. The ureterocele on left side also entered the ureteric orifice, but was smaller than the one on the right (Figure 4). After performing the incision, we confirmed that, on both sides, the ureteric orifices did not act like golf holes to permit VUR after the operation. The symptoms significantly improved after the procedure, and the patient was satisfied with the outcome. No postoperative complications or pyelonephritis occurred, and the symptoms have not reappeared in the 3 months following surgery.
Figure 2. Cystoscopy performed in 2015 shows that the bilateral ureterocele had increased in size ((a) right; (b) left).

Figure 3. Computed tomography (CT) performed in 2015 showed normal locations of the ureteric orifice, bilateral ureterocele ((a) coronal; (b) sagittal) with a right-sided predominance and a cobra head sign (red arrow), and bilateral hydroureter ((c) blue arrows).

Figure 4. Intraoperative finding in 2015 showing the right ureterocele on the ureter orifice, expansion of the right ureterocele at the time of urine discharge (a), incision of the ureterocele (b), and the ureterocele after incision (c).

3. Discussion

Ureteroceles are more common in women, with the female to male ratio of 4 to 1 [3].
Ureteroceles are bilateral in 10% and occur in 80% in upper poles in duplex systems [4]. This case was a simple bilateral single system ureterocele located in a normal ureteric orifice. A simple ureterocele usually has few symptoms. Symptoms that lead to the discovery of a simple ureterocele include urinary tract infection, sharp pain caused by a calculus in the ureterocele, dysuria caused by a hugecele that obstructs the internal urethral orifice, or a palpable abdominal tumor that acts like a hugecele [5]. A case of prolapse of a simple ureterocele presenting as perineural tumor was reported in a young woman [6]. Treatment of simple ureteroceles is generally conservative. If recurring urinary tract infections caused by urinary tract obstruction, cannot be resolved by treatment with antibiotics or VUR, surgical treatment may be considered. Transurethral incision is often performed for ureteroceles, and VUR is a common surgical complication [7]. This patient’s chief complaint was urgency and pain on urination, which rarely leads to suspicion of a ureterocele. He had no episodes of repeated urinary tract infection or VUR, but based on the cystoscopy findings, we have concluded his symptoms might have been caused by expansion of the ureteroceles and tugging of the vesical trigon at the time of urine discharge. We also have concluded that the symptoms would have improved following removal of the ureteroceles. We performed the transurethral incision of the ureteroceles when the patient gave his consent after being told that his symptoms might not improve following the procedure. Considering the improvement of the symptoms and the patient’s satisfaction with the outcome, we believe that the ureteroceles may have been responsible for the urinary symptoms.

4. Conclusion

We report the case of an adult male with simple orthotopic bilateral ureteroceles and pain on urination that was improved by transurethral incision.

Acknowledgements

The authors would like to thank Enago (www.enago.jp) for reviewing the English language of the original manuscript.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Patient Consent

Obtained.

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Late Presentation of Bronchial Foreign Body in Young Adult: A Case Report

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Abstract

Foreign body aspiration is common in young children or in adults with advanced age and altered mentation. Usually symptoms present acutely, leading to early intervention. We present a rare case of a healthy young female who aspirated her nasal ring but remained asymptomatic for almost eight years. The patient ultimately required a right upper lobe lobectomy for bronchiectasis.

Keywords

Bronchial, Foreign Body, Lobectomy

1. Introduction and Review of Literature

Accidental inhalation of foreign body in children has been known since time immemorial. They present with life threatening symptoms of choking, coughing, wheezing and respiratory distress or go undetected till investigated for frequent upper respiratory infection and persistent cough. Aspiration of foreign body in adults is less common, and usually confined to advanced age or patients with poor dentition, altered sensorium, drug use, etc. One of the largest series published identified 65 adults with tracheobronchial foreign body aspiration over a period of 12 years [1]. A nonasphyxiating foreign body may be asymptomatic. Many of these objects are wedged distally and patients do not often remember choking, the aspiration, or any precipitating event. Thus, foreign body aspiration is commonly misdiagnosed. Diagnosis can be delayed for months to years from the initial event [2] [3], and often results from fortuitous discov-
ery when fibrobronchoscopy is performed to evaluate a chronic cough, hemoptysis, or a slow resolution of pneumonia. The use of bronchoscopy for foreign body removal was introduced by Gustav Killian in 1897. Currently, both rigid and flexible bronchoscopes are utilized for this indication, depending on the age of the patient and the characteristics of the foreign body. Because of improvement in the accessories used to remove foreign body, survival is good and morbidity is low following bronchoscopic foreign body removal. We report a rare case of accidental aspiration of a nasal ring in a 35-year-old female. The patient had remained essentially asymptomatic for eight years since she first presented with hemoptysis.

2. Case Report

A 35-year-old married female presented to us with history of hemoptysis of six months duration. There was no other history of excessive cough or fever. A chest X-Ray (Figure 1 & Figure 2) showed, to our utter surprise, a nasal ring in the upper zone of right lung with evidence of mild bronchiectatic changes in the right lung. On close interrogation, the patient did remember losing this ring about eight years back while asleep at night and had been searching for it since next day morning. The patient was unaware of having aspirated nasal ring as she did not experience any symptoms of choking, coughing or respiratory distress. She remained asymptomatic for next eight years except for occasional episodes of upper respiratory tract infections and fever off and on, which were ignored by her primary care physician.

On admission, a computerised tomography scan of chest was performed, which revealed collapse of right upper lobe with bronchiectatic changes (the CT scan images are however not available). Bronchoscopic removal of the foreign body was attempted but was not successful, as the foreign body was deeply impacted in the right upper lobe. A decision was taken to proceed with right upper lobectomy. The procedure was performed via a standard right postero-lateral thoracotomy. During the procedure, the upper lobe bronchus was encircled and divided carefully to recover the offending ring (Figure 3). The patient had an uneventful post-operative recovery and is doing well on last follow-up which was 6 months after the surgery.

3. Signs and Symptoms of Inhaled Foreign Body

The diagnosis of foreign body aspiration can be difficult, especially if the patient does not recall an aspiration episode. Foreign body aspiration has variable clinical manifestations, ranging from trivial symptoms to irreversible lung damage and life-threatening infection, atelectasis, and massive hemoptysis. Patients may present with a history of fever, breathlessness, or wheezing or with features of a non-resolving pneumonia. On physical examination, these patients may have decreased breath sounds on the side with the foreign body or localized wheezing, or they may be asymptomatic. The clinical triad that is considered to be diagnostic of foreign body aspiration consists of wheezing, coughing, and diminished or absent breath sounds.

Complications include: recurrent pneumonias, bronchiectasis, bronchial strictures,
Figure 1. Chest X-ray postero-anterior view showing the retained foreign body in upper zone of the right lung along with bronchiectatic changes in the adjacent lung.

Figure 2. Chest X-ray lateral view showing the retained foreign body.

Figure 3. Intra-operative photograph showing the foreign body in the bronchial lumen
hemoptysis and development of inflammatory polyps at the site of impaction.

4. Discussion

Foreign body aspiration refers to the inhalation of solid and liquid material into the airways. The foreign body may be lodged in to the main bronchus and its branches and may even reach the lungs. The right main bronchus is frequently implicated because of more vertical path. The upper lobe and the superior segment of the lower lobes are thus commonly affected when the patients are in recumbent position. Foreign body aspiration is a common accident in childhood with a high incidence of mortality and morbidity. Children between one to three years of age are more vulnerable [4] [5], due to their lack of dentition and inherent nature to explore their world by putting objects in their mouth. The other age group which is affected by foreign body aspiration is elderly people with poor dentition and with altered sensorium, such as with history of drug or alcohol use. It is extremely rare to find foreign bodies in young healthy adults, as in our case, and that too without producing any apparent symptoms.

Many varieties of foreign bodies may be inhaled, with organic foreign bodies being more common than inorganic (67% versus 33%) [6]. The severity of signs and symptoms depend on site, size, composition and duration of lodgement of the foreign body. Organic foreign bodies are more dangerous as compared to metallic ones due to the severe lipoid reaction caused by them. Amongst the inorganic foreign bodies those with sharp edges cause early symptoms. Longer the foreign body stays in the airway, the more likely it is to migrate distally and to give rise to inflammatory reactions, granulation and impaction. This is what happened in our case, because of which bronchoscopic removal was unsuccessful.

Diagnostic imaging plays a variable role. Most of the foreign bodies are not radiopaque. Plain films, therefore, may be fallacious unless they are taken in full expiratory phase. On expiration, air trapping, obstructive emphysema and mediastinal shift may be documented as corroborative evidences. When chest radiograph is normal but suspicion is high helical CT and virtual bronchoscopy can be considered [7].

The rigid bronchoscope remains the gold standard for safe removal of tracheobronchial foreign body [8]. Surgery is very rarely called for. In our case, the only feasible option was a lobectomy as endoscopic removal had failed due to the chronicity of the foreign body and the patient was having hemoptysis due to bronchiectatic changes in the lung.

Informed consent was taken from the patient to report this case. The type of foreign body reported here is not unusual but the course of events is. Often patients have remembered the history of inhalation. However, in our case the patient missed both the inhalation and signs and symptoms of aspiration. Probably inert gold material had minimal tissue reaction and the metallic object allowed the movement of air in the lung without complete obstruction. She did not have any predisposing factors either like advanced age, neurological disorders, alcohol consumption, poor dentition and sedative use [9] [10]. Diagnosis was delayed for eight long years probably due to incomplete ob-
struction of the airway. Gradually patient may have developed bronchiectatic changes, due to chronic irritation by the foreign body, superimposed with some lower respiratory tract infection which led to delayed presentation with hemoptysis and the ultimate penalty was in the form of lobectomy.

What makes this case unusual is the rather delayed and innocuous presentation after aspiration of such a large foreign object. It emphasizes the fact that healthy adults may tolerate aspiration of foreign bodies for a long time without acute life-threatening consequences. This case shows the importance of further investigation in patients with obstructive airway disease who are unresponsive to routine therapies. Although the diagnosis of foreign body aspiration could be established rather easily in our case due to the radio-opaque nature of the object, this might be particularly difficult in others particularly those with aspiration of radiolucent foreign bodies. The inclusion of foreign body aspiration in the differential diagnosis for such patients allows for early recognition and appropriate management, thereby decreasing the incidence of costly and unnecessary complications. This unusual case emphasizes the need to maintaining a high index of suspicion when an adult presents with chronic complaints of upper respiratory tract infection without any obvious reasons and highlights the need to create awareness about this potentially avoidable situation at the level of general practitioners.

5. Experience Summary

In conclusion, bronchoaspiration of foreign bodies by adults is rare. However, it is important to suspect bronchoaspiration of a foreign body in the presence of suggestive symptoms. In adults, respiratory infection (acute or recurrent pneumonia) is the most common clinical presentation. Chest radiography is not always useful for diagnosis. Sometimes metallic foreign bodies can be observed, but other times only indirect signs of alveolar infiltrate are present. Surgical treatment is reserved for cases in which bronchoscopy is unsuccessful or there are irreversible bronchial or lung complications.

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Apical Left Ventricular Hypertrophic Cardiomyopathy: A Case Report

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Abstract
Apical hypertrophic cardiomyopathy (apical HCM) is a rare variant of hypertrophic cardiomyopathy with a prevalence of 1% - 2% in Asian population and carries a benign prognosis. It is usually silent in early stages and manifests in adults with a suspicion of typical ECG changes of giant T wave inversion in left precordial leads. Transthoracic echocardiography is the mainstay of non-invasive diagnosis and provides a heterogeneous appearance of its morphological features with a spade-shaped LV (left ventricular) cavity. Background of this case study describes the apical HCM in an asymptomatic male at the age of 54 years old and also predicts the mixed and mid-ventricular forms of left ventricular HCM at this region of Thoothukudi in India.

Keywords
Apical Hypertrophy, Left Ventricle, Spade-Shaped LV Cavity, Echocardiography, Yamaguchi Syndrome, Apical Ballooning Syndrome

1. Introduction
Apical hypertrophic cardiomyopathy (apical HCM) is an atypical phenotype of non-obstructive HCM (hypertrophic cardiomyopathy) and it is more prevalent in Japanese people [1]. Usually it is considered as a benign condition and is detected incidentally by echocardiography.

Review of Literature
Apical HCM was first described in Japan. Sakamoto et al. first described the ECG pattern of apical HCM in 1976 [2] in Japanese patients. But it was Yamaguchi that described the syndrome and its ventriculographic features in 1979 [3]. Kubo and col-
leagues [4] used echocardiography in a cohort of 264 patients to define the imaging characteristics and Chen et al. [5] utilized 2D echocardiography to further classify apical HCM and to assess the wall thickness. Kitaoka et al. found the apical HCM in 15% of Japanese and 3% of American patients of HCM. Apical HCM constitutes 8% - 10% [6] [7] of non-Japanese population. It is rare in the West (1 to 11%) [8], but more common in oriental people and accounts for 13% - 41% of all variants of HCM among Asian individuals [9], 16% - 41% of entire HCM population in Chinese [10] and 24.5% of all HCM in Taiwan [11]. Sakamoto et al. noticed the benign prognosis of apical HCM in a study on 200 patients in Japan [12].

Published Indian studies are limited for comparison, so this case had been reported.

2. Case Report

A 54-year-old asymptomatic male was referred for echocardiographic evaluation due to an LVH (left ventricular hypertrophy) pattern of ECG changes as shown in Figure 1 and a normal X-Ray chest as in Figure 2. His pulse rate was 88 bpm and blood pressure 130/80 mmHg. Blood chemistry revealed normal. He had no physical findings. Trans-thoracic echocardiographic images revealed an asymmetric apical hypertrophy (AAH) in four chamber views and a concentric apical hypertrophic pattern in apical long axis views as shown in Figures 3-10 given below. Screening of family members revealed normal and the patient was given small dose of ACE (angiotensin-converting enzyme) inhibitors such as ramipril 1.25 mg daily and advised close follow up.

3. Discussion

3.1. Etiopathogenesis

Hypertrophic cardiomyopathy (HCM) is the most common genetically transmitted cardiovascular disease and it is usually familial with heterogeneous expression. Several disease-causing mutations in genes encoding proteins of the sarcomere have been reported [13]. A family history is more common in patients with asymmetric septal hypertrophy (ASH) than with apical hypertrophic cardiomyopathy (AHCM). Apical hypertrophic cardiomyopathy is frequently sporadic, a few families have been reported with autosomal dominant inheritance and a sarcomere gene mutation in the alpha-cardiac actin gene (Glu101Lys) has been shown to consistently producing the apical HCM phenotype [14]. Alpha and beta cardiac myosin heavy chain (MHC) mutations affect the polypeptides crucial to the structure of myofibrils and might be responsible for the myocyte and myofibrillar disarray, characteristic of familial HCM. The cause of sporadic apical HCM is unknown, but genetic, racial and even environmental factors could be responsible and many have lesser degree of hypertrophy, the outflow gradients are usually lacking, symptoms are often absent and the disease is detected only by echocardiography. An association with HLA-DR2 antigen in patients with apical HCM was recently reported in Japan [15]. Other investigators consider a secondary genesis as the underlying pathogenetic mechanism, i.e., hypertension or heavy physical exercise [16].
Figure 1. ECG showing the “strain” pattern—deep T wave inversion (7 mm) with 5 mm J point depression in precordial and limb leads and a positive Sokolow-Lyon index (SV$_1$ + RV$_5$ = 50 mm) as a sign of LVH (left ventricular hypertrophy) (1 mV = 5 mm standardization).

Figure 2. X-ray chest PA (posterior-anterior) view revealed normal.
Left ventricular hypertrophy is a gross anatomic marker and major determinant of the clinical feature of the disease [17]. The increased left ventricular mass is almost entirely due to increased wall thickness and the left ventricular cavity is usually small or normal in size. The distribution of wall thickening varies so greatly in HCM and there is no single classic morphologic pattern. Relatives with same genetic substrate usually have dissimilar pattern of left ventricular hypertrophy. All possible patterns of hypertrophy have been observed and occasionally, HCM shows segmental wall thickening confined to the left ventricular apex, a morphological form that in Japan has been associated with a spade shaped deformity of the left ventricle and giant negative T waves in electrocardiography (ECG) [18]. In some patients with HCM have substantial hyper-
Figure 5. Showing mild mitral regurgitation, due to trauma on AML (anterior mitral leaflet) as the result of contact with septum and hypertrophied segment during cardiac cycle in apical long axis view. Arrow mark indicates apical hypertrophy.

Figure 6. Showing the “spade-shaped” or “bird’s beak” LV (left ventricular) cavity at the onset of systole-mitral valve begins to close) in Tilted apical view. Arrow mark indicates apical hypertrophy.

trophy in unusual locations such as the posterior portion of the septum, the postero-basal free wall and the mid-ventricular level [19].

Embryologically, asymmetric septal hypertrophy results from postnatal persistence of a normal anatomic feature of the developing heart [20] [21]. The disproportionate thickening of the ventricular septum is characteristic of the normal embryonic and fetal human heart. Even though prominent hypertrophy may be found in infants, the typical patient develops left ventricular hypertrophy during adolescence [22] after a period of prolonged latency. However, HCM may occur at any time in adult life due to mutation of cardiac myosin-binding protein C [23].
3.2. Morphological Types of Apical HCM

According to the distribution of hypertrophied musculature, the morphologic sub classification as “true apical” phenotype (hypertrophy of only the apical segment below the papillary muscle) and “distal-dominant” phenotype (hypertrophy extended into the middle LV segments) have been recently recognized in North America [24]. Mid ventricular obstruction or apical obliteration was frequently found in patients with distal-dominant form and it is more often symptomatic and more likely to develop cardi-
Figure 9. Showing the Apical HCM in short axis view. The ratio of apical thickness to posterior wall thickness is 3.7:1, suggesting apical HCM (hypertrophic cardiomyopathy, i.e., the ratio is > 1.5:1).

Figure 10. Apical left ventricular long axis view showing the hypertrophy confined to LV (left ventricular) apex below the papillary muscles (PM) and the papillary muscles are not hypertrophied.

Ovvascular events. Other investigators have separated the AHCM into “pure form” (apical segment only) and “mixed form” (hypertrophy extending into other segments) [25]. Based on CMR (cardiac magnetic resonance) imaging, apical HCM is divided into 3 types as true apical form, a type with additional asymmetric involvement of ventricular wall segments and a type with symmetric involvement of ventricular wall segments. Generally, apical HCM is divided into two groups as isolated asymmetric apical HCM (pure AHCM) and co-existent hypertrophy of interventricular septum (mixed AHCM) [26]. The pure form is predominant in Japanese, while the mixed form has been linked to Caucasian patients [27] [28].
A relatively normal-sized, diffusely hypokinetic left ventricle with an unexplained, pathological hypertrophy is termed as “burned-out” hypertrophic cardiomyopathy.

### 3.3. Clinical Presentation

Clinical expression of apical HCM is highly variable. Apical HCM may manifest early in adulthood [29] and most series reported a mean age of at least 41 years [30]. About 54% of patients with apical HCM are symptomatic and the most common symptoms are chest pain, followed by palpitations, dyspnea and syncope. According to one large published series, AHCM presented with atypical chest pain (14%), palpitations (10%), dyspnea (6%), presyncope/syncope (6%) [31]. Atypical chest pain is the most frequent symptom and typical angina may also occur due to diminished vasodilator reserve. Atrial fibrillation (12%), apical myocardial infarction (10%), ventricular arrhythmia and apical thrombosis with subsequent embolization may occur up to 33% of cases [32]. [33]. The mismatch between fixed epicardial blood supply and large muscle mass leads to sustained myocardial ischemia and necrosis resulting apical aneurysm which may complicate with ventricular tachycardia. A 65-year-old Indian woman having apical aneurysm and presented with long standing angina had been reported [34].

Apical hypokinesis and aneurysm formation may also result from midventricular (mid-cavity) obstructive type of hypertrophic cardiomyopathy and transient LV (left ventricular) apical ballooning, a characteristic feature of Takotsubo cardiomyopathy (stress cardiomyopathy or apical ballooning syndrome or broken-heart syndrome) which was originally described in Japan in 1990 and it is most commonly seen in elderly post-menopausal women, characterized by ST segment elevation in anterior precordial leads with elevated cardiac enzymes and may be due to excess catecholamines (diffuse microvascular spasm or dysfunction, resulting myocardial stunning or direct myocardial toxicity), coronary vasospasm and triggered by emotional or physical stress. It may presents with sudden onset of chest discomfort, shortness of breath and reversible within weeks to months. A criteria formulated by Mayo clinic as regional hypokineses with ECG evidence of ST-segment elevation in the absence of obstructive coronary artery disease, pheochromocytoma or myocarditis to diagnose this condition. During the event, the shape of LV cavity resembles the fishing pot of Japanese (Tako-tsubo) to trap octopuses and so it is called as Takotsubo cardiomyopathy. The typical form of this cardiomyopathy is apical type, characterized by systolic apical ballooning with hypokineses of apical and mid segments and hyperkineses of basal walls. The atypical variants such as mid-ventricular type (hypokineses restricted to mid-ventricle with sparing of apex), basal type (hypokineses of base with sparing of mid-ventricle and apex), focal type (dysfunction of a segment, usually anterolateral) and global type (global hypokineses) were also described.

### 3.4. ECG Features

The most common ECG findings are negative T waves in the precordial leads, found in 93% of patients (a depth >10 mm in 47%) and a documented left ventricular hypertro-
phy on imaging is seen in 65% of patients with HCM. Giant T wave negativity (defined as depth or voltage >1 mV or 1.2 mV in any of the leads) in the left precordial leads is the hallmark feature of apical HCM as a result of balance of electrical forces emanating from the left versus the right ventricle [35] due to abnormal repolarization of hypertrophied apical musculature. Yamaguchi et al. published a report in 1979 determining that apical hypertrophy is the only specific hypertrophic pattern that shows characteristic ECG abnormalities, i.e., giant negative T waves and high QRS voltage in the left precordial leads. Giant negative T waves are characteristic of hypertrophic cardiomyopathy with predominant apical thickening, especially in patients from the Pacific Rim, called as Yamaguchi syndrome [36].

Altered repolarization changes of ventricular myocardium may produce ST segment/T wave changes in 70% of cases of left ventricular hypertrophy. There is significant variability on manifestation of strain pattern as minimally inverted or >5 mm depth, asymmetric T waves with rapid return to baseline and terminal positivity, depression of the J point, T wave inversion in lead V₇ > 3 mm and greater in V₇ than V₄ [37] and high QRS voltage (R in V₅ > 26 mm, S in V₁ + R in V₅ ≥ 35 mm - positive Sokolow-Lyon index as a sign of LVH).

In apical HCM, larger degree of T wave inversions are seen and it’s depth does not correlate with severity of apical hypertrophy [38]. Presence of giant T wave inversions in Japanese HCM patients has been identified as a predictor of favourable outcome and it is more common in sporadic cases of apical HCM. Reports from outside Asia would suggest, however, that apical hypertrophy is uncommonly accompanied by marked T wave inversion and associated with adverse outcome in some patients. This heterogeneous morphologic expression even in first degree relatives is due to great dissimilarity in the pattern of left ventricular wall thickening [39]. In some non-Japanese studies, T wave negativity was often less pronounced and not necessarily localized to the precordial leads [40].

Abnormal ECG changes and no echocardiographic evidence of hypertrophic cardiomyopathy is seen in some patients due to abnormal gene in first degree relatives and called as carrier or preclinical state of hypertrophic cardiomyopathy. Incidence of deep T inversion may diminish considerably with advancing age. Myocardial infarction with a secondary apical LV aneurysm may occur in 10% of cases, that might determine the disappearance of the giant T waves in apical HCM and the “spade” shaped LV configuration may become “bottle-gourd” shaped. In normal variant of hypertrophic cardiomyopathy (HCM), the LV (left ventricular) cavity is usually “banana” shaped. However, in some cases, non-giant T waves may become a giant one on later life. The T wave changes associated with ischemia are usually narrow and symmetric (deep T wave inversion) and “CVA (cerebrovascular accident)-T wave pattern” is associated with marked QT prolongation especially in subarachnoid hemorrhage.

3.5. Transthoracic Echocardiography

The preferred initial imaging test is Transthoracic Echocardiography and it is the most frequently utilized diagnostic modality [41]. Hypertrophy (apical or elsewhere) is de-
fined as a compacted myocardial wall thickness in diastole greater than or equal to 14 mm and it is 15 mm as a threshold in conventional echocardiographic measurements, which was performed perpendicular to the axis of the wall and at the point of maximal thickness in short axis view at apical level as shown in Figure 9. In apical hypertrophic cardiomyopathy, the asymmetric hypertrophy of the left ventricle is predominantly confined to LV apex as shown in Figures 3-10, with a maximal apical thickness ≥15 mm and a ratio of maximal apical to posterior wall thickness ≥1.5 based on two-dimensional echocardiography is a diagnostic criterion of apical HCM [42]. Figure 9 shows the maximal apical thickness of 37.5 mm and a ratio of maximal apical to posterior wall thickness (10.2 mm) 3.7:1 (>1.5:1) in short axis view, satisfying the diagnosis of apical HCM in this patient.

Relative hypertrophy was defined as the absence of hypertrophy (wall thickness <14 mm) but with the apical wall thickness greater than the basal thickness (apex:base ratio (ABR) wall thickness >1). An apical-to-basal LV wall thickness ratio of 1.3 - 1.5 is diagnostic of apical HCM [43] and in this patient, it is 2:1 in parasternal long axis view as shown in Figure 8.

Apical HCM is characterized as concentric, circumferential hypertrophy of the entire apex due to apical left ventricular thickening of the anterior and posterior walls, resulting in a spade-like morphology of the left ventricular cavity during end diastole in LV long axis view of MRI and RAO projection of angiography. A subtype of apical HCM in which the distribution of hypertrophied myocardium proved to be confined to the apical lateral wall (AAH-asymmetric apical hypertrophy) and cannot be evaluated in long axis MRI or RAO projection in angiography and this subtype was called as non-spade apical HCM in short axis images of MRI [44]. A recent long-term study showed that patients with non-spade apical HCM may develop the typical spade-like configuration after some years [45]. Thus, the non-spade type is possibly an early stage of apical HCM and giant negative T waves are common in both types.

A criteria was formulated to diagnose apical HCM in a distinct phenotype [46] not captured by existing disease classification. This phenotype comprises

1) Two essential criteria
   - Deep ECG T wave inversion
   - Relative apical hypertrophy (ABR > 1)
2) Four minor or supportive criteria
   - Myocardial scarring by LGE CMR (late gadolinium enhancement cardiac magnetic resonance)
   - Presence of apical aneurysm or microaneurysm
   - Left atrial dilatation
   - Apical cavity obliteration >20 mm

The presence of two or more of four minor criteria is the supportive evidence of apical HCM. The differential features of Japanese and non-Japanese type of apical HCM is shown in the Table 1 given below.

In transthoracic echocardiography, the lateral wall hypertrophy is more than the
Table 1. Showing the differential features of Japanese and Non-Japanese type of HCM (hypertrophic cardiomyopathy).

| Japanese Type | Non-Japanese Type |
|---------------|-------------------|
| Mostly seen in males | Elderly female in Asians and younger in West |
| “Pure” form is predominant | “Mixed” form is predominant |
| Asymptomatic and benign | Complications may occur |
| T-wave negativity is more pronounced | Less pronounced |
| Increased hypertrophy confined to apex | Segmental distribution may occur. Basal septal hypertrophy producing sub aortic obstruction in 50%, mid-septal hypertrophy with mid-cavity obstruction in 25%, apical septal hypertrophy with apical obliteration in 25% of cases. Occasionally, papillary muscle hypertrophy alone may be seen |
| Apical wall thickness lesser degree | More than Japanese in Americans |

Apical HCM is generally associated with good prognosis in both Asian and Caucasian population and a long-term mortality is 0.1% per year. The approach to management of apical HCM depends on symptoms and risk of sudden cardiac death (SCD). There is lesser incidence of sudden cardiac death in apical variant, compared to patients with normal variant HCM.

In asymptomatic patients, no specific therapy has been outlined, but counseling is certainly recommended for symptomatic monitoring to notice any syncope or presyncopeal events on follow up. The medical regimen in symptomatic patients primarily consists of beta-blockers, which have been shown to decrease symptoms as well as apical septum and producing a classical “ace of spades sign” in apical four chamber [47] views as shown in Figure 3 and Figure 4. In apical long axis view as shown in Figure 7 and parasternal long axis view as in Figure 8, it is seen as concentric LVH pattern of apex with a spade shaped LV cavity and thus, resulting a heterogeneous appearance in echocardiographic images of various views. Interestingly, spade shaped configuration of LV cavity is visualized in all views in both systolic and diastolic images as assessed by the movement of mitral valve. Kitaoka et al., demonstrated that the wall thickness at the apex was greater in Americans (23 ± 4 mm) than in the Japanese patients (18 ± 2 mm). Duygu et al. showed that mean maximum apical thickness was 18 mm in a review of 17 patients with apical HCM. Figure 3 shows a maximum apical thickness 55.3 mm in apical 4 chamber view of this patient in end-diastolic image. The anterior mitral leaflet is long and elongated and mildly regurgitant as shown in Figure 5 due to trauma as a result of contact with septum as shown in Figures 4.

Lateral wall thickening was smooth, homogeneous, non-trabeculated in apical HCM and thus differentiated from non-compact LV (left ventricular) cardiomyopathy and apical HCM may mimic as apical LV thrombus which may be associated with wall motion abnormality. It can also mimic LV EMF (Endomyocardial fibrosis), but EMF is characterized with firm, rolled edges and a rugose surface over the endocardium.

3.6. Treatment

Apical HCM is generally associated with good prognosis in both Asian and Caucasian population and a long-term mortality is 0.1% per year. The approach to management of apical HCM depends on symptoms and risk of sudden cardiac death (SCD). There is lesser incidence of sudden cardiac death in apical variant, compared to patients with normal variant HCM.
The major clinical features associated with increased risk of SCD in HCM patients are non-sustained ventricular tachycardia, maximum LV wall thickness of ≥3 cm, family history of SCD at younger age, unexplained syncope, abnormal BP response during exercise and increased left atrial diameter (>36 mm) as a potential SCD risk modifier. Current guidelines do not recommend ICD (implantable cardioverter defibrillator) implantation as a primary prevention for SCD in apical HCM and these clinical features should be used to assess the prognosis on follow up.

In apical HCM complicated with apical aneurysm formation, the size and symptoms of the aneurysm were ameliorated by sub selective coronary angiography and alcohol injection into the small vessels supplying a limited segment of mid-LV obstructive muscle. Others have reported the use of surgical apical myectomy in similar circumstances [49].

An increased apical thickness of 55.3 mm indicate massive hypertrophy (>50 mm [50]) and LA dilatation (51.2 mm) predict this patient as a high risk category and so close monitoring is recommended in cardiologic clinic.

3.7. Screening of Population

3.7.1. Transthoracic Echocardiographic Screening

Echocardiographic screening of population was done in those individuals having ECG changes of LVH (left ventricular hypertrophy). A mixed form of apical HCM (with involvement of IVS) was found in a 65-year-old asymptomatic hypertensive female as shown in Figure 11, Figure 12 and a mid-cavity form of hypertrophic cardiomyopathy as shown in Figures 13 was detected in a 60-year-old asymptomatic normotensive male, having a similar ECG features of LVH in both cases. A mid-cavity type of HCM is also found in a 65-year-old asymptomatic normotensive female with a normal ECG pattern as shown in Figure 14 and Figure 15. Occasionally, papillary muscle hypertrophy alone may be seen as shown in Figure 16. An isolated basal hypertrophy of the interventricular septum in a 61-year-old, hypertensive male with normal ECG is detected by transthoracic echocardiography as shown in Figure 17.

A 60-year-old hypertensive woman having a mid-ventricular obstructive form of HCM with LVH (left ventricular hypertrophy) ECG pattern was observed and the gradient of obstruction may mimic apical dyskinesis with aneurysm as shown in Figures 18-22. The woman developed sudden onset of angina with an elevation in blood pressure up to 160/90 mmHg and cardiac enzymes (CK-MB fraction rises to 125 IU/L, normal < 25 IU/L). The ECG during the event showed LVH (left ventricular hypertrophy) pattern with VPCs (ventricular premature complexes) as shown in Figure 22 and no evidence of ST-segment elevation and wall motion abnormalities, but with a mild increase in gradient of obstruction in the LV apex as shown in Figure 20 and Figure 21 and it was controlled with antihypertensive medications such as calcium channel blockers and anti-anginal drugs with nitrates. Coronary angiography and selective alcohol injection into the small vessels supplying the mid-segment of left ventricle is preferred in this patient to prevent further episodes. This type of mid-ventricular obstructive
Figure 11. Showing the apical hypertrophic cardiomyopathy in a 65-year-old hypertensive female in apical four chamber view. Arrow mark indicates the lateral wall hypertrophy of LV (left ventricular) apex.

Figure 12. Apical HCM (hypertrophic cardiomyopathy) with involvement of IVS (interventricular septum) (“mixed type”)—“Bird’s Beak” LV (left ventricular) cavity in Apical long axis view in a 65-year-old hypertensive female. Left arrow indicates lateral wall hypertrophy of apical region and right arrow indicates septal hypertrophy of left ventricle.

form of HCM (hypertrophic cardiomyopathy) is frequently observed in elderly hypertensives with LVH (left ventricular) pattern of ECG changes and more prone to complications such as ischemia and infarction.

3.7.2. Genetic Screening
Genetic screening may be done in younger population if there is a family history of sudden death since hypertrophic cardiomyopathy (HCM) is the most common cause of
Figure 13. Hypertrophic cardiomyopathy (HCM)—“mid-cavity type” in parasternal long axis view in a 60-year-old normotensive male. Arrow mark indicates hypertrophy of mid-lateral wall of LV (left ventricle).

Figure 14. Mid-cavity HCM (hypertrophic cardiomyopathy) in a 65-year-old normotensive, asymptomatic female. Arrow mark indicates hypertrophy of mid-lateral wall of LV.

death in competitive athletes [51]. It is believed that hypertrophy in HCM is a compensatory phenomenon that occurs in response to mutant myosin protein peptide. By inhibiting transcription or translation of mRNA of this mutant allele, it is possible to induce regression of cardiac hypertrophy by abolishing the synthesis of this mutant peptide. The heart is capable to renew the proteins and the myosin, the longest lasting protein has a half-life of 5 days. There is an inherent potential for corrective resynthesis and inhibition of defective gene in adults and hypertrophy may revert it into normal within weeks to months. The ACE (angiotensin-converting enzyme) gene plays a remote role in the development of hypertrophy and ACE inhibitors may be given in those
Figure 15. Showing the normal ECG pattern in the 65-year-old asymptomatic normotensive female having the mid-ventricular HCM (hypertrophic cardiomyopathy. 1 mV = 5 mm standardization).

Figure 16. Showing the isolated papillary muscle hypertrophy in the left ventricle (arrow) in a 48-year-old normotensive female in Parasternal long axis view.
individuals without outflow tract obstruction. The exact molecular pathogenesis of HCM is to be determined and so the definite therapy based on gene manipulation remains speculative.

4. Conclusion

The initial echocardiographic evaluation was sufficient to diagnose the patients with apical HCM [52]. Typical ECG changes are the signs of left ventricular hypertrophy such as pronounced negative T waves (>1 mV) in the precordial leads and a positive
Figure 19. The Pulsed-Doppler imaging. Arrows show the dagger jet with late systolic peak and a minimal gradient of obstruction.

Figure 20. Apical left ventricular long axis view showing the gradient in LV apex mimicking apical aneurysm when the woman developed angina of sudden onset masquerading as “apical ballooning syndrome”. Upper arrow indicates the apical gradient and lower lateral arrow indicate mid-lateral wall hypertrophy.

Sokolow-Lyon index. The diagnosis of apical hypertrophic cardiomyopathy was primarily made from ECG changes and specific morphologic criteria as assessed non-invasively by Transthoracic echocardiography. The condition should be considered when encountering a 12-lead ECG showing giant T-wave inversion [53] and it was found that giant inverted T wave was associated with a severe form of apical HCM [54]. The echocardiographic findings may vary with the extension of hypertrophy and in apical HCM; the obliteration of apical cavity by the hypertrophy resulting in a spade-shaped LV (left
Figure 21. Pulsed-Doppler imaging shows the dagger jet (right arrow) of mild increase in gradient with a presystolic forward flow (left arrow) due to atrial systole during the event of angina.

Figure 22. ECG showing LVH (left ventricular hypertrophy pattern-SV_1 + RV_5 = 46 mm, a positive Sokolow-Lyon index and a strain pattern of 2 mm J point depression with 4 mm T-wave inversion) and no ST-segment elevation during the angina episode, VPCs (ventricular premature complexes seen during the event (1 mV = 5 mm standardization).
ventricular) cavity in all views of echocardiographic imaging was recognized and it remained asymptomatic in this 54-year-old male at this belt of Thoothu-kudi in India. The mixed and mid-cavity forms were detected in this region with a relatively benign and late onset disease in adult life. Mid-ventricular obstructive form with an anginal episode was also reported in an elderly hypertensive female at the age of 60 years old.

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Hiatal Hernia by Rolling Revealed by Respiratory Symptoms: A Rare Condition and Atypical Presentation

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Abstract

Hiatal hernia by rolling is a rare pathology estimated at 5% of all diaphragmatic hernias. It is usually asymptomatic. Sometimes, it can be accompanied by different symptoms, such as post prandial bloating, dysphagia, regurgitation, retrosternal pain, even respiratory symptoms. The aim of this article is to show that a hiatus hernia by rolling can cause a misleading clinical picture that may be taken wrongly for a heart or respiratory disease. We report the case of an 80-year-old patient whose presentation was suggestive of an acute heart condition, but the scan confirmed a rolling diaphragmatic hernia. Because of the rarity and the atypical presentation of this case, we have found it desirable to bring this new observation.

Keywords

Hiatal Hernia, Diaphragmatic Hernia, Rolling Hernia, Retrosternal Pain, Respiratory Symptoms

1. Introduction

The diaphragmatic hernia is defined by permanent or intermittent passage of the abdominal viscera in the chest by an abnormal diaphragmatic hole. This diaphragmatic hole may be abnormally far (Larrey slot, Bochdalek hernia) or abnormally enlarged (hiatal hernia). Hiatal hernias are due to movement of the gastroesophageal junction into the thorax. There are 3 types of hiatal hernia:

• The most frequent (85% of cases) are sliding hernias.
• Hernias by rolling (5% of cases), in which the cardia is intra-abdominal and fundus in intrathoracic position.
• Mixed hernias (10% of cases).
The rotating hiatal hernias do not cause gastroesophageal reflux or esophagitis.

The symptoms are related to the volume of the hernia, which can manifest as chest pain, angina, respiratory symptom, sometimes chronic anemia. Complications are represented by gastrointestinal bleeding related to ulceration of the neck hernia (see necrosis and perforation of the collar, mediastinitis, and gastric volvulus). Because of the risk of complications, surgical indication arises when the diagnosis of hiatal hernia is made.

Given the rarity of this disease, we report the case of a patient admitted to our department for respiratory symptoms revealing hiatal hernia by rotation.

2. Case Report

Mrs. L A, 80 years old, with a history of dyslipidemia and type II diabetes put on oral anti-diabetic and placed under symptomatic treatment for gastroesophageal reflux 10 years ago. This patient was presented to the emergency room in an array of acute retrosternal chest pain, chronic dyspnea since 3 months, associated with a dry cough without hemoptysis nor dysphagia, nor hydatidoptysis or other respiratory or extra-respiratory signs associated. Clinical examination showed an obese patient with a BMI 26 kg/m², tachypnea at 20 cycles/minute, cyanosis of the lips and extremities, 87% SaO₂ in the ambient air, without rattling in auscultation. The cardiac examination has not objectified abnormally outside except a tachycardia of 110 beats/minute. Ischemic heart disease was mentioned in the first place (according to age and cardiovascular risk factors presented by the patient). Troponin, ECG and echocardiography had not objectified signs for cardiac ischemia. Chest radiograph Figure 1 suggested the existence of some retrocardiac clarities, with horizontal air-fluid levels suggesting a diagnosis of a diaphragmatic hernia. After stabilization of the patient, a chest CT scan was requested showing a large defect of the left diaphragm with ascension through the esophageal hiatus of the large gastric tuberosity, of the 2/3 of the stomach and perigastric fat on retrocardiac region of the posteroinferior mediastinum. This CT aspect is suggestive of a hiatal hernia by rolling. Surgical treatment was proposed to the patient, but given the age and the medical history of the patient, surgery seemed to bring more risk than benefit. Therefore, we opted to keep it under strict surveillance to detect possible complications. A rhythm of regular monitoring has been proposed to the patient every 3 months. Lifestyle and dietary rules and anti reflux treatment have been proposed for the patient. The patient evolved well, no complications did not occur after 18 months.

3. Discussion

Diaphragmatic hernia rotating or para esophageal hernia or hiatal firewall represents 5% of hiatal hernias. The hernia orifice has often variable diameter [1]. The omentum is uniquely the only herniated structure in almost all cases, the bag contains all or a part of the stomach, as the case of our patient. In fact, the content of the hernia sac depends on the volume of the hernia. In small hernia, only the gastric fundus is intrathoracic. In large hernias, all the stomach switches from the left to the right around the small curvature into the thorax, realizing volvulus organoaxial [2]. In the voluminous hernias
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Figure 1. Retrocardiac clarities, with horizontal air-fluid levels.

Figure 2. Large defect of the left diaphragm with ascension through the esophageal hiatus of the large gastric tuberosity, of the 2/3 of the stomach and perigastric fat on retrocardiac region of the posteroinferior mediastinum.

omentum, transverse colon, spleen or even hail can engage with stomach. Hernia occupies the lower posterior mediastinum behind the heart and in front of the esophagus. Usually the cardia remains in place with an intact ligament and a normal configuration of the His angle [3]. The gastro esophageal reflux is the most common symptom [4]. It is frequently observed in women 70 to 80 years as the case of our patient. Thoracic manifestations are the prerogative of young subjects, their appeared or worsened in postprandial and are frequently accompanied by intermittent symptoms of oesophageal or pyloric obstruction (dysphagia, vomiting) and often iron deficiency anemia with or without signs of digestive hemorrhage [5]. The respiratory symptoms can be present regardless of the size of the hernia, such as dyspnoea and chest pain that gets worse especially in postprandial, this was the case of our patient. The diagnosis is suggested by the presence of one or much retrocardiac clarity containing a hydroaer level lateralized to the right when it is voluminous. This X-ray image increases in volume after in-
gestion of a soft drink. It is usually accompanied by a disappearance of the gastric air pocket. The latter may still be in place, chest radio while revealing two clarities with hydro aeric level, one above, the other under diaphragmatic indicating a cascade stomach. When the hernia is large, it can be observed in addition to gastric hernia image, evocative clarities of colic hernia and/or opacity of a herniated spleen. So, any clarity of the postero-inferior mediastinum with hydro aeric level must lead to gastroesophageal transit that will affirm the diagnosis and eliminate other conditions sometimes evocated (bronchogenic cyst, abscess of the mediastinum…) [6]. In our patient, the diagnosis was suggested on chest radiograph, which showed the retro-cardiac clarities, which can be relevant to digestive clarities. CT allows affirming the epiploic hernia (a fat mass having an omental aspect occupying the postero-inferior mediastinum extending through the esophageal hiatus with intra abdominal omentum) [7]. In our patient, CT showed a climb through the oesophageal hiatus of the large gastric tuberosity, of 2/3 of the stomach and the perigastric fat in retrocardiac region at posteroinferior mediastinum. The complications are the hemorrhages by ulceration or necrosis of the collet with perforation and mediastinitis, or acute stomach volvulus. Conversely to hiatal hernias by sliding, the risk of occurrence of acute complications of hernia by rolling is for all authors is a formal indication for surgery. The surgical treatment of hiatal hernia by rolling requires resection of the hernia sac, suture of the pillars of the esophagus and the making of a Nissen or toupet fundoplication. Moreover, the advent of laparoscopy 10 years ago transformed the surgical treatment of hiatal hernias. It is now possible to reproduce in laparoscopy the same techniques as those used in conventional surgery by laparotomy. Our patient given her age and her major clinical history, was refused from surgery, but close monitoring has been proposed for her to watch for possible complications.

4. Conclusion

The hiatal hernia by rolling remains fairly a rare disease. Diagnosis can be made by simple chest X-ray. We have to think about in case of gastroesophageal reflux with respiratory symptoms worsening especially in post-prandial. The treatment is surgical, and can avoid complications. But we must always consider the benefit risk before starting a surgical cure and prefer laparoscopy if it is available.

Conflicts of Interest

Authors declare they have no conflicts of interest.

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Pseudomonas aeruginosa Community Acquired Pneumonia with Septicemia in a Previously Healthy Woman

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Abstract
A previously healthy 53-year-old woman was urgently hospitalized due to septic shock. She was diagnosed with bacterial pneumonia based on chest radiograph and computed tomography findings of right upper lobe consolidation. Sputum Gram stain at the time of admission showed gram-negative rods with phagocytosis. Intravenous meropenem was immediately initiated as empiric antibacterial therapy. Bacterial culture specimens from sputum and blood were positive for Pseudomonas aeruginosa. Following appropriate antibiotic therapies, the patient recovered from a shock state and gradually became well. There has been no evidence of recurrence at 6 months after discharge. P. aeruginosa community acquired pneumonia with septicemia is rapidly progressive and often fatal. The choice of initial empiric antibiotic treatment that is active against P. aeruginosa is critical in improving outcome.

Keywords
Pseudomonas aeruginosa, Community Acquired Pneumonia, Septicemia

1. Introduction
P. aeruginosa is a frequent pathogen causing hospital-acquired pneumonia and healthcare-associated pneumonia, but is a rare cause of Community Acquired Pneumonia (CAP) in an immune competent host [1]. However, P. aeruginosa CAP is prone to develop septicemia, is often rapidly progressive, and may be fatal [2]. Appropriate initial empiric antibiotic treatment is crucial in improving outcomes. We herein report a case of P. aeruginosa CAP with septicemia in a previously healthy woman who was success-
fully treated by appropriate antibiotic treatment.

2. Case Report

A 53-year-old woman was brought by ambulance to the emergency department of our hospital due to fever and right shoulder pain for a few hours. She had no pertinent past medical history, family history, or recent history of overseas travel. However, she had been a smoker of 20 cigarettes per day for 30 years. On examination, her body temperature was 36.3°C, blood pressure was 90/40 mmHg, pulse rate was 100 beats/min, respiratory rate was 22 breaths/min, and oxygen saturation was 97% at an oxygen support of 10 L/min by face mask. Auscultation of the chest revealed coarse crackles on the right upper lobe. Initial laboratory tests revealed white blood cell (WBC) count of 6740 cells/µL, and elevations of C-reactive protein (CRP; 2.94 mg/dl), procalcitonin (PCT; 37.73 ng/ml). And arterial blood gas revealed lactic acidosis (pH; 7.363, base excess; -0.5 mmol/L, lactate; 22.6 mg/dl). Chest radiograph showed right upper lobe consolidation and computed tomography (CT) scan showed air space consolidation in the posterior segment of the right upper lobe (Figure 1).

Figure 2 summarizes her clinical course in the hospital. She was diagnosed as septic shock due to lobar pneumonia. Treatment with bolus infusion of lactated Ringer’s solution and norepinephrine (0.3 µg/kg/min) was started at the emergency room. She did not require emergency intubation, but she was transferred to the intensive care unit. Sputum Gramstain on admission showed Gram-negative rods with phagocytosis. At the emergency room, empiric antibiotic treatment with intravenous meropenem (MEPM) at 3 g/day was immediately started. The results of blood and sputum cultures were positive for P. aeruginosa. An acid-fast stain of the sputum was negative. After the 4th day of hospitalization, cardiovascular stabilization was achieved and norepinephrine drip was discontinued. Subsequently, her clinical symptoms and the level of CRP improved and her body temperature normalized. On the 7th day of hospitalization, antibiotic treatment was de-escalated to intravenous ciprofloxacin (CPFX) at 0.6 g/day.

A repeat chest CT scan on the 11th hospital day showed reduction of air space consolidation, but anew cavity formation was observed (Figure 3). Although not detected in any of the specimens, co-infection with an anaerobe was suspected and intravenous clindamycin (CLDM) at 2.4 g/day was added and continued for 10 days. Follow-up chest radiograph and CT scan on the 24th hospital day revealed further improvement of the consolidation and resolution of the cavity (Figure 4). Intravenous CPFX was continued until discharge on the 28th hospital day and oral CPFX was prescribed for one more week after discharge. Six months after discharge, there were no clinical symptoms of recurrence and follow-up chest radiograph revealed resolution of the infiltrates (Figure 5).

3. Discussion

P. aeruginosa is a frequent pathogen causing hospital-acquired pneumonia and healthcare-associated pneumonia [1] [3]. However, CAP secondary to P. aeruginosa is un-
common, with an incidence of only 0.6%, as shown in Sibila’s cohort study on 62,689 pneumonia patients aged ≥65 years old [4]. It is often rapidly progressive and may be fatal [4], with a 30-day mortality rate of 17.4% [4].

There have been only 12 case reports found in PubMed on *P. aeruginosa* CAP with septicemia in patients without comorbidities (Table 1) [3] [5]-[13]. The clinical characteristics of these cases were pleuritic chest pain and right upper lobe predilection; in addition, mortality rate was high. The outcome was shown to be dependent on initiation of appropriate antibiotic therapy against *P. aeruginosa* within the first 48 hours of

![Figure 1](image1.png)

**Figure 1.** Chest radiograph and computed tomography (CT) scan of the chest. (a) Chest radiograph obtained at the emergency room shows right upper lobe consolidation (red arrow); (b) CT scan of the chest showed air space consolidation in the posterior segment of the right upper lobe (red arrow).

![Figure 2](image2.png)

**Figure 2.** Course of treatment. The level of C-reactive protein (CRP) improved and her body temperature normalized. MEPM: meropenem, CPFX: ciprofloxacin, CLDM: clindamycin, NA: norepinephrine.
admission [14], therefore, timely appropriate empiric antibiotic treatment is crucial [14]. For this patient, we chose MEPM because the main target of treatment was sepsisemia due to lobar pneumonia and based on the presence of gram-negative rods on sputum. Gram stain is the most commonly performed microbiology test that can immediately identify the cause of a bacterial infection. Henderson et al. [10] suggest that P. aeruginosa CAP should be suspected in any patient who has gram-negative bacilli seen on sputum sample. Gram-negative bacteria include P. aeruginosa, which is reported to be one of the risk factors for death in CAP patients [15].

P. aeruginosa CAP may sometimes progressive to cavitary pneumonia [2] [3] or lung abscess; for the latter, anaerobes are the major pathogens involved and may co-infect

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**Figure 3.** Follow-up chest radiograph and CT scan of the chest. (a) Follow-up chest radiograph on the 15th day of hospitalization shows improvement of the right upper lobe consolidation (red arrow); (b) Follow-up CT scan of the chest on the 15th day of hospitalization showed improvement of air space consolidation, but a new cavity on the right upper lobe (red arrow).

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**Figure 4.** Follow-up chest radiograph and CT scan of the chest. (a) Follow-up chest radiograph on the 24th day of hospitalization shows further improvement of the right upper lobe consolidation (red arrow); (b) Follow-up CT scan of the chest on the 24th day of hospitalization showed stability of the air space consolidation, but resolution of the cavitation (red arrow).
Figure 5. Follow-up chest radiograph. Follow-up chest radiograph 6 months after discharge revealed almost complete resolution of the infiltrates.

Table 1. Laboratory findings in a previously healthy woman who developed *Pseudomonas aeruginosa* pneumonia. CRP: C-reactive protein, PCT: procalcitonin.

| Reference | Age | Sex | Symptom               | Infected location | Treatment                                      | Outcome   |
|-----------|-----|-----|-----------------------|-------------------|------------------------------------------------|-----------|
| Fujii [3] | 29  | Male| Shoulder pain         | Right upper lobe  | PIPC                                           | Recovered |
| Quirk [5] | 40  | Female| Unknown              | Unknown           | PC, EM (2d); PIPC, GM, PC, CFX, CAZ         | Recovered |
| Harris [6]| 39  | Male| Chest pain            | Left lower lobe   | ABPC, GM, CLDM, CBPC, GM                      | Recovered |
| Govan [7]| 49  | Male| Unknown               | Unknown           | PC, ABPC, MC                                   | Recovered |
| Hoogwrf [8]| 64 | Male| Lost consciousness    | Right upper lobe  | ABPC, GM (3d); CBPC, GM                       | Recovered |
| Fishman [9]| 29 | Male| Unknown               | Unknown           | GM, EM, CET (3d); CBPC, GM                    | Recovered |
| Henderson [10]| 52 | Male| Cough                 | Left lower and right upper lobe | GM, ABPC, EM, MFIPC | Death     |
| Henderson [10]| 27 | Female| Cough               | Right upper lobe  | PC, GM, EM                                     | Death     |
| Ishihara [11]| 48 | Female| Back pain           | Right upper lobe  | PIPC                                           | Recovered |
| Okamoto [12]| 39 | Female| Chest pain           | Right upper lobe  | CTRX (12h); CPFX, MEPM (10d); TAZ/PIPC, AMK  | Recovered |
| Takakura [13]| 52 | Male| Chest and back pain  | Right upper lobe  | PAPM/BP, CPFX                                  | Death     |
| Takakura [13]| 73 | Male| Chest pain           | Right upper lobe  | TAZ/PIPC, LVFX                                  | Death     |

In this case, although anaerobes were not detected in blood and sputum, cavitary pneumonia was seen on follow-up CT scan. Co-infection with anaerobes might have been associated with this disease. In such cases, combination therapy with antibiotics against both *P. aeruginosa* and anaerobes might contribute to resolution of the cavitary pneumonia [3].
We experienced a rare case of *P. aeruginosa* CAP with septicemia in a previously healthy woman. The clinical course might be rapidly progressive and is often fatal. The choice of an initial empiric antibiotic treatment that is active against *P. aeruginosa* is critical in improving outcome.

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Prolotherapy Injections for Diastasis Recti: A Case Report

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Abstract

This case report detailed the history and treatment of a female postpartum patient with diastasis recti. Treatment for this patient included the use of prolotherapy, an injection-based therapy using dextrose as the active compound. The solution used during the course of therapy was composed of 6 mL of 50% dextrose, 3 mL of 1% lidocaine, and 1 mL of methylcobalamin (1000 mcg/mL). Injections were administered every 2 weeks for a total of 7 prolotherapy sessions. Following the series of prolotherapy injections, there was a marked closure observed in the diastasis, decreasing from 2.7 cm to 0.5 cm. The patient did not report any side effects, and no complications were observed or recorded. This appears to be the first case report documenting an improvement in abdominal diastasis recti following a course of prolotherapy. Within the limitations of the study design, further research is recommended to evaluate prolotherapy for diastasis recti in postpartum patients.

Keywords

Injections, Rectus Abdominus, Prolotherapy, Hernia

1. Introduction

Prolotherapy is a complementary and alternative medical therapy that has been used for over 100 years [1]. It is commonly used for chronic musculoskeletal pain conditions [2], such as knee osteoarthritis [1], chondromalacia patella [3], coccygodynia [4], chronic low back pain [5], lateral epicondylitis [6], Achilles' tendinosis [7], and plantar fasciitis [8]. It is thought to work by introducing an irritant solution into injured tissue to stimulate the release of tissue growth factors [1]. It may also serve as a central pain modulator [1]. Different types of solutions may be selected by physicians' performing prolotherapy. The more common of these include dextrose [9], morrhuate sodium [10], and phenol-glycerine-glucose (P2G) [11].
Diastasis recti is a term used to describe a separation of the left and right sides of the abdominal wall of at least 2.7 centimeters [12]. It is a common result of childbearing caused by the repetitive stretching of the abdominal wall, which can result in midline or paraumbilical hernia [13]. Although it tends to be self-limiting postpartum, surgical intervention is often used for cosmetic purposes, as well as for hernia repair [14]. Exercise options are rare, but have been reported [15]. Even in cases where the diastasis recti improves, but doesn’t resolve, after 6 months post-partum, the abdominal muscle strength does not return to normal [16]. Our case report details the management plan of a patient diagnosed with diastasis recti treated with prolotherapy. This report appears to be the first to document the use of prolotherapy to treat this common disorder. Written informed consent was obtained from this patient to use her non-identifying information.

2. Case Report

2.1. Patient History and Exam

A 25-year-old female Caucasian patient presented to a private integrative medicine office with a history of abdominal diastasis recti. This developed following the birth of her second child 8 months prior to initial presentation. Patient had 2 pregnancies, both of which were full term, vaginal deliveries. The second of her two children weighed 9 lbs, 5 oz at birth, while the first weighed 8 lbs, 2 oz. At the time of initial presentation, the patient was 5’ 3’ tall, and 120 lbs. She had a past medical history of diastasis recti, alopecia, irritable bowel syndrome, Hashimoto’s thyroiditis, and headaches during menstruation. Laboratory testing demonstrated positive serum levels of IgM (21 U/ml) and IgA (23 U/ml) candida antibodies, thyroglobulin antibodies (1.5 IU/mL), 25(OH) vitamin D3 level of 25 ng/mL, as well as decreased urinary levels of dehydroepiandrosterone (DHEA) (14.3 nmol/dL), low testosterone, low estrone (3.2 mcg/g Cr), and low estradiol (1.3 mcg/g Cr).

Upon inspection of the patient’s abdomen, a separation of the abdominal wall was apparent just superior to the umbilicus, measuring 2.9 mm. The patient was told by another provider that this separation would self-resolve approximately 6 months post-partum, but 8 months later it was still evident. The patient’s desire was to correct this non-surgically. Figure 1 shows an illustration of this patient’s diastasis recti.

2.2. Treatment

After she was informed of the potential benefits, risks, and complications associated with prolotherapy, the patient affirmed her intent to proceed with the procedure. A total of 4 injections were administered on the first treatment around the palpable site of supra umbilical muscle division. Solution was comprised of a combination of 6 mL of 50% dextrose, 3 mL of 1% lidocaine, and 1 mL of methylcobalamin (1000 mcg/mL). Prolotherapy injections were administered every two weeks for a total of 7 prolotherapy sessions.

During the first 5 sessions, a total of 4 injections were used to administer the dextrose
solution. For the final two prolotherapy sessions this decreased to 2 injections. The patient did not report any complications or side effects throughout the duration of treatment, and tolerated the injections well. Figure 2 shows prolotherapy injection points for this patient.

Figure 1. Normal rectus abdominus (left); Diastasis recti (right). Illustration reprinted with permission from beyondfitmom.com

Figure 2. Prolotherapy injection points used in this case.
2.3. Outcome

Patient was re-evaluated at 14 weeks following the first prolotherapy session. Good approximation of the rectus abdominus was observed, with palpable closure measuring 0.5 cm, a decrease from the initial 2.9 cm.

3. Discussion

Although prolotherapy has been consistently used for chronic pain syndromes, due to its postulated mechanism of action, prolotherapy may be a novel way to treat diastasis recti when it is not self-limiting. This may be preferable to surgical options to repair any subsequent hernias or negative cosmetic impact.

It is important to also discuss the safety profile of prolotherapy. In a survey of approximately 500,000 patients treated for a variety of health conditions, they included 29 reports of a pneumothorax, 24 accounts allergic reactions, and 12 cases of hospitalization for an undisclosed reason [17]. Common side effects can include injection site redness and localized tenderness for 5-7 days post-injection [1]. This track record makes prolotherapy a desirable intervention when compared to more invasive alternatives.

Over the course of the treatment presented in this case report, the patient was also prescribed various nutraceuticals based upon her symptoms and laboratory findings, including DHEA 5 mg b.i.d., pregnenolone 50mg daily, and cholecalciferol 10,000 mg daily. While it is possible that these nutraceutical interventions may have helped to play a part in the observed improvement in her diastasis recti, this has never been previously reported to do so in the literature.

Because this is a case report, there are limitations associated with this study design. First, a lack of a control limits our ability to generalize or make assumptions on broader patient populations. Second, the apparent reduction in abdominal wall separation following prolotherapy could possibly be, however unlikely given the length of time this patient had the diagnosis, attributed to self-resolution over time as many of these cases do. Future studies on this topic could include testing multiple types of injection solutions to determine which work the best, as well as to compare against control patients to determine effectiveness compared to natural history.

4. Conclusion

In this case report, the treatment and the outcome of a patient with abdominal diastasis recti who received a therapeutic trial of dextrose prolotherapy were reported. Following this therapeutic trial, the patient’s abdominal separation closed from 2.9 cm to 0.5 cm. The patient will be followed to assess long-term benefits.

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