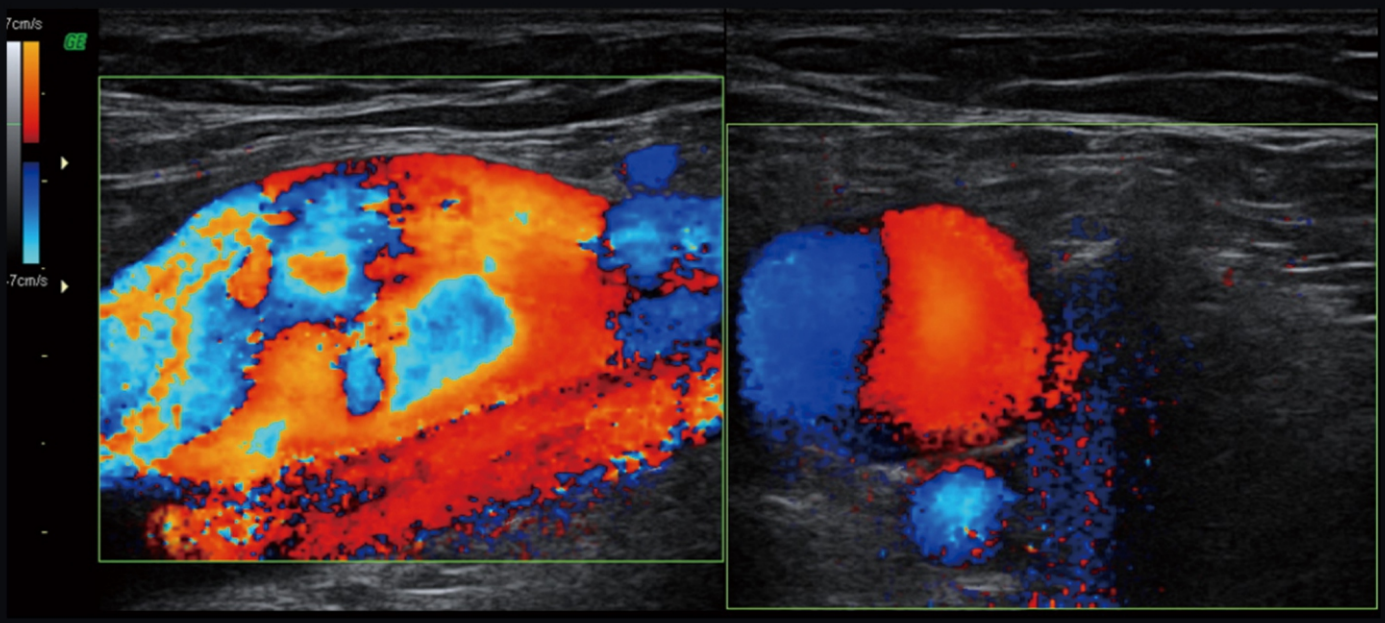
ISSN: 2325-7075 Volume 6, Number 1, January 2017

Scientific Research Publishing

Case Reports in Clinical Medicine



ISSN: 2325-7075

www.scirp.org/journal/crcm

Journal Editorial Board

ISSN Print: 2325-7075 ISSN Online: 2325-7083
http://www.scirp.org/journal/crcm

Editor-in-Chief

Editorial Board

Scientific Research Publishing

Case Reports in Clinical Medicine, 2017, 6, 1-30
http://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Table of Contents

Volume 6 Number 1 January 2017

The figure on the front cover is from the article published in Case Reports in Clinical Medicine, 2017, Vol. 6, No. 1, pp. 19-23 by Ahmad Rezaee Azandaryani, Pezhman Ghaderzadeh, Leili Ebrahimi Farsangi and Sayad Nasirzadeh.

Case Reports in Clinical Medicine (CRCM)
Journal Information

SUBSCRIPTIONS

The *Case Reports in Clinical Medicine* (Online at Scientific Research Publishing, www.SciRP.org) is published monthly by Scientific Research Publishing, Inc., USA.

Subscription rates:
Print: $59 per issue.
To subscribe, please contact Journals Subscriptions Department, E-mail: sub@scirp.org

SERVICES

Advertisements
Advertisement Sales Department, E-mail: service@scirp.org

Reprints (minimum quantity 100 copies)
Reprints Co-ordinator, Scientific Research Publishing, Inc., USA.
E-mail: sub@scirp.org

COPYRIGHT

PRODUCTION INFORMATION

For manuscripts that have been accepted for publication, please contact:
E-mail: crcm@scirp.org

Scientific Research Publishing

Case Reports in Clinical Medicine, 2017, 6, 1-18
http://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Coronary Sinus Atrial Septal Defect (Unroofed Coronary Sinus) with Total Anomalous Pulmonary Venous Connection—A Case Report

Ramachandran Muthiah

Thoothukudi Medical College Hospital, Thoothukudi, India
Email: cardioramachandran@yahoo.co.uk

How to cite this paper: Muthiah, R. (2017) Coronary Sinus Atrial Septal Defect (Unroofed Coronary Sinus) with Total Anomalous Pulmonary Venous Connection—A Case Report. *Case Reports in Clinical Medicine*, **6**, 1-18.
http://dx.doi.org/10.4236/crcm.2017.61001

Received: November 14, 2016
Accepted: January 10, 2017
Published: January 13, 2017

Abstract

Introduction: To present a rare occurrence of coronary sinus atrial septal defect (ASD) associated with total anomalous pulmonary venous connection (TAPVC). **Case Report:** A 16-year-old girl was diagnosed with features of an atrial septal defect (ASD) by transthoracic echocardiography and the absence of PLSVC (persistent left superior vena cava) connection to coronary sinus was confirmed by saline contrast echocardiography ("bubble study"). **Discussion:** The cause of the dilated coronary sinus was due to total anomalous pulmonary venous connection. **Conclusion:** Coronary sinus atrial septal defect (ASD) is a rare interatrial shunt that is commonly associated with a persistent left superior vena cava (PLSVC) and occurs in less than 1% of cases of ASDs. It is characterized by the absence of at least a portion of the common wall that separates the coronary sinus and left atrium. Isolated defects are associated with low rate of morbidity and mortality.

Keywords

Ccoronary Sinus ASD, Raghib Complex, Bubble Study,
Total Anomalous Pulmonary Venous Connection (TAPVC)

1. Introduction

Coronary sinus is usually not dilated on echocardiography. Anomalous venous drainage into the coronary sinus either directly or through a persistent left superior vena cava (PLSVC) is one of the etiologies for a dilated coronary sinus [1]. Mild dilatation occurs in patients with poor left ventricular systolic function, greater dilatation in persistent LSVC and huge dilatation (Giant coronary sinus) occurs in PLSVC associated with absence of a right superior vena cava. The conditions associated with dilated coronary sinus are shown in Table 1 given.

Table 1. Conditions associated with dilated coronary sinus.

Dilated coronary sinus (CS)
Persistent left SVC
Anomalous pulmonary venous drainage into The CS
Absence of the upper inferior vena cava , a Large left hemiazygos vein draining into the CS
Coronary arteriovenous fistula
Unroofing of the CS (septal defect between LA (left atrium) and coronary sinus)

Coronary sinus ASDs are uncommon and so this case had been reported.

Review of Literature

Mantini's *et al.* proposed four categories of CS anomalies as enlarged CS, absent CS, atresia of the right atrial CS ostium and hypoplasia of the CS [2]. The enlarged CS category has two subtypes—subtype A (without left-to-right shunt) and subtype B (with left-to-right shunt). Mantini postulated that a connection between the LA and CS may serve to drain the CS in RA (right atrium) ostial stenosis. Eliot, *et al.* [3] described a fibrous band that narrowed the right atrial ostium of the CS. Scheller *et al.* presented a case of CS with two outlets, one to the RA, and the other to the LA [4].

Unroofed coronary sinus was first described by Raghib and colleagues in 1965 with the findings of left SVC drains into the left atrium, an ASD (which lies in the postero-inferior angle of the atrial septum and above the postero-medial commissure of the mitral valve and separated from the mitral valve by a small amount of septal tissue—the defect is considered as a true defect of a specific tissue) and absent coronary sinus which are collectively termed as "Raghib complex or syndrome" [5]. In unroofed coronary sinus, there is a direct CS to LA communication through a wall defect and the CS is usually enlarged.

Total anomalous pulmonary venous connection (TAPVC), a very unusual malformation of the heart was published by Philosophical Transactions of the Royal Society in London in 1798 [6]. It represents 2% of all congenital heart defects and 100 cases of anomalous pulmonary venous connections were reported in 1942 [7].

2. Case Report

A 16-year old girl was referred for echocardiographic evaluation with a history of exertional dyspnea and recurrent episodes of respiratory infection from childhood. Her pulse rate was 88 bpm and blood pressure 110/70 mmHg. Physical examination revealed mild cyanosis and clubbing. Auscultation revealed grade 2/6 soft systolic murmur in the left second intercostal space with a wide,

fixed splitting of second heart sound. Ultrasound abdomen revealed normal liver and spleen. Blood chemistry revealed normal. ECG revealed right axis deviation and right bundle branch block (RBBB) pattern as shown in Figure 1. X-ray chest revealed increased pulmonary vascular markings with peripheral extension and a central prominence suggesting a left-to-right shunt at atrial level as shown in Figure 2. Transthoracic 2D echocardiography revealed an unroofed coronary sinus draining into the left atrium with anomalous pulmonary venous connection and an associated ostium secundum atrial septal defect (ASD) as shown in Figures 3-14. The girl was treated with antibiotics for the respiratory infection and advised early surgical correction.

3. Discussion

Coronary sinus is a systemic venous structure embryologically derived from the

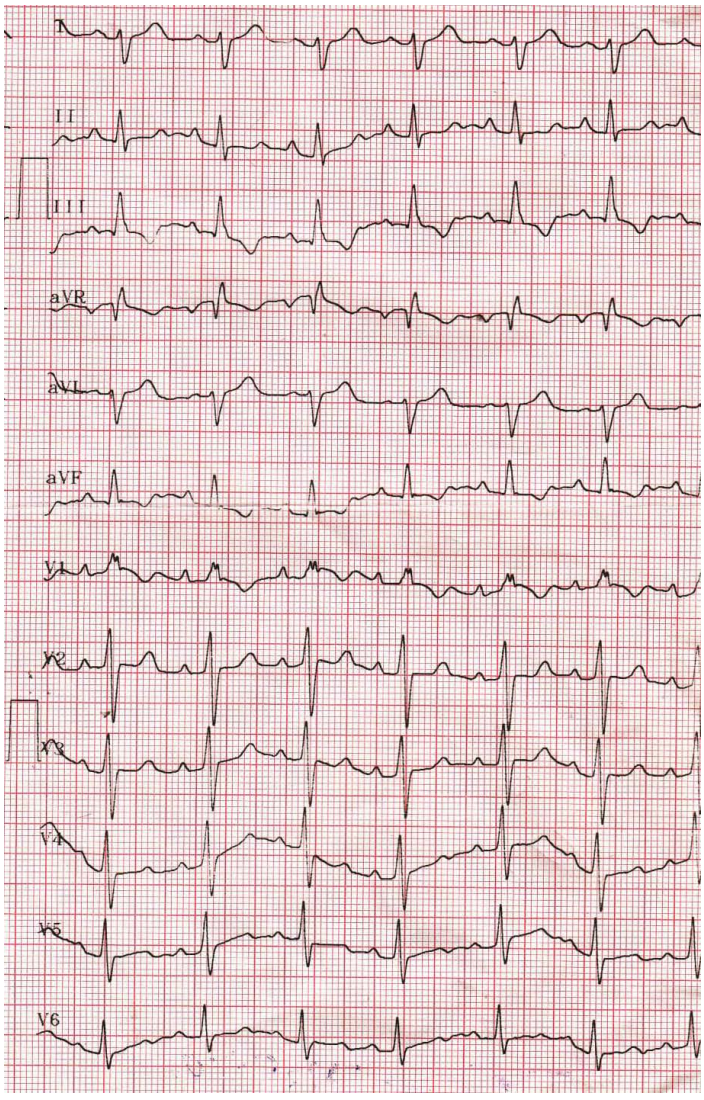


Figure 1. ECG revealed right axis deviation and right bundle branch block pattern in V_1 suggesting a RV volume overload pattern of left-to-right shunt at atrial level.

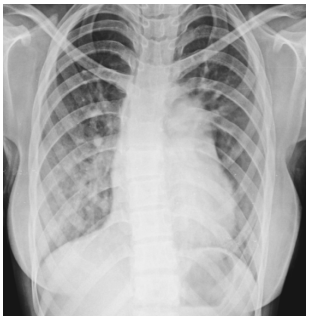


Figure 2. X-ray chest PA (posteroanterior view) revealed increased pulmonary vascular markings with a central prominence and peripheral extension suggesting a left-to-right shunt at atrial level.

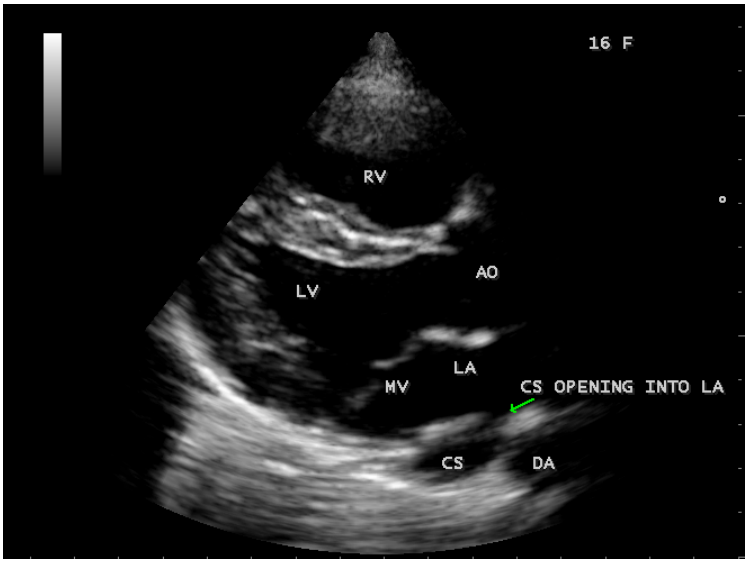


Figure 3. Parasternal long axis view showing the opening of coronary sinus into left atrium.

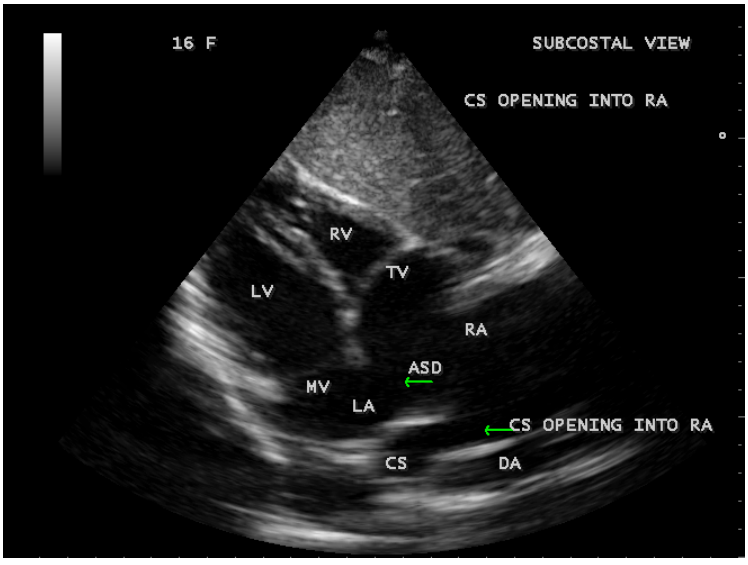


Figure 4. Subcostal view showing the opening of coronary sinus into the right atrium and ostium secundum atrial septal defect (ASD).

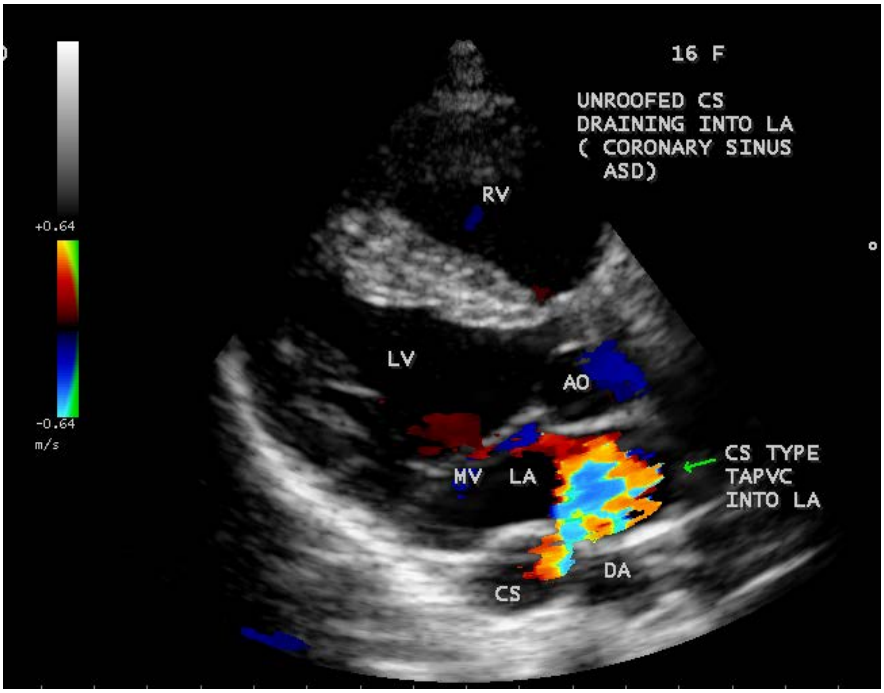


Figure 5. Parasternal long axis view showing the drainage of unroofed coronary sinus into the left atrium, constituting a right-to-left shunt-coronary sinus ASD (atrial septal defect) with total anomalous pulmonary venous connection (TAPVC).

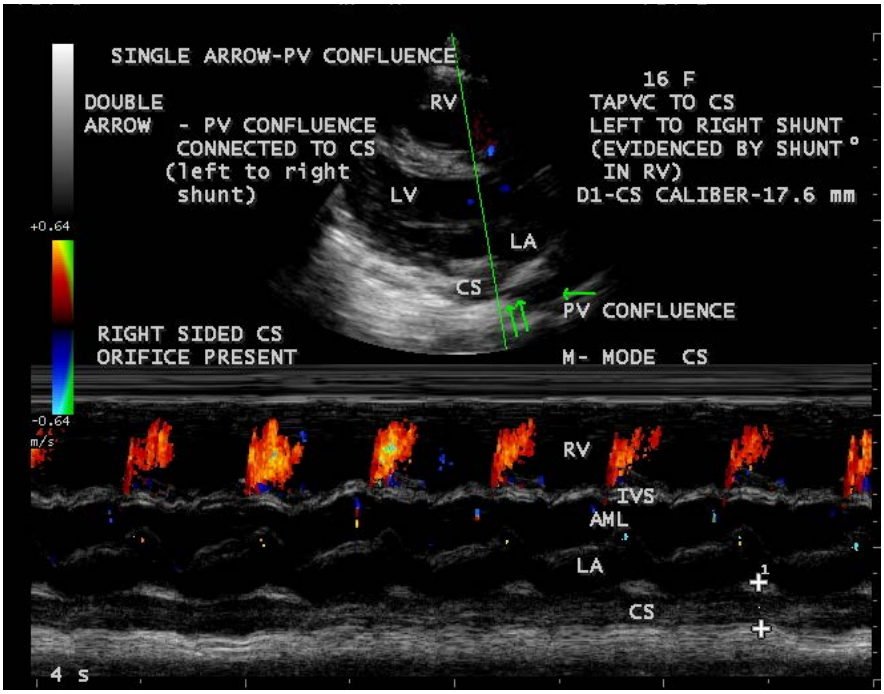


Figure 6. M-mode at CS (coronary sinus) level showing the shunting of blood into RV (right ventricle) from the coronary sinus and CS caliber—17.6 mm.

left common cardinal vein and normally drains into the right atrium. An 'unroofed coronary sinus' abnormally communicate with left atrium and called as "coronary sinus ASD (atrial septal defect)", in addition to draining the cardiac

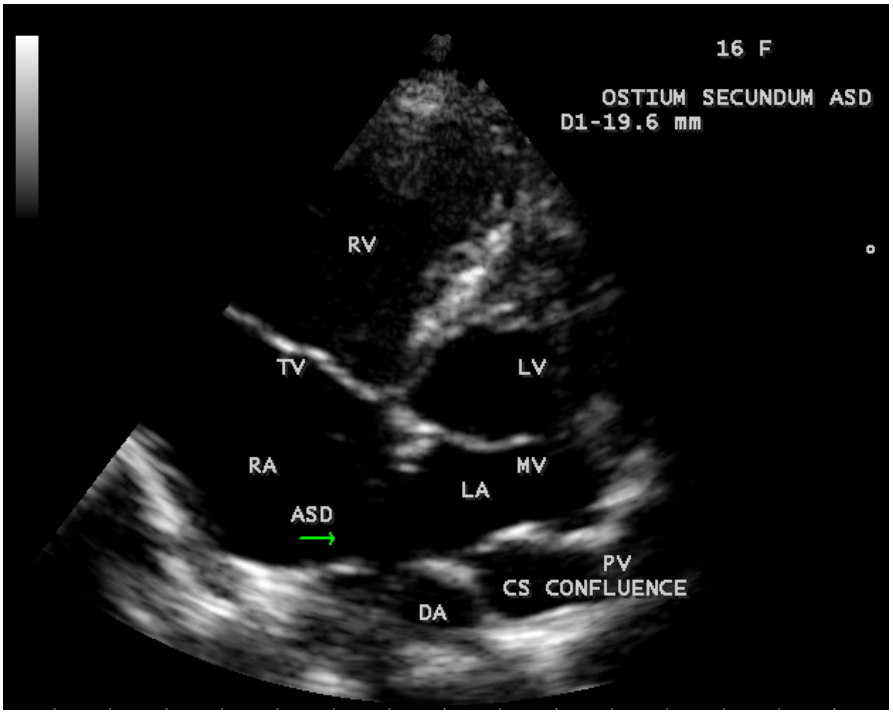


Figure 7. Showing the pulmonary venous confluence and ostium secundum ASD (atrial septal defect) in apical four chamber view (the size of secundum ASD is 19.6 mm).

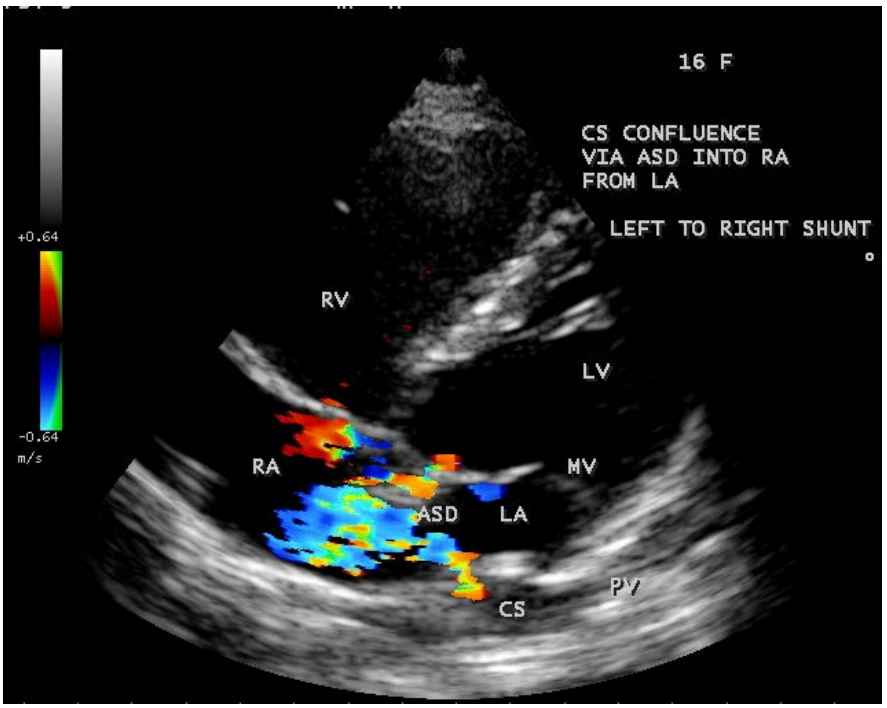


Figure 8. Tilted apical view showing the drainage of pulmonary venous confluence connected to coronary sinus (CS) into the right atrium through the secundum defect.

veins due to impaired development of partition between the left atrium and coronary sinus, *i.e.*, subsequent dissolution of this partition [8], the roof coronary sinus either partially or completely. It results from imperfect or complete failure

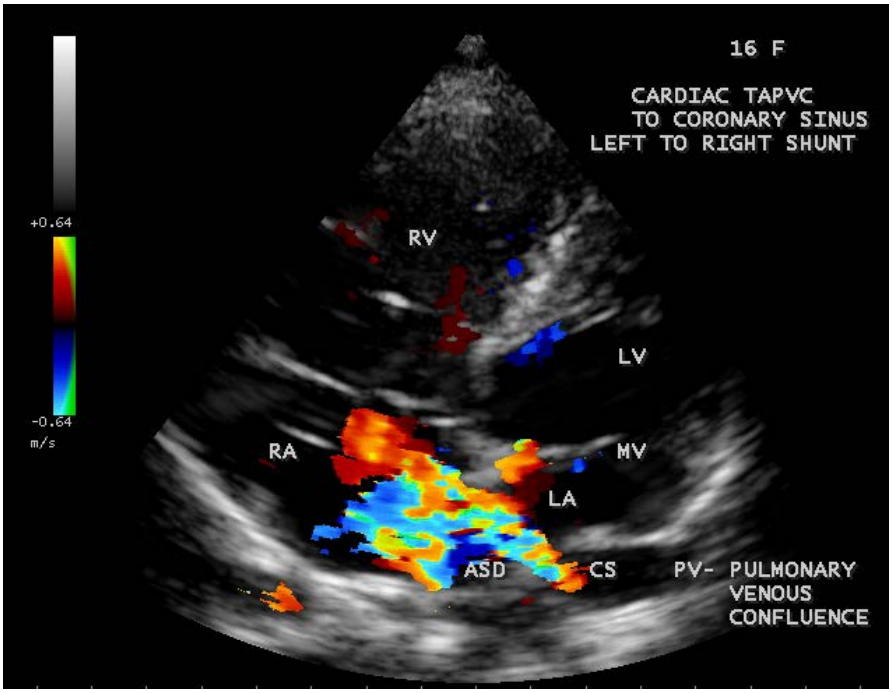


Figure 9. Tilted apical view showing the drainage of pulmonary venous confluence through ASD and constituting a left-to-right shunt.

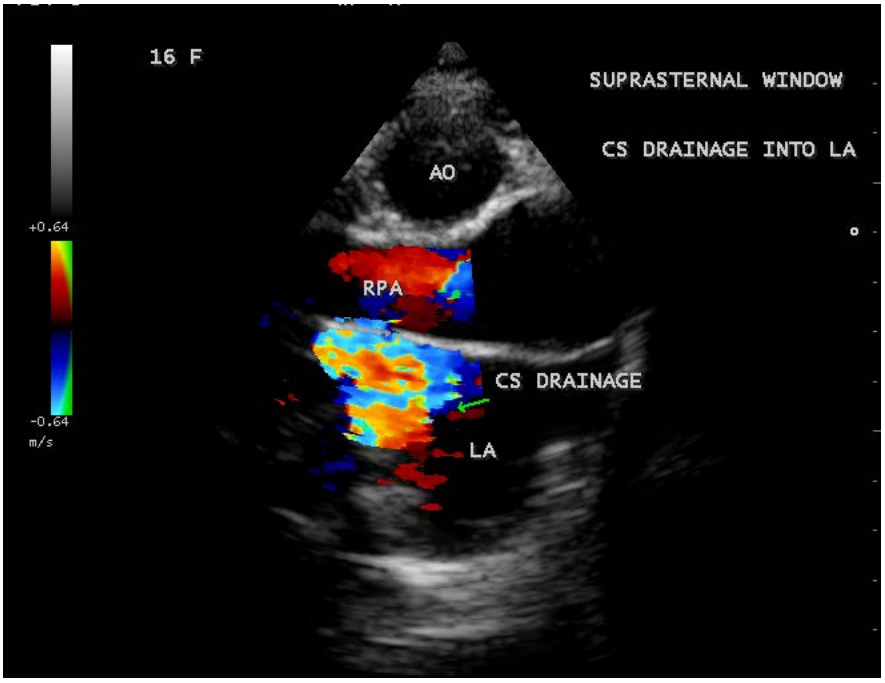


Figure 10. Suprasternal window showing the drainage of coronary sinus into left atrium (Coronary sinus ASD (atrial septal defect)).

of development of the left atriovenous fold and manifested as a focal (fenestration or partial unroofing) or complete absence of the coronary sinus septum. The fenestration into left atrium (LA) typically occurs between LA appendage and left upper pulmonary vein. Thus, coronary sinus ASDs are believed to arise

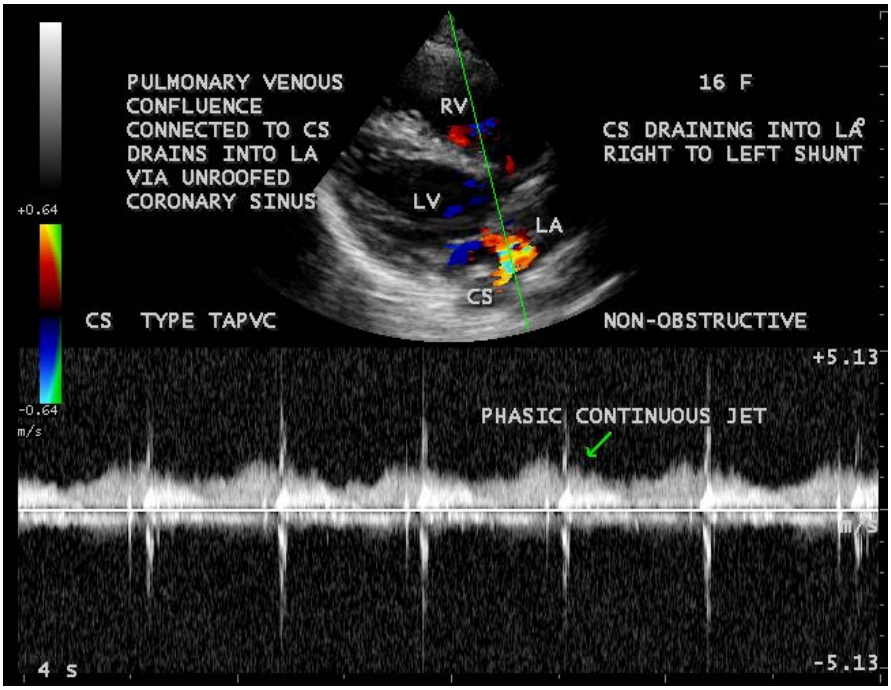


Figure 11. CW (Continuous Wave) Doppler showing the phasic continuous jet of pulmonary venous confluence connected to CS (coronary sinus)—non-obstructive type, draining into left atrium.

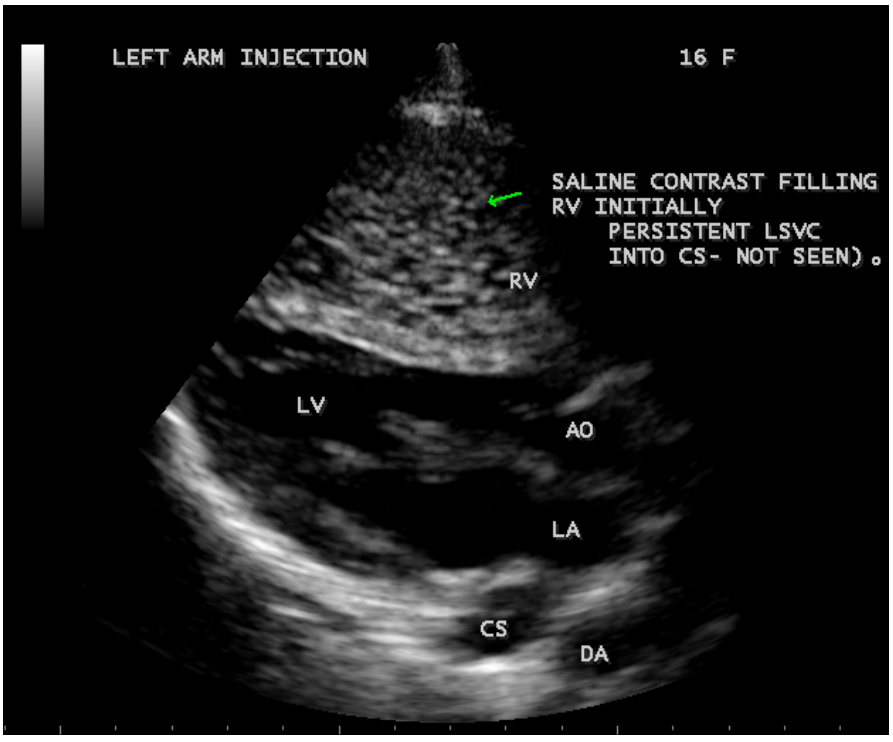


Figure 12. Bubble study showing the absence of persistent left superior vena cava (PLSVC) to coronary sinus by early appearance of saline contrast in the right ventricle (RV)—left arm injection (Parasternal long axis view). (Note: In PLSVC to CS, saline contrast appears earlier in CS (coronary sinus) than in the right sided cardiac chambers (RA (right atrium), RV (right ventricle)).

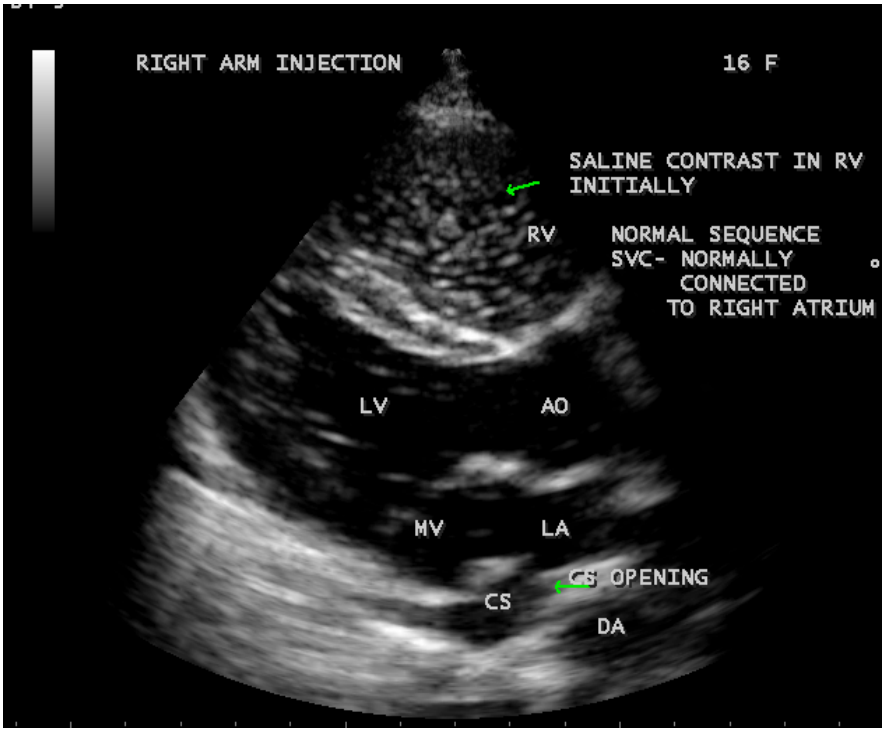


Figure 13. Bubble study showing the normal connection of right superior vena cava (RSVC) by early appearance of saline contrast in the right ventricle (RV)-right arm injection (Parasternal long axis view). (Note: If saline contrast appears earlier in CS than in the right-sided cardiac chambers (RA, RV), RSVC (right superior vena cava) is absent, PLSVC to CS is possible and the CS is aneurysmally dilated (giant CS).

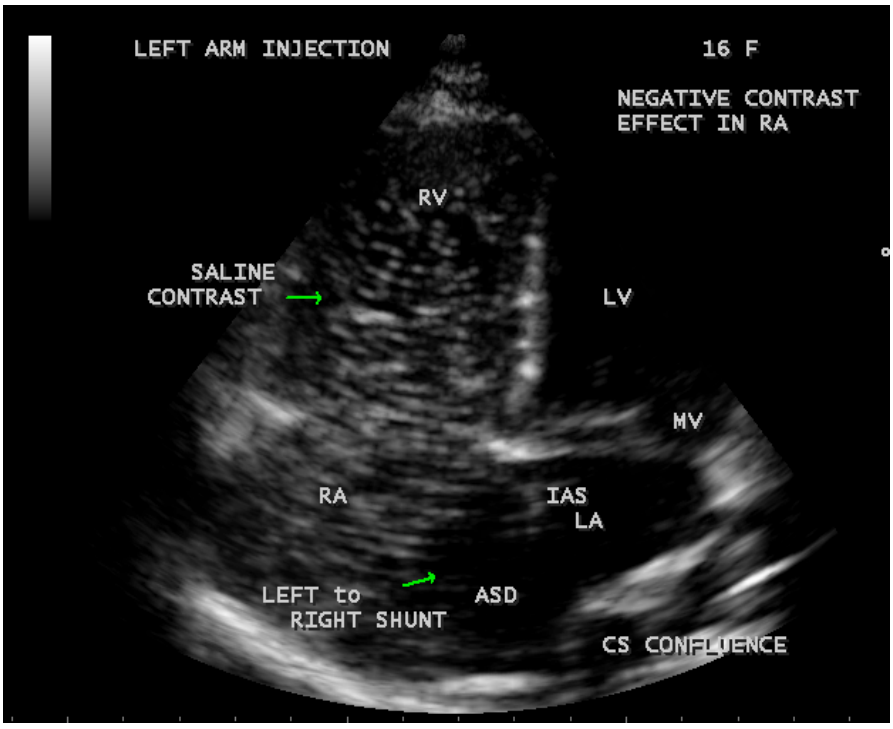


Figure 14. Bubble study showing the negative contrast effect in the right atrium due to left to right shunt through a secundum defect in apical four chamber view.

from developmental failure of the wall between the left atrium and CS. It is the rarest type of atrial septal defect [9] [10] in which the anterior wall of coronary sinus was deficient and therefore allowed an interatrial communication [11]. The anatomic abnormality is variable and classified into four types by Kirklin and Barrat-Boys [12] as shown in **Table 2**.

No specific risk factors or known teratogens have been associated with coronary sinus ASDs. It may be observed most often in association with atrial situs abnormalities and heterotaxy syndromes with polysplenia or asplenia. No other genetic syndromes are known to be associated with coronary sinus ASDs.

In TAPVC (total anomalous pulmonary venous connection), the pulmonary veins connect directly to the systemic venous circulation via persistent splanchnic connections and results from the failure of the transfer of pulmonary venous drainage from the splanchnic plexus to the left atrium in the normal developmental sequence. Four types have been described as supracardiac, cardiac, infracardiac and mixed by Darling *et al.* [13]. Cardiac type TAPVC occurs in 25% of cases in which the pulmonary venous confluence drains into the coronary sinus or directly to the right atrium.

3.1. Echocardiographic Features

Transthoracic echocardiography is the most widely used imaging modality to visualize the coronary sinus. In parasternal long axis view, the coronary sinus is represented by a small sonolucency at the posterior left atrioventricular (AV) junction and moving with cardiac motion. In this view, the coronary sinus is anterior to and quite separate from the larger, the more posterior sonolucency of the descending thoracic aorta as shown in **Figure 3** which is fixed and not move with cardiac motion. It has been suggested that there is a symmetric distension of coronary sinus in elevated right heart pressures and in persistent LSVC, it maintains its oval shape with an eccentricity index of <0.8 [14]. The normal CS caliber is 8.7 ± 2.5 mm. In this patient, the CS is dilated and its caliber was 17.6 mm as shown in **Figure 6**. The dilated coronary sinus is sometimes the initial echocardiographic abnormality detected and this finding should always prompt a search for anomalous pulmonary venous drainage. Dilated coronary sinus is the most common echocardiographic finding of a PLSVC and the diagnosis is then confirmed by saline contrast echocardiography ("bubble study").

After agitated saline injection into a left-sided brachial vein, bubble contrast appears in the right ventricle as shown in **Figure 12**, suggesting the absence of PLSVC draining to coronary sinus. When agitated saline is injected into a right-sided brachial vein, the echo contrast enhances the right ventricle before the CS,

Table 2. Types of unroofed coronary sinus.

Type I	Completely unroofed with LSVC
Type II	Completely unroofed without LSVC
Type III	Partially unroofed midportion
Type IV	Partially unroofed terminal portion

thus confirming a normal right SVC [15] as shown in **Figure 13**. A negative contrast effect in the right atrium as shown in **Figure 14** indicates a left-to-right shunt via secundum defect [16]. An unroofed coronary sinus draining into the left atrium (right-to-left shunt) was shown in **Figure 5** and M-mode at CS level revealed the normal orifice of CS at right side (as evidenced by shunting of blood into RV—left to right shunt due to connection of pulmonary venous confluence to CS) as shown in **Figure 6**.

Most children with isolated coronary sinus defects are asymptomatic. The size of the defect and the degree of shunting determine the symptoms similar to the other types of ASDs. Pulmonary venous obstruction may occur in all types of anomalous connections and in cardiac type, obstruction seldom develops, but may occur at the junction of common pulmonary vein to the coronary sinus. Obstructive TAPVC may present with pulmonary edema or severe hypercyanotic episodes and it is more common in infradiaphragmatic type. A continuous non-phasic pulmonary venous flow is characteristic of obstruction in TAPVC. In this case, the continuous flow is phasic as shown in **Figure 11** suggesting a non-obstructive flow pattern.

3.2. Management

3.2.1. Medical Therapy

No specific medical therapy is necessary and Intervention is not required in asymptomatic patients.

In patients with TAPVC to coronary sinus, if pulmonary venous obstruction occurs, the hypercyanotic episodes are treated with pulmonary vasodilators such as inhaled nitric oxide, lower-dose magnesium sulphate and PGE_1 (Prostaglandin E_1-alprostadil IV). Pulmonary edema is best treated with surgical relief of obstruction, but diuretics, assisted ventilation with high fraction of inspired oxygen (FIO_2) and end-expiratory pressure are often helpful.

3.2.2. Catheter Therapy

Transcatheter device closure is not considered a feasible option for coronary sinus ASDs due to its proximity to tricuspid valve and conduction system and a lack of adequate tissue rim for device seating [17]. Klijima *et al.* reported success in two cases and the orifice of coronary sinus was closed using the Amplatzer septal occluder without any complications in isolated defects [18]. Successful closure is possible in small coronary ASDs [19]. However, St. Jude Medical's Amplatzer atrial septal occluder may cause tissue erosion and leads to problems such as cardiac tamponade.

No catheter-corrective therapy is possible for TAPVC (total anomalous pulmonary venous connection).

3.2.3. Surgical Therapy

Surgical closure in childhood is recommended for CS-ASDs similar to secundum defects. Indication for surgery in small defects are controversial, but the risk of paradoxical embolism and cryptogenic stroke remains. Severe pulmonary

hypertension (pulmonary vascular resistance (PVR) > 15 Wood units is a contraindication for surgical repair.

Surgical treatment is complicated by its proximity to AV node and sutures must be placed close to the superior rim of the defect and therefore patch repair (pericardial patch or bioprosthetic material) is recommended. The mortality from surgical closure of coronary sinus ASDs appear to be low [20].

In TAPVC, the goal of surgery is to redirect pulmonary vein flow to the left atrium. In cardiac connection (to coronary sinus), the atrial septum is resected partially and a new septum is surgically created, directing pulmonary veins to the left atrium. The coronary sinus may be separately tunneled to the right atrium or left to drain with pulmonary veins to the left atrium which may be preferred in this girl.

3.3. Screening of Population

A SVC (superior vena cava) type total anomalous pulmonary venous connection (TAPVC) was found by echocardiographic imaging in a 6-year old girl as shown in **Figure 15** and **Figure 16** and the right atrium is grossly dilated. A coronary sinus ASD with TAPVC was detected in a newborn as shown in **Figures 17-22** and the right atrium is not much dilated as in **Figure 15** and **Figure 16**.

4. Conclusion

Coronary sinus ASD is a rare congenital anomaly which might be difficult to di-

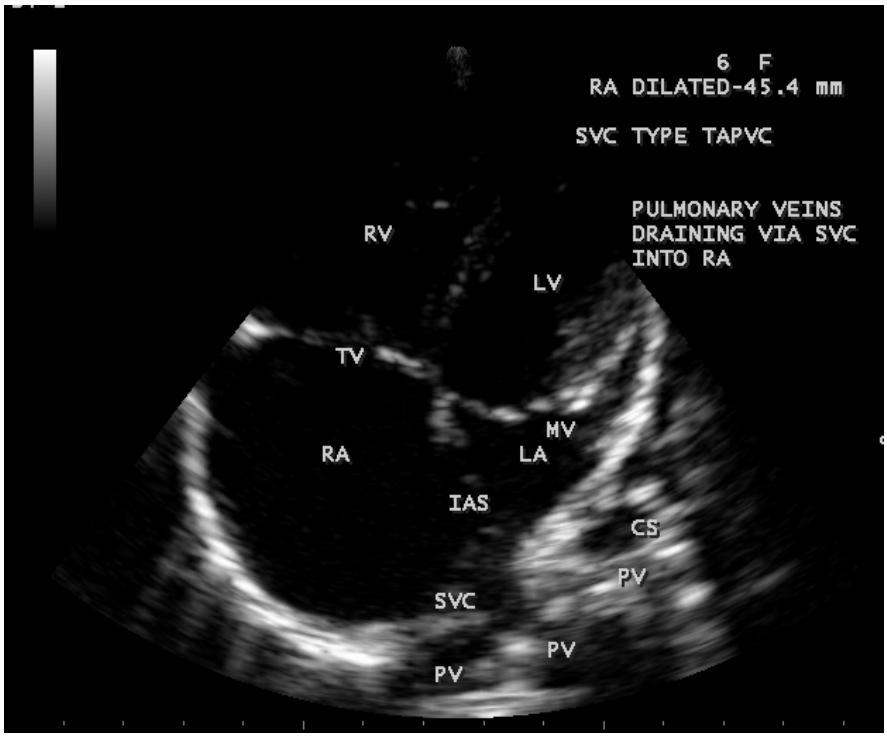


Figure 15. SVC (superior vena cava) type total anamolous pulmonary venous connection (TAPVC)—Apical four chamber view in a 6-year old girl. Right atrium (RA) dilated—45.4 mm.

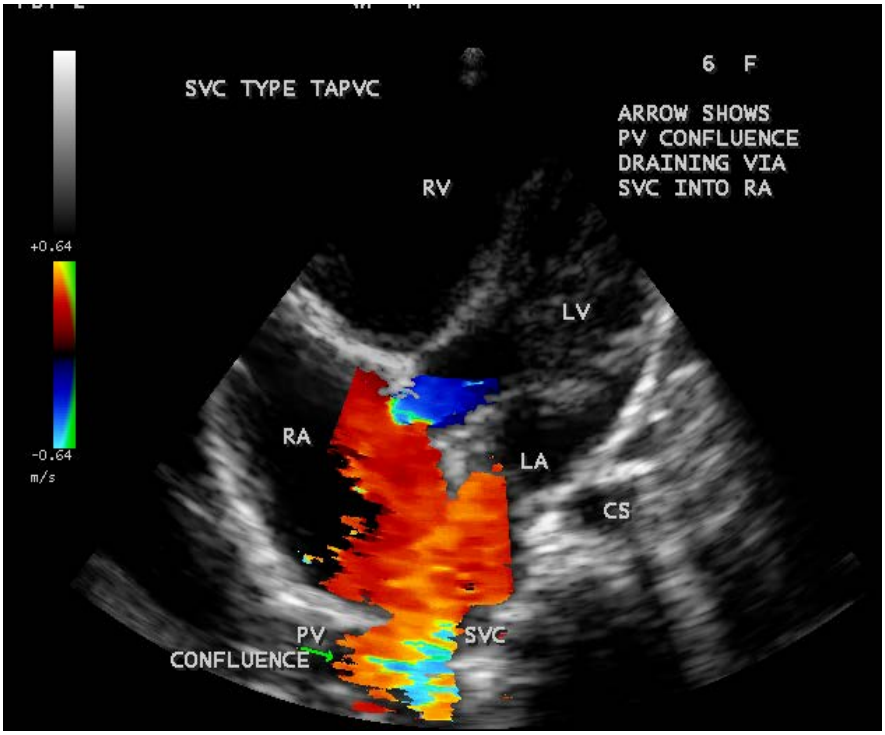


Figure 16. Color flow imaging showing the SVC (superior vena cava) type total anomalous pulmonary venous connection (TAPVC) in a 6-year old girl in apical four chamber view.

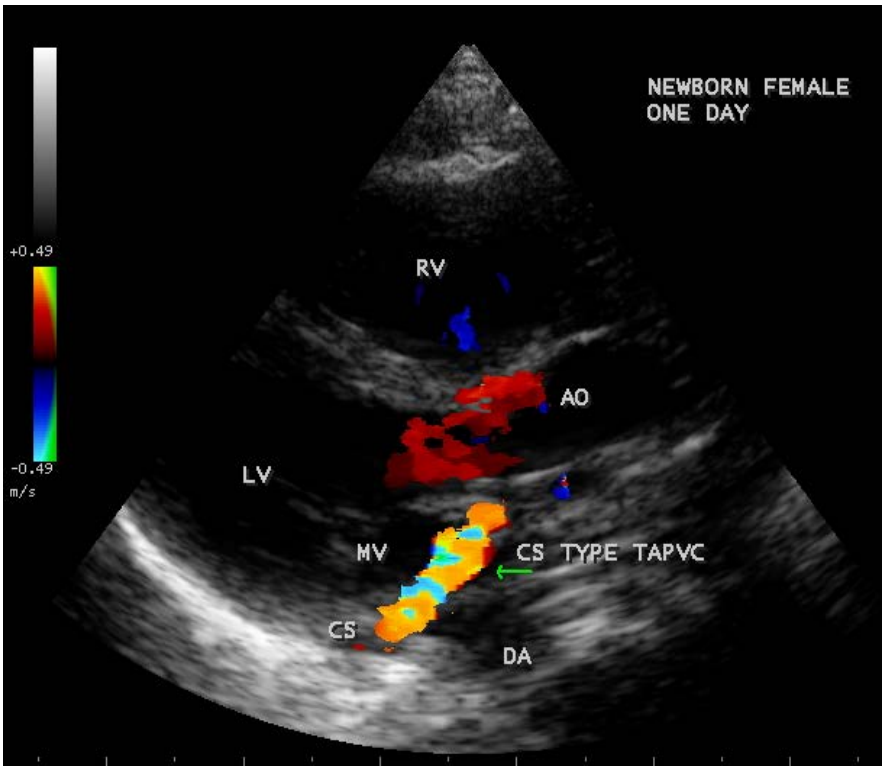


Figure 17. Parasternal long axis view showing the coronary sinus ASD (atrial septal defect) with total anomalous pulmonary venous connection (CS type TAPVC) in a new born.

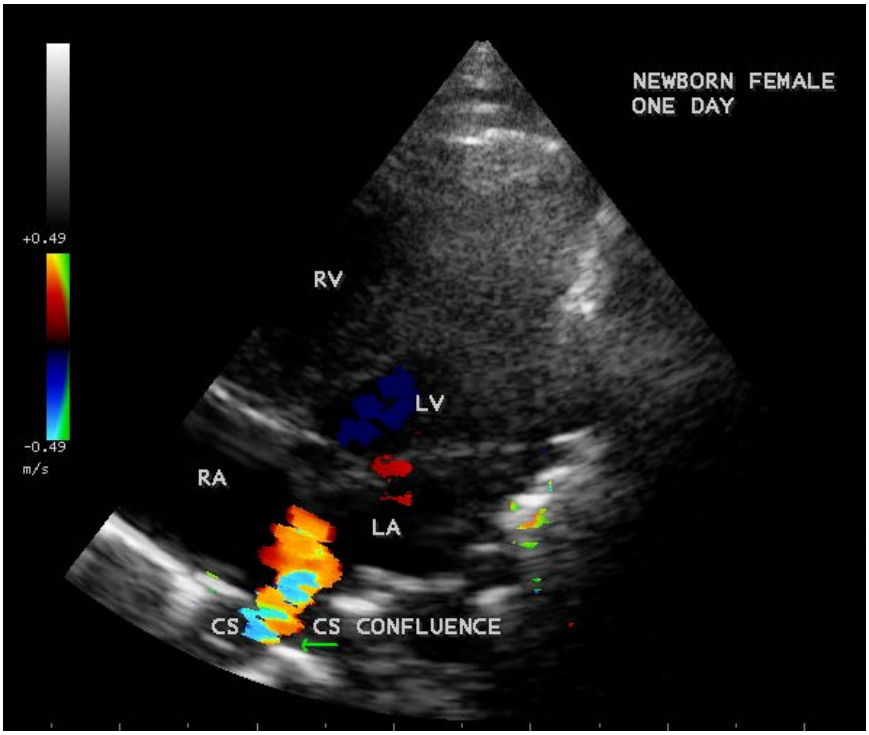


Figure 18. Tilted apical view showing the CS confluence in a newborn.

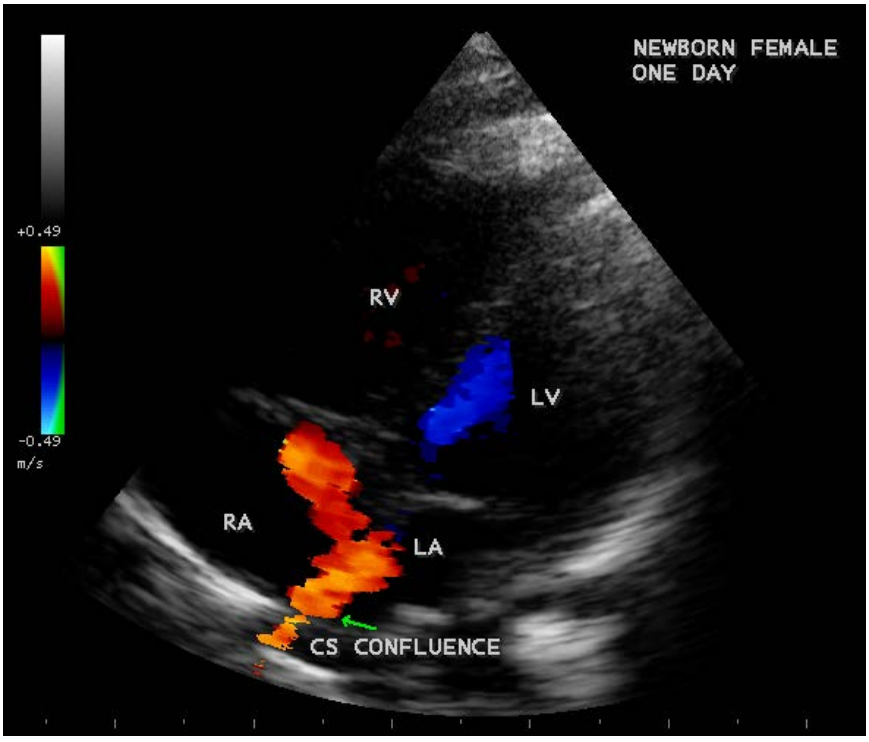


Figure 19. Tilted apical view showing the drainage of CS confluence into Left atrium (LA) in apical four chamber view in a newborn.

agnose [21] and it has been observed in patients with isolated secundum ASD [22]. A coronary sinus defect without an associated PLSVC (persistent left superior vena cava) is rare. It is not a true defect of the atrial septum [23] and its

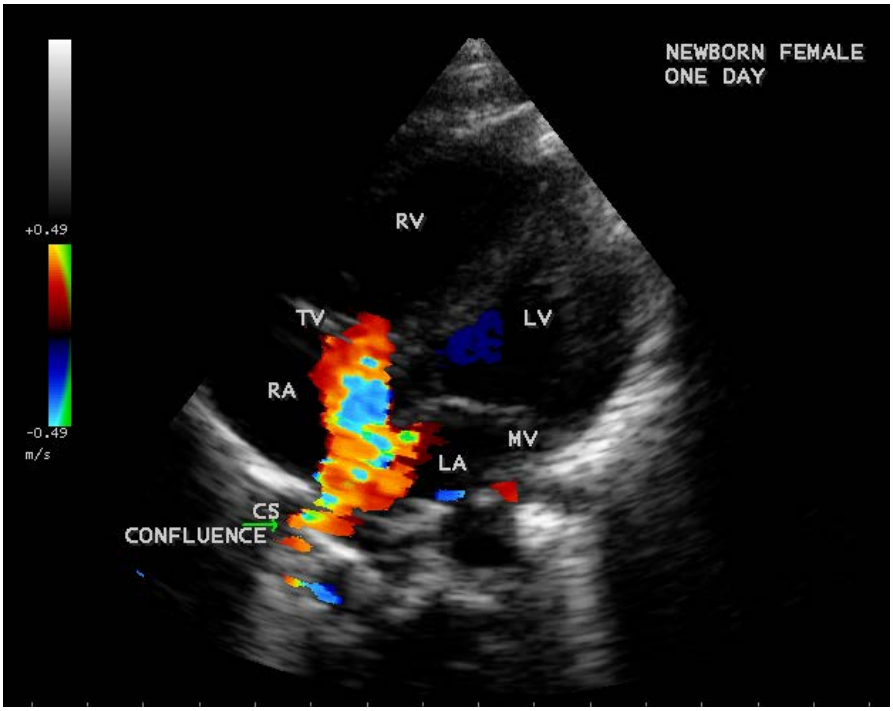


Figure 20. Tilted apical view—Color flow imaging showing the CS confluence jet mimicking as superior vena cava type total anomalous pulmonary venous connection (SVC type TAPVC) in a newborn.

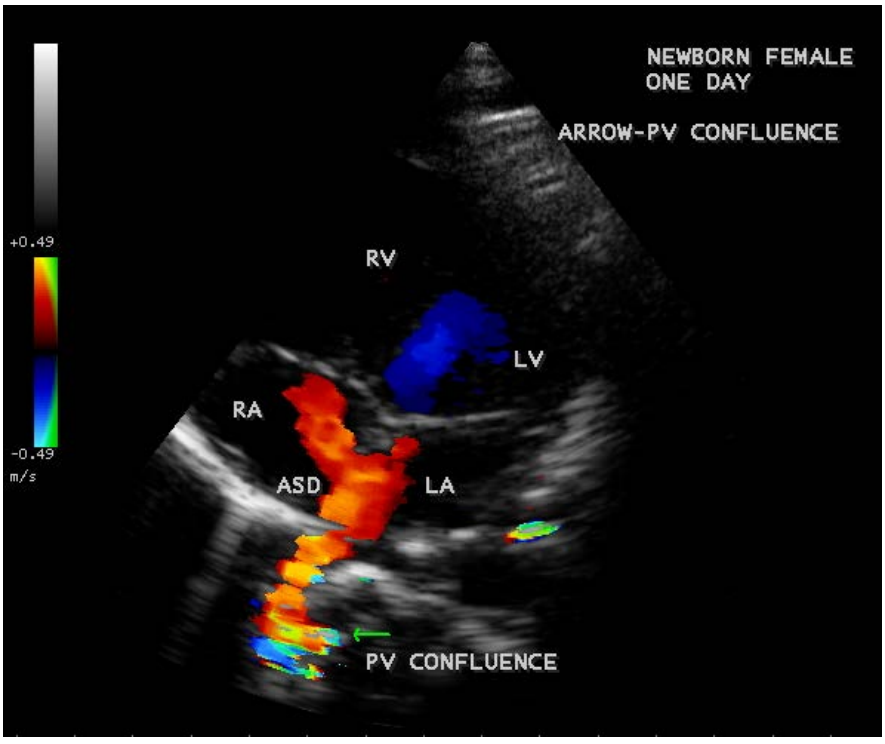


Figure 21. Apical four chamber view showing the coronary sinus type total anomalous pulmonary venous connection in a newborn and a secundum ASD (atrial septal defect). Right atrium (RA) is not dilated and thus differentiated from superior vena cava type total anomalous pulmonary venous connection (SVC type TAPVC) as shown in Figure 15 and Figure 16 in which the right atrium is grossly dilated.

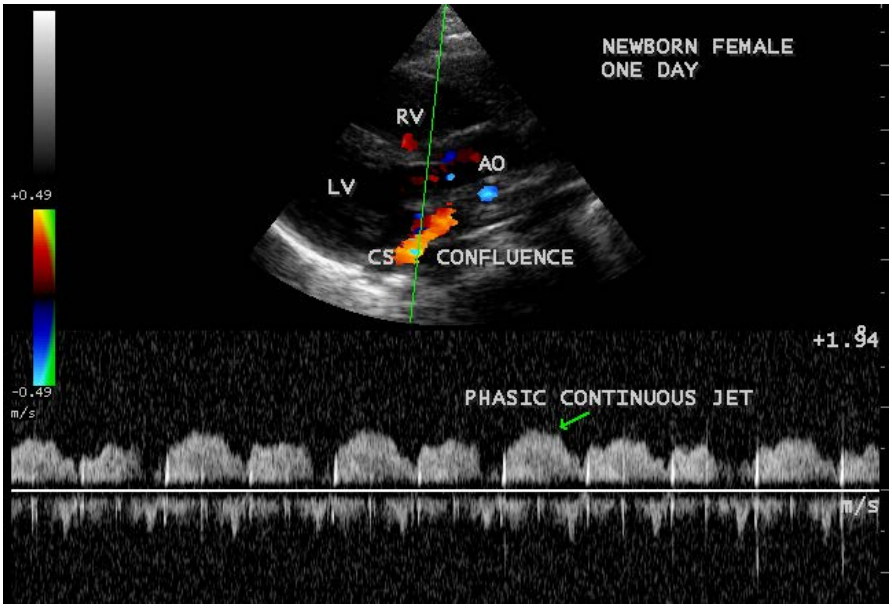


Figure 22. CW (Continuous Wave) Doppler Showing the phasic continuous jet of CS (coronary sinus) confluence in a newborn.

physiology is similar to other ASDs. Interatrial communication is through the mouth of the coronary sinus, which is located below and posterior to the fossa ovale and Imaging plays a crucial role in the diagnosis. A coronary sinus with partially unroofed terminal portion (Type IV) was found by transthoracic echocardiography, it is dilated due to total anomalous pulmonary venous connection (TAPVC) rather than PLSVC and constituting a cardiac type TAPVC to coronary sinus in a 16-year old girl.

References

[1] Weyman, A.E. (1994) Left Ventricular Inflow Tract II: The Left Atrium, Pulmonary Veins, and Coronary Sinus. In: *Principles and Practice of Echocardiography*. 2nd Edition, Lea & Febiger, Philadelphia, 491.

[2] Mantini, E.G.C., Lillehei, C.W. and Edwards, J.E. (1966) Congenital Anomalies Involving the Coronary Sinus. *Circulation*, **33**, 317-327. https://doi.org/10.1161/01.CIR.33.2.317

[3] Eliot, R.S., Wang, Y., Elliott, L.P., Varco, R.L. and Edwards, J.E. (1963) Clinical Pathologic Conference. *American Heart Journal*, **66**, 542-551. https://doi.org/10.1016/0002-8703(63)90388-0

[4] Scheller, V., Mazur, W., Kong, J. and Chung, E.S. (2009) Coronary Sinus to Left Atrial Communication. *Case Reports in Medicine*, **2009**, Article ID: 790715. https://doi.org/10.1155/2009/790715

[5] Raghib, G., Ruttenberg, H.D., Anderson, R.C., Amplatz, K., Adams Jr., P., Edwards, J.E. (1965) Termination of Left Superior Vena Cava in Left Atrium, Atrial Septal Defect, and Absence of Coronary Sinus: A Developmental Complex. *Circulation,* **31**, 906-918. https://doi.org/10.1161/01.CIR.31.6.906

[6] Wilson, J. (1798) A Description of the Very Unusual Formation of the Human Heart. *Philosophical Transactions of the Royal Society of London*, **88**, 346. https://doi.org/10.1098/rstl.1798.0014

[7] Brody, H. (1942) Drainage of Pulmonary Veins into the Right Side of the Heart. *Archives of Pathology & Laboratory Medicine,* **33**, 221.

[8] Knauth, A., McCarthy, K.P., Webb, S., Ho, S.Y., Allwork, S.P., Cook, A.C., *et al.* (2002) Interatrial Communication Through the Mouth of the Coronary Sinus. *Cardiology in the Young*, **12**, 364-372. https://doi.org/10.1017/S104795110001297X

[9] Ootaki, Y., Yamaguchi, M., Yoshimura, N., Oka, S., Yoshida, M. and Hasegawa, T. (2003) Unroofed Coronary Sinus Syndrome: Diagnosis, Classification and Surgical Treatment. *The Journal of Thoracic And Cardiovascular Surgery,* **126**, 1655-1656. https://doi.org/10.1016/S0022-5223(03)01019-5

[10] Yeager, S., Chin, A. and Sanders, S. (1984) Subxiphoid Two-Dimensional Echocardiographic Diagnosis of Coronary Sinus Septal Defects. *American Journal of Cardiology*, **54**, 686-687. https://doi.org/10.1016/0002-9149(84)90282-0

[11] Gould, S.E. (1960) Raghib Complex: Pathology of the Heart. 2nd Edition, Charles C. Thomas, Spring Field, 275. *Echocardiography Journal*.

[12] Kirklin, J. and Barrat-Boys, B.G. (1986) Cardiac Surgery. John Wiley and Sons, New York.

[13] Craig, J.M., Darling, R.C. and Rothney, W.B. (1957) Total Pulmonary Venous Drainage into the Right Side of the Heart; Report of 17 Autopsied Cases Not Associated With Other Major Cardiovascular Anomalies. *Laboratory Investigation*, **6**, 44-64.

[14] Kolski, B.C., Khadivi, B., Anawati, M., *et al.* (2011) The Dilated Coronary Sinus: The Utility of Coronary Sinus Cross Sectional Area And Eccentricity Index in Differentiating Right Atrial Pressure Overload From Persistent Left Superior Vena Cava. *Echocardiography*, **28**, 829-832. https://doi.org/10.1111/j.1540-8175.2011.01445.x

[15] Dearstine, M., Taylor, W. and Kerut, E.K. (2000) Persistent Left Superior Vena Cava, Chest X-Ray and Echocardiographic Findings. *Echocardiography*, **17**, 453-455. https://doi.org/10.1111/j.1540-8175.2000.tb01164.x

[16] Feigenbaum, H., Armstrong, W.F. and Ryan, T. (2005) Congenital Heart Diseases, Feigenbaum's Echocardiography. 6th Edition, Lippincott Williams & Wilkins, Philadelphia, Chapter 18, Figure 18.51 A, 588.

[17] Wang, J.K., Chen, S.J., Hsu, J.Y., *et al.* (2014) Midterm Follow-Up Results of Transcatheter Treatment in Patients with Unroofed Coronary Sinus. *Catheterization and Cardiovascular Interventions*, **83**, 243-249.

[18] Klijima, Y., Taniguchi, M. and Akagi, T. (2011) Catheter Closure of Coronary Sinus Atrial Septal Defect Using Amplatzer Septal Occluder. *Cardiology in the Young*, **22**, 223-226.

[19] Di Bernardo, S., Fasnacht, M. and Berger, F. (2003) Transcatheter Closure of a Coronary Sinus Defect with an Amplatzer Septal Occluder. *Catheterization and Cardiovascular Interventions*, **60**, 287-290.

[20] Jost, A., Connolly, C.H., Danielson, M., Dearani, G.K., Wames, J.A., Jamil, C.A. and Tajik, A. (2007) Clinical Features and Surgical Outcome in 25 Patients with Fenestrations of the Coronary Sinus. *Cardiology in the Young*, **17**, 592-600.

[21] Ngee, T., Lim, M.C., de Laarazabal, C. and Sundaram, R.D. (2011) Unroofed Coronary Sinus Defect. *Journal of Computer Assisted Tomography*, **35**, 246-247. https://doi.org/10.1097/RCT.0b013e31820828c2

[22] Freedom, R.M., Culham, J.A.G. and Rowe, R.D. (1981) Left Atrial to Coronary Sinus Fenestration (Partially Unroofed Coronary Sinus): Morphologic and Angiocar-

diographic Observations. *British Heart Journal*, **46**, 63-68.
https://doi.org/10.1136/hrt.46.1.63

[23] Martin, S.S., Shapiro, E.P. and Mukherjee, M. (2014) Atrial Septal Defects. Clinical Manifestations, Echo Assessment, and Intervention. *Clinical Medicine Insights Cardiology*, **8**, 93-98.

Scientific Research Publishing

Case Reports in Clinical Medicine, 2017, 6, 19-23
http://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

A Rare Presentation of Popliteal Vein Aneurysm: Case Report

Ahmad Rezaee Azandaryani[1], Pezhman Ghaderzadeh[1], Leili Ebrahimi Farsangi[2*], Sayad Nasirzadeh[3]

[1]Radiology Department, Hamadan University of Medical Sciences, Besat Hospital, Hamadan, Iran
[2]Alborz University of Medical Sciences, Karaj, Iran
[3]Hamadan University of Medical Sciences, Besat Hospital, Hamadan, Iran
Email: Ahmad_rezaee20@yahoo.com, pejmanhsr92@gmail.com, *lilieage@gmail.com, sayadnasirzadeh@yahoo.com

How to cite this paper: Azandaryani, A.R., Ghaderzadeh, P., Farsangi, L.E. and Nasirzadeh, S. (2017) A Rare Presentation of Popliteal Vein Aneurysm: Case Report. *Case Reports in Clinical Medicine*, **6**, 19-23.
http://dx.doi.org/10.4236/crcm.2017.61002

Received: November 28, 2016
Accepted: January 10, 2017
Published: January 13, 2017

Abstract

Popliteal venous aneurysms (PVAs) are very rare. More often it is an incidental finding found on duplex ultrasonography. Venous thrombosis and pulmonary emboli is the primary presentation in most cases of PVAs, hence it is a clinically significant abnormality and a potentially morbid and fatal condition. Early surgical repair should be performed immediately after the diagnosis is made because of the risk of pulmonary emboli. We report a case of symptomatic PVA presenting primarily with local symptoms and chronic pain in popliteal region.

Keywords

Aneurysm, Popliteal Vein

1. Introduction

Popliteal venous aneurysms (PVAs) are very rare [1] [2]. More often it is an incidental finding found on duplex ultrasonography. Until 2000, 117 cases of PVAs have been reported in the literature, although its commonly asymptomatic nature warrants its actual prevalence to be higher [3]. In 7300 patients who presented with symptoms suggestive of venous disease PVA were diagnosed using duplex ultrasonography in 0.1% - 0.2% of all studies [4]. Venous thrombosis and pulmonary emboli is the primary presentation in most cases of PVAs, hence it is a clinically significant abnormality and a potentially morbid and fatal condition. Also, its other complications such as paradoxical embolization and postthrombotic syndrome have been reported [3] [6]. However, we report a symptomatic case of PVA presenting with local symptoms and chronic pain in popliteal region.

2. Case Report

A 38-year-old white male, previously healthy soccer player referred to our center for investigation of a chronic severe pain in the posterolateral region of the left knee and swelling in the left popliteal region. The patients' complaints had begun around 2.5 years ago. The pain was severe and disabling in the first year and then it was moderate and bearable. The pain was recurrent and occurred during routine daily activities and on palpation. The patient did not have any history of deep vein thrombosis, phlebitis and claudication but being a professional soccer player, our patient mentioned the history of recurrent trauma to legs and knee regions. Also, before the onset of the pain, the patient had experienced a trauma in the left knee while playing soccer. The patient in our case had undergone evaluations by orthopedics, physiatrists, neurologists and vascular surgeons prior to referral to our center. On physical examination, a small soft mass was present in the upper part of the left popliteal fossa, tenderness was also present on palpation. However, there were no signs of local inflammation and erythema, lower extremity edema and peripheral arterial angiopathy. Femoral, tibial and dorsalis pedis pulses of both sides were palpable. Head, neck, chest and abdomen examination, blood pressure, arterial oxygen saturation, chest radiography and electrocardiogram were normal. Noteworthy, according to knee joint magnetic resonance imaging, plain left knee radiography and physical examination performed by orthopedics, the patient was given reassurance that there was no pathology in the left knee joint that could explain and relate to our patients' complaints. Duplex ultrasonography was done for the patient on referral nearly 1 year after the onset of the symptoms, venous aneurysm with antero-posterior (AP) diameter of 16 milimiters was present in the left popliteal vein (Figure 1 and Figure 2).

Then the patient was referred to vascular surgeon for surgical care. The patient referred to two vascular surgery specialists, and they believed surgery was not indicated. The patient referred once again for follow up to our center around 1.5 years later (2.5 years after the onset of the symptoms). During this period the patient had still experienced pain and mild swelling in the left popliteal region

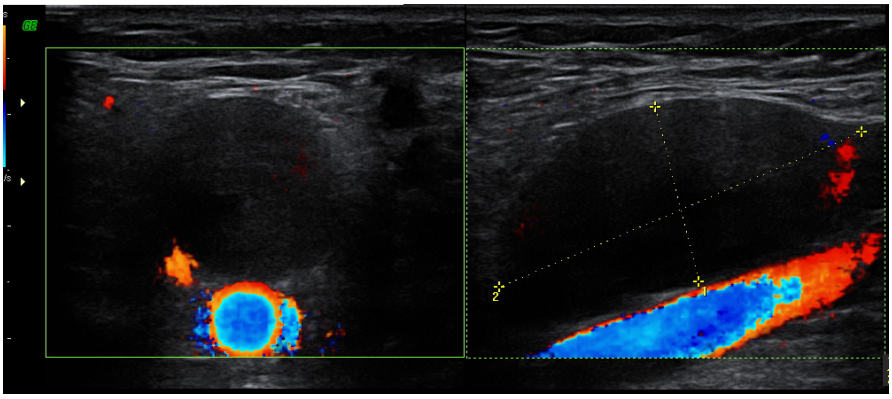


Figure 1. Axial and sagittal color duplex ultrasonographic images of left popliteal cavity before calf veins squeezing demonstrates echo free popliteal aneurysm anterior to popliteal artery.

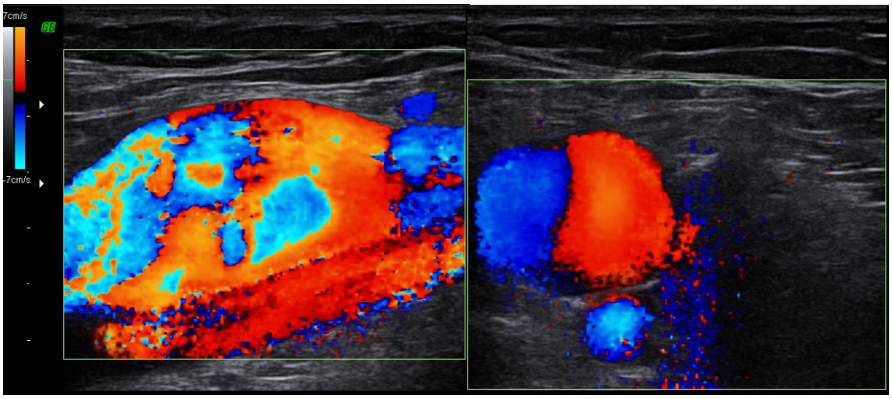


Figure 2. Axial and sagittal color duplex ultrasonographic images of left popliteal cavity after calf veins squeezing demonestrates popliteal aneurysm anterior to popliteal artery.

which was milder and denied any history or signs related to severe complications of PVAs such as deep vein thrombosis or pulmonary emboli. We performed duplex ultrasonography once again plus magnetic resonance imaging (MRI) which this time revealed an AP diameter of 19milimiters in the left popliteal vein (**Figure 3**).

Urgent surgical management was recommended for the patient and the patient was referred to vascular surgery specialists as the next step in care. Noteworthy, informed consent was obtained from our patients regarding case report publication and there was no objection.

3. Discussion

Popliteal venous aneurysms are extremely uncommon abnormality with an incidence of almost <0.5% [7]. PVA is a true aneurysm which consists of intact vessel wall [8]. However, popliteal venous aneurysms still lack an exact definition. Different definitions such as fusiform dilatation at least three times that of normal vein [9] and isolated venous dilatation twice the normal venous diameter have been recommended [10]. In our case, the popliteal aneurysm was saccular type. Inflammation, trauma, degenerative changes and congenital wall weakness may be some of the probable etiologies for PVAs [11]. Nearly all cases of PVAs are diagnosed after patients present with complications due to PVA, deep vein thrombosis and pulmonary emboli being the most common of all [12] [13] [14]. However, in our case, chronic posterolateral left knee pain and local sign such as mild swelling in the popliteal region was the primary presentation. PVAs presenting with knee pain and local symptoms is reported by some other authors too [15] [16] [17]. Coffman *et al.* reported a case with recurrent sharp and severe pain in the popliteal fossa which was later revealed to be due to PVA [17]. Fiori *et al.* reported a case with local discomfort in the left popliteal fossa during daily activities and palpation [16]. In a meta-analysis knee pain and popliteal mass was reported as the clinical presentation secondary to PVA in 35% of cases [4]. Being a soccer player, we believe that trauma may be the probable cause for popliteal aneurysm in our case. Duplex ultrasonography, computed tomography scanning, and magnetic resonance imaging are non-invasive diagnostic methods

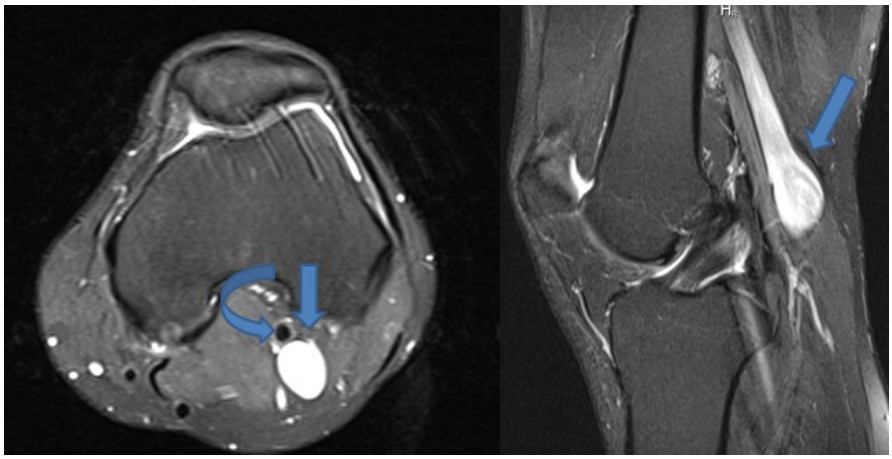


Figure 3. Axial and sagittal fat suppresed proton density MRI images demonstrates left popliteal aneurysm (arrows) posterior to popliteal artery (curved arrow).

applicable in diagnosis of PVAs. Duplex ultrasonography can be used as the initial detection technique because it is non-invasive, inexpensive and conveniently repeatable [18]. Also, ultrasonography could rule out PVAs differential diagnosis such as Baker's cyst and tibiofibular joint cysts.

Surgery is believed to be the gold standard option in treatment of PVAs regardless of being symptomatic or not. Some of the surgical treatment options of symptomatic and asymptomatic cases are resection with end-to-end anastomosis, tangential aneurysmectomy, resection with interposition graft and ligation of the proximal and distal vein. Early post-surgical patency rates are high and there has not been any report regarding recurrent pulmonary embolism post-surgery, however the long-term consequences of surgical repair is not known [3] [19].

4. Conclusion

Popliteal venous aneurysm is a very uncommon abnormality but due its severe consequences such as deep vein thrombosis and pulmonary emboli, it is potentially fatal. This case report demonstrates knee pain and local discomfort as one of the presenting symptoms for PVAs. Therefore, after ruling out more common causes, PVAs should be considered in a patient with chronic knee pain and local discomfort, especially if it is accompanied with swelling. Early surgical repair should be performed immediately after the diagnosis is made because of risk of pulmonary emboli.

References

[1] Sarap, M.D. and Wheeler, W.E. (1988) Venous Aneurysms. *Journal of Vascular Surgery*, **8**, 182-183. https://doi.org/10.1016/0741-5214(88)90407-7

[2] Friedman, S.G., Krishnasastry, K.V., Doscher, W. and Deckoff, S.L. (1990) Primary Venous Aneurysms. *Surgery*, **108**, 92-95.

[3] Sessa, C., Nicolini, P., Perrin, M., Farah, I., Magne, J.L. and Guidicelli, H. (2000) Management of Symptomatic and Asymptomatic Popliteal Venous Aneurysms: A Retrospective Analysis of 25 Patients and Review of the Literature. *Journal of Vas-*

cular Surgery, **32**, 902-912. https://doi.org/10.1067/mva.2000.110353

[4] Bergqvist, D., Bjorck, M. and Ljungman, C. (2006) Popliteal Venous Aneurysm—A Systematic Review. *World Journal of Surgery*, **30**, 273-279. https://doi.org/10.1007/s00268-005-7982-y

[5] Winchester, D., Pearce, W.H., McCarthy, W.J., McGee, G.S. and Yao, J.S. (1993) Popliteal Venous Aneurysms. *Surgery*, **114**, 600-607.

[6] Willinek, W.A., Strunk, H., Born, M., Remig, J., Becher, H. and Schild, H. (2001) Popliteal Venous Aneurysm with Paradoxical Embolization in a Patient with Patent Foramen Ovale. *Circulation*, **104**, E60-E61. https://doi.org/10.1161/hc3701.095641

[7] Labropoulos, N., Volteas, S.K., Giannoukas, A.D., Touloupakis, E., Delis, K. and Nicolaides, A.N. (1996) Asymptomatic Popliteal Vein Aneurysms. *Vascular and Endovascular Surgery*, **30**, 453-457. https://doi.org/10.1177/153857449603000602

[8] Chahlaoui, J., Julien, M., Nadeau, P., Bruneau, L., Roy, P. and Sylvestre, J. (1981) Popliteal Venous Aneurysm: A Source of Pulmonary Embolism. *AJR American journal of Roentgenology*, **136**, 415-416. https://doi.org/10.2214/ajr.136.2.415

[9] Maleti, O., Lugli, M. and Collura, M. (1997) Anevrysmes veineux poplites: Experience personnelle: Les anévrysmes veineux de la fosse poplitée (à l'exclusion de la veine saphène externe). *Phlebologie*, **50**, 53-59.

[10] McDevitt, D.T., Lohr, J.M., Martin, K.D., Welling, R.E. and Sampson, M.G. (1993) Bilateral Popliteal Vein Aneurysms. *Annals of Vascular Surgery*, **7**, 282-286. https://doi.org/10.1007/BF02000255

[11] Hong, D. and Song, S.W. (2013) Pulmonary Embolism Caused by Popliteal Venous Aneurysm. *The Korean Journal of Thoracic and Cardiovascular Surgery*, **46**, 76-79. https://doi.org/10.5090/kjtcs.2013.46.1.76

[12] Grice, G.D., Smith, R.B., Robinson, P.H. and Rheudasil, J.M. (1990) Primary Popliteal Venous Aneurysm with Recurrent Pulmonary Emboli. *Journal of Vascular Surgery*, **12**, 316-318. https://doi.org/10.1016/0741-5214(90)90154-3

[13] Dawson, K. and Hamilton, G. (1991) Primary Popliteal Venous Aneurysm with Recurrent Pulmonary Emboli. *Journal of Vascular Surgery*, **14**, 437. https://doi.org/10.1016/0741-5214(91)90141-G

[14] Lang, E., Bouwman, O. and Faiss, J. (1993) Recurrent Lung Embolisms in Aneurysm of the Popliteal Vein. *Der Chirurg, Zeitschrift fur alle Gebiete der operativen Medizen*, **64**, 503-504.

[15] Premaratne, S., Tan, T.W., Coulter, A.H., Doumite, D. and Zhang, W.W. (2014) Symptomatic Popliteal Vein Aneurysm. *Vascular and Endovascular Surgery*, **48**, 275-276. https://doi.org/10.1177/1538574413518608

[16] Fiori, R., Chiappa, R., Gaspari, E. and Simonetti, G. (2010) A Rare Case of Popliteal Venous Aneurysm. *Case Reports in Medicine*, **2010**, Article ID: 579256. https://doi.org/10.1155/2010/579256

[17] Coffman, S.W., Leon, S.M. and Gupta, S.K. (2000) Popliteal Venous Aneurysms: Report of an Unusual Presentation and Literature Review. *Annals of Vascular Surgery*, **14**, 286-290. https://doi.org/10.1007/s100169910050

[18] Seino, Y., Fujimori, H., Shimai, S., Tanaka, K., Takano, T., Hayakawa, H., *et al.* (1994) Popliteal Venous Aneurysm with Pulmonary Embolism. *Internal Medicine*, **33**, 779-782. https://doi.org/10.2169/internalmedicine.33.779

[19] Gallagher, J.J. and Hageman, J.H. (1985) Popliteal Vein Aneurysm Causing Pulmonary Embolus. *Archives of Surgery*, **120**, 1173-1175.

Scientific Research Publishing

Case Reports in Clinical Medicine, 2017, 6, 24-30
http://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Intravesical Migration of Intrauterine Device and Calculi Formation 12 Years Post Missing: A Case Report

Ahmad Rezaee Azandaryani[1], Pezhman Ghaderzadeh[1], Leili Ebrahimi Farsangi[2*]

[1]Radiology Department, Hamadan University of Medical Sciences, Besat Hospital, Hamadan, Iran
[2]Cardiology Department, Alborz University of Medical Sciences, Karaj, Iran
Email: Ahmad_rezaee20@yahoo.com, pejmanhsr92@gmail.com, *lilieage@gmail.com

How to cite this paper: Azandaryani, A.R., Ghaderzadeh, P. and Farsangi, L.E. (2017) Intravesical Migration of Intrauterine Device and Calculi Formation 12 Years Post Missing: A Case Report. *Case Reports in Clinical Medicine*, **6**, 24-30.
http://dx.doi.org/10.4236/crcm.2017.61003

Received: November 20, 2016
Accepted: January 16, 2017
Published: January 19, 2017

Abstract

Intraunterine device (IUD) is a safe, effective and feasible method used for reversible contraception worldwide. Transvesical migration of an Intrauterine device (IUD) is a rare complication. We describe a case in whom initially a plain abdominopelvic radiography demonstrated calcified string of an IUD in a 42-year-old woman with recurrent lower urinary symptoms (LUTS) and urinary tract infection since 2 years earlier. The IUD had been inserted 12 years earlier and 3 years after placement of the IUD, the patient experienced an uneventful pregnancy and a successful delivery. Sonographic images and later on the cystoscopic procedure confirmed the diagnosis of transvesical migration of the IUD. The IUD was removed using cystoscopic procedure, leaving no complication.

Keywords

Bladder Stone, Intrauterine Contraceptive Device, Intravesical Migration

1. Introduction

Currently, Intrauterine device (IUD) is a safe, cheap, effective and feasible method used for reversible contraception by nearly 100 million women worldwide, mainly because of its high efficiency and low complication rate [1] [2] [3]. IUD could cause complications ranged from mild discomfort to sepsis leading to death [4]. Some of its uncommon complications include infection, bleeding, ectopic pregnancy and uterine perforation [5] [6]. Uterine perforation by IUD can occur in an incidence of 1 - 3 in 1000 insertions [7]. The precise reason behind migration of IUD to organs and cavity near the uterus is unknown. Even so, misplacement or transvesical migration of IUD is a very infrequent complication

and a high proportion of them form calculus [3] [8]. We present this case in order to note that recurrent urinary tract infection (UTI), lower urinary tract symptoms (LUTS) and unexpected pregnancy in a woman with history of missed IUD could be associated with transvesical migration of IUD and calculi formation.

2. Case Report

A 42-year-old woman para 3 with no history of abortion was referred to our hospital for investigation of recurrent lower abdominal pain, irritative lower urinary tract symptoms (LUTS) and strangury which had begun in the last 2 years. The patient had a history of IUD placement in the form of Copper T for contraception, 12 years earlier (10years before the onset of these symptoms). She had had no symptoms after insertion of the IUD. Follow-up examinations had been performed during the first year after placement of the IUD and the insertion had been successful. The woman in our case had been lost to medical follow up for the next 2 year (3 years from the IUD insertion); until she became pregnant. She had not removed the IUD, but routine prenatal sonography did not show any IUD in her endometrial cavity, and the patient delivered her baby by caesarean section at term on account of two previous caesarean sections without any complications. Noteworthy, she underwent simultaneous bilateral tubal ligation (TL) during her caesarean section surgery.

About 2 years before her referral to our hospital, the patient had developed recurrent lower abdominal pain, irritative lower urinary tract symptoms including dysuria, nocturia, increased frequency of urination and strangury. The patient had no history of macroscopic hematuria, but the urinalysis revealed numerous red blood cells and pyuria every time. She referred to different clinics and doctors with these symptoms in the past 2 years and was treated medically for urinary tract infection (UTI) repeatedly. Other laboratory tests were normal.

Then the patient was referred to our hospital for further evaluation. Initially a plain abdominopelvic radiography was performed for the patient and calcification of the IUD string and the IUD itself was visible in the pelvic region (**Figure 1**). Then transabdominal sonography of the pelvis was performed using an ultrasound scanner (GE, USA) equipped with a 3.5-MHz phased-array transducer. The sonographic images revealed a dense intra bladder stone like structure with metallic artifact consistent with a calculus IUD (**Figure 2**). Also, three dimensional ultrasound imaging showed intra bladder IUD and its calcified components (**Figure 3**).

The location of the IUD on sonography did not change when the patient was positioned differently. The patient was referred to Urologist and was hospitalized for a cystoscopic examination, which confirmed the sonographic findings of a calculus IUD implanted in the left anterior wall of the bladder. At first Holmium laser lithothripsy was performed then allowed removal of a copper-T IUD (**Figure 4**). After the procedure, the patient's symptoms resolved completely on follow-up visits. Noteworthy, informed consent was obtained from the patient regarding publishing the case report and there was no objection.

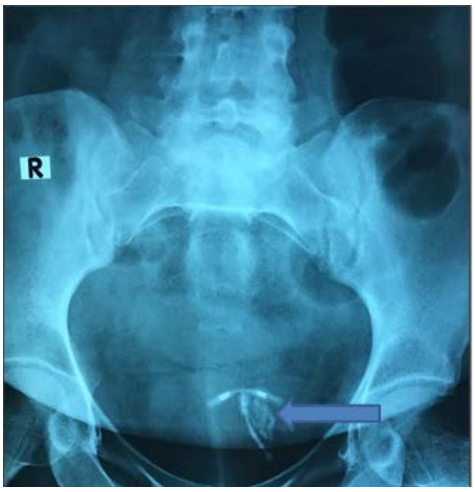


Figure 1. Plain abdominopelvic radiography shows calcification of the IUD string (arrow), intrauterine IUD calcification is unusual and provokes intra bladder IUD migration.

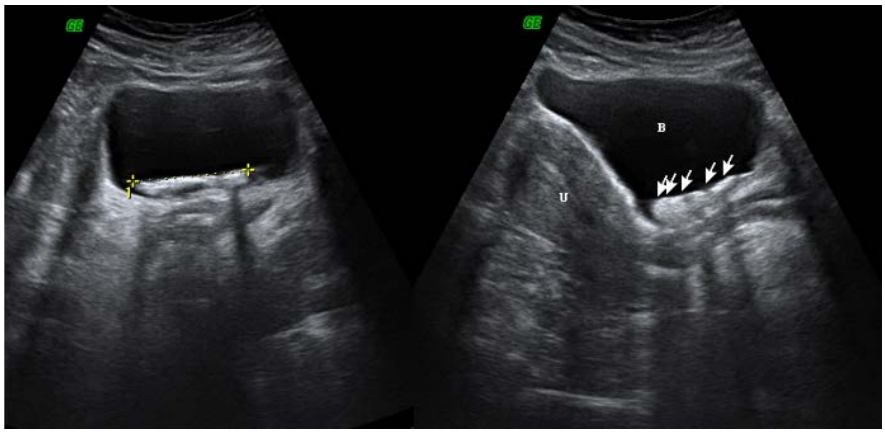


Figure 2. Axial and sagittal trans abdominal ultrasound images shows dense intra bladder stone like lesion with metallic artifact (arrows), (U) uterus, (B) bladder.

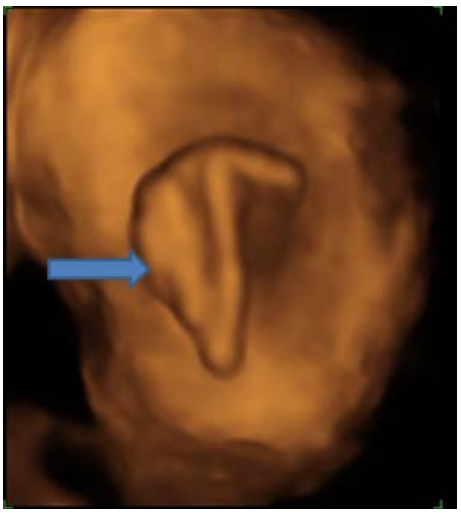


Figure 3. Three dimensional ultrasound image, elegantly demonstrates intra bladder IUD and its calcified component (arrow).

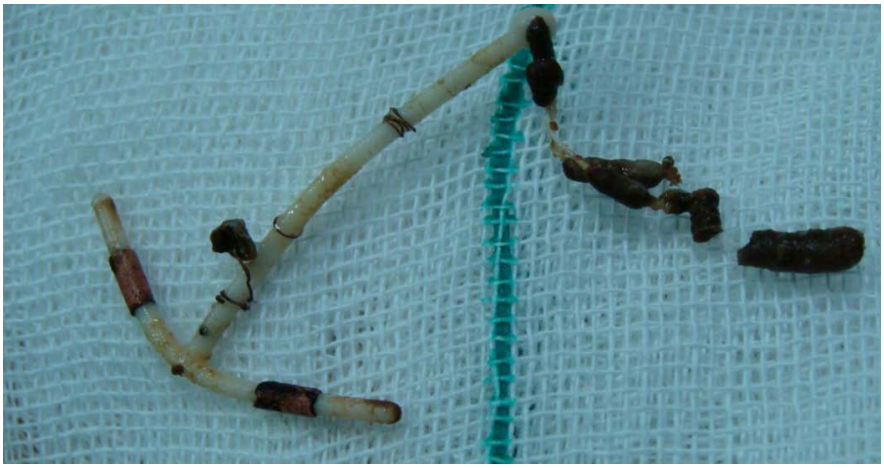


Figure 4. Photograph shows removed IUD by transuretral cystoscopy procedure.

3. Discussion

IUD is one of the most safe, effective and reversible contraceptive method popular all over the world. Though rare, it can cause uterine perforation and migration to adjacent organs such as bowels, bladder, peritoneum, omentum, adnexa and iliac vein [8] [9] [10] [11]. The exact mechanism of uterine perforation is yet to be known. Probable factors that can influence the risk of uterine perforation are the time of insertion, insertion technique, congenital anomalies, infections, thinning of the uterine wall and hypoestrogenemia in the lactation and postpartum periods and anatomy of the cervix and uterus [12] [13] [14]. Perforation of the uterine by an IUD may basically occur via two mechanisms [15]. First, perforation at the time of placement primarily, called misplacement, which can present with bleeding, acute pelvic pain, or lost thread. IUD applicator's experience and placement by specialists is an extremely important factor in prevention of misplacement, supported by many authors [16] [17] [18] [19]. Second, gradual and spontaneous perforation of the uterine after a long time since IUD insertion called migration, with late development of symptoms or being asymptomatic [20]. Forceful contractions of the uterine because of sexual stimulation or delivery, genital or bladder trauma, spontaneous irregular bladder contractions, bowel movements, and inflammatory effect of the IUD itself accompanied by the aforementioned risk factors, may be the mechanism of migration.

Kassab and Audra *et al.* collected 165 cases of uterine perforation in a literature review in 1999. Transvesical migration with or without calculi formation was seen in about 90 cases [21]. Although, the commonly asymptomatic nature of perforation supports the fact that the true incidence of perforation is most likely higher than what is reported in the literature.

Transvesical migration of the IUD is either within the bladder lumen or embedded in the bladder wall [22]. Most patients with intra bladder IUD are symptomatic with recurrent or persistent UTI and its symptoms, being the most common presentation [23]. Kart *et al.* reported a case of a 44 year old woman with transvesical migration of IUD who presented with dysuria and intermittent

hematuria [19]. Gunbey *et al.* reported a case of a 46 year old woman with IUD partially embedded in the wall of the cervix and extending into the bladder lumen, presented with lower abdominal pain lasting for 2 years [24]. The patient in our case presented with recurrent LUTS, lower abdominal pain and UTIs. The coexistence of pregnancy and misplaced or migrated IUDs should be evaluated first by transabdominal or transvaginal sonography, especially in cases with amenorrhoea. Otherwise, initial investigation via plain abdominopelvic radiography may be of choice. In our case, the patient had a history of bilateral TL. Therefore, we initially performed a plain abdominopelvic radiography and calcified string of the IUD was visible in the pelvic region. Then sonographic images revealed a dense intra bladder stone like structure with metallic artifact consistent with a calculus IUD. Also, three dimensional ultrasound imaging showed intra bladder IUD and its calcified components. After pregnancy has been ruled out, the ideal and least expensive method for detecting a misplaced or migrated IUD is plain abdominopelvic radiography since all IUDs are radio-opaque [25]. Sonography is a useful method in the detection of patients with a suspected intra bladder IUD. IUDs misplaced in the abdominal cavity can be evaluated by CT scan for almost exact localization [5]. All transvesical migrated IUDs must be removed even if they are symptomless, to prevent bladder rupture and calculi formation [22]. Transvesical migrated IUDs could be removed by cytoscopic removal or by open suprapubic cystotomy [26]. Removal of the IUD and stone fragments with cystoscopic procedure after laser lithotripsy seems to be the least invasive and adequate treatment modality. In our case a cystoscopic examination confirmed the sonographic findings of a calculus IUD implanted in the left anterior wall of the bladder. Holmium laser lithothripsy was performed then allowed removal of a copper-T IUD leaving no complications. Open suprapubic cystotomy is performed at centres which do not have cystoscopic facilities and also intra bladder IUDs that could not be removed with cystoscope. We suggest that in our case the uterine perforation did not occur at the time of IUD placement, because the patient became pregnant around 3 years after the insertion of the IUD. Soft consistency of uterus in pregnancy, strong uterine contractions in delivery, and uterine contractions due to sexual intercourse, genital and bladder trauma, inflammatory effect of the IUD itself after pregnancy may be the cause of IUD migration in our case. Hence in similar cases with unexpected pregnancy, persistent or recurrent UTI and LUTS and intra bladder calculus should raise the suspicion for uterine perforation and transvesical IUD migration. Whenever suspected, evaluation could be done using plain abdominopelvic radiography. Nevertheless, CT scan could be used whenever sonography cannot detect the IUD and exact localization is required.

4. Conclusion

Follow up visits and examination immediately after IUD insertion and periodically, could prevent IUD misplacement, migration and other complications. Unexpected pregnancy, LUTS, persistent or recurrent UTI, intra bladder calcu-

lus should raise the suspicion for uterine perforation and transvesical IUD migration. Early sonography for evaluation of recurrent or persistent UTI is recommended. Removal of the IUD and stone fragments with cystoscopic procedure after laser lithotripsy seems to be the least invasive and adequate treatment modality.

References

[1] Mosher, W.D. and Pratt, W.F. (1990) Contraceptive Use in the United States, 1973-88. *Patient Education and Counseling*, **16**, 163-172. https://doi.org/10.1016/0738-3991(90)90092-Y

[2] Oruc, S., Vatansever, H.S., Karaer, O., Eskicioglu, F. and Narlikuyu, B. (2005) Changes in Distribution Patterns of Integrins in Endometrium in Copper T380 Intrauterine Device Users. *Acta Histochemica*, **107**, 95-103. https://doi.org/10.1016/j.acthis.2005.01.001

[3] Tuncay, Y.A., Tuncay, E., Guzin, K., Ozturk, D., Omurcan, C. and Yucel, N. (2004) Transuterine Migration as a Complication of Intrauterine Contraceptive Devices: Six Case Reports. *The European Journal of Contraception & Reproductive Health Care*: *The Official Journal of the European Society of Contraception*, **9**, 194-200. https://doi.org/10.1080/13625180400007165

[4] el-Diasty, T.A., Shokeir, A.A., el-Gharib, M.S., Sherif, L.S. and Shamaa, M.A. (1993) Bladder Stone: A Complication of Intravesical Migration of Lippes Loop. *Scandinavian Journal of Urology and Nephrology*, **27**, 279-280. https://doi.org/10.3109/00365599309181267

[5] Sinha, M., Gupta, R. and Tiwari, A. (2013) Minimally Invasive Surgical Approach to Retrieve Migrated Intrauterine Contraceptive Device. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, **2**, 147-151.

[6] Nitke, S., Rabinerson, D., Dekel, A., Sheiner, E., Kaplan, B. and Hackmon, R. (2004) Lost levonorgestrel IUD: Diagnosis and Therapy. *Contraception*, **69**, 289-293. https://doi.org/10.1016/j.contraception.2003.11.017

[7] Heinberg, E.M., McCoy, T.W. and Pasic, R. (2008) The Perforated Intrauterine Device: Endoscopic Retrieval. *JSLS*: *Journal of the Society of Laparoendoscopic Surgeons*, **12**, 97-100.

[8] Ozcelik, B., Serin, I.S., Basbug, M., Aygen, E. and Ekmekcioglu, O. (2003) Differential Diagnosis of Intra-Uterine Device Migrating to Bladder Using Radiographic Image of Calculus Formation and Review of Literature. *European Journal of Obstetrics, Gynecology, and Reproductive Biology*, **108**, 94-96. https://doi.org/10.1016/S0301-2115(02)00240-3

[9] Schoenfeld, A., Pardo, J., Engelstein, D., Ovadia, J. and Servadio, C. (1991) Bladder Perforation by an Intrauterine Device. *Journal of Clinical Ultrasound*: *JCU*, **19**, 175-177. https://doi.org/10.1002/jcu.1870190310

[10] McNamara, M., Kennan, N. and Buckley, A.R. (1985) Copper-7 Perforation of the Uterus and Urinary Bladder with Calculus Formation—Sonographic Demonstration. *The British Journal of Radiology*, **58**, 558-559. https://doi.org/10.1259/0007-1285-58-690-558

[11] Guvel, S., Tekin, M.I., Kilinc, F., Peskircioglu, L. and Ozkardes, H. (2001) Bladder Stones around a Migrated and Missed Intrauterine Contraceptive Device. *International Journal of Urology*, **8**, 78-79. https://doi.org/10.1046/j.1442-2042.2001.00249.x

[12] Farmer, M. and Webb, A. (2003) Intrauterine Device Insertion-Related Complications: Can They Be Predicted? *The Journal of Family Planning and Reproductive Health Care*, **29**, 227-231. https://doi.org/10.1783/147118903101197854

[13] Behtash, N., Akhavan, S. and Mokhtar, S. (2010) Pelvic Mass Due to Transmigrated IUD. *Acta medica Iranica*, **48**, 125-126.

[14] Boortz, H.E., Margolis, D.J., Ragavendra, N., Patel, M.K. and Kadell, B.M. (2012) Migration of Intrauterine Devices: Radiologic Findings and Implications for Patient Care. *Radiographics*, **32**, 335-352. https://doi.org/10.1148/rg.322115068

[15] Zakin, D., Stern, W.Z. and Rosenblatt, R. (1981) Complete and Partial Uterine Perforation and Embedding Following Insertion of Intrauterine Devices. II. Diagnostic Methods, Prevention, and Management. *Obstetrical & Gynecological Survey*, **36**, 401-417. https://doi.org/10.1097/00006254-198108000-00001

[16] Atakan, R.H., Kaplan, M. and Ertrk, E. (2002) Intravesical Migration of Intrauterine Device Resulting in Stone Formation. *Urology*, **60**, 911. https://doi.org/10.1016/S0090-4295(02)01883-6

[17] Hoscan, M.B., Kosar, A., Gumustas, U. and Guney, M. (2006) Intravesical Migration of Intrauterine Device Resulting in Pregnancy. *International Journal of Urology*, **13**, 301-302. https://doi.org/10.1111/j.1442-2042.2006.01291.x

[18] Guner, B., Arikan, O., Atis, G., Canat, L. and Caskurlu, T. (2013) Intravesical Migration of an Intrauterine Device. *Urology Journal*, **10**, 818-820.

[19] Kart, M., Gulecen, T., Ustuner, M., Ciftci, S., Yavuz, U. and Ozkurkcugil, C. (2015) Intravesical Migration of Missed Intrauterine Device Associated with Stone Formation: A Case Report and Review of the Literature. *Case Reports in Urology*, **2015**, Article ID: 581697. https://doi.org/10.1155/2015/581697

[20] Eke, N. and Okpani, A.O. (2003) Extrauterine Translocated Contraceptive Device: A Presentation of Five Cases and Revisit of the Enigmatic Issues of Iatrogenic Perforation and Migration. *African Journal of Reproductive Health*, **7**, 117-123. https://doi.org/10.2307/3583296

[21] Kassab, B. and Audra, P. (1999) The Migrating Intrauterine Device. Case Report and Review of the Literature. *Contraception, Fertilite, Sexualite*, **27**, 696-700.

[22] Kandıralı, E., Topcuoglu, M.A., Semerciöz, A. and Metin, A. (2008) Double Intrauterine Device: Presented with Protruding Urethral Stone. *Marmara Medical Journal*, **21**, 61-63.

[23] Dietrick, D.D., Issa, M.M., Kabalin, J.N. and Bassett, J.B. (1992) Intravesical Migration of Intrauterine Device. *The Journal of Urology*, **147**, 132-134.

[24] Gunbey, H.P., Sayit, A.T., Idilman, I.S. and Aksoy, O. (2014) Migration of Intrauterine Devices with Radiological Findings: Report on Two Cases. BMJ Case Reports 2014.

[25] Bozkurt, M., Yumru, A.E., Coskun, E.I. and Ondes, B. (2011) Laparoscopic Management of a Translocated Intrauterine Device Embedded in the Gastric Serosa. *JPMA The Journal of the Pakistan Medical Association*, **61**, 1020-1022.

[26] Hick, E.J., Hernandez, J., Yordan, R., Morey, A.F., Aviles, R. and Garcia, C.R. (2004) Bladder Calculus Resulting from the Migration of an Intrauterine Contraceptive Device. *The Journal of Urology*, **172**, 1903. https://doi.org/10.1097/01.ju.0000142135.94531.bb

Call for Papers

Case Reports in Clinical Medicine

ISSN Print: 2325-7075 ISSN Online: 2325-7083
http://www.scirp.org/journal/crcm

Case Reports in Clinical Medicine (CRCM) is a monthly open access journal. The goal of this journal is to provide a platform for scientists and academicians all over the world to promote, share, and discuss various new issues and developments by publishing case reports in all aspects of Clinical Medicine.

Case Reports in Allergy and Clinical Immunology
Case Reports in Cancer Research and Clinical Oncology
Case Reports in Clinical Anaesthesiology
Case Reports in Clinical Anatomy
Case Reports in Clinical and Applied Thrombosis/Hemostasis
Case Reports in Clinical and Experimental Allergy
Case Reports in Clinical and Experimental Dermatology
Case Reports in Clinical and Experimental Hypertension
Case Reports in Clinical and Experimental Immunology
Case Reports in Clinical and Experimental Medicine
Case Reports in Clinical and Experimental Metastasis
Case Reports in Clinical and Experimental Nephrology
Case Reports in Clinical and Experimental Ophthalmology
Case Reports in Clinical and Experimental Optometry
Case Reports in Clinical and Experimental Otorhinolaryngology
Case Reports in Clinical and Experimental Pathology
Case Reports in Clinical and Experimental Pharmacology and Physiology
Case Reports in Clinical and Molecular Allergy
Case Reports in Clinical and Translational Oncology
Case Reports in Clinical Anesthesia
Case Reports in Clinical Apheresis
Case Reports in Clinical Autonomic Research
Case Reports in Clinical Biochemistry and Nutrition
Case Reports in Clinical Biomechanics
Case Reports in Clinical Cardiology
Case Reports in Clinical Case Studies
Case Reports in Clinical Child Psychology and Psychiatry
Case Reports in Clinical Chiropractic
Case Reports in Clinical Densitometry
Case Reports in Clinical Effectiveness in Nursing
Case Reports in Clinical Endocrinology and Metabolism
Case Reports in Clinical Epidemiology
Case Reports in Clinical Forensic Medicine
Case Reports in Clinical Gastroenterology and Hepatology
Case Reports in Clinical Genetics
Case Reports in Clinical Haematology
Case Reports in Clinical Hypertension
Case Reports in Clinical Imaging
Case Reports in Clinical Immunology
Case Reports in Clinical Implant Dentistry and Related Research
Case Reports in Clinical Interventions in Aging
Case Reports in Clinical Laboratory Analysis
Case Reports in Clinical Linguistics & Phonetics
Case Reports in Clinical Lipidology
Case Reports in Clinical Microbiology and Antimicrobials
Case Reports in Clinical Microbiology and Infection
Case Reports in Clinical Microbiology and Infectious Diseases
Case Reports in Clinical Molecular Pathology
Case Reports in Clinical Monitoring and Computing
Case Reports in Clinical Neurology and Neurosurgery
Case Reports in Clinical Neurophysiology
Case Reports in Clinical Neuropsychology
Case Reports in Clinical Neuroradiology
Case Reports in Clinical Neuroscience
Case Reports in Clinical Nursing
Case Reports in Clinical Nutrition
Case Reports in Clinical Obstetrics and Gynaecology
Case Reports in Clinical Oncology and Cancer Research
Case Reports in Clinical Oral Implants Research
Case Reports in Clinical Oral Investigations
Case Reports in Clinical Orthopaedics and Related Research
Case Reports in Clinical Otolaryngology
Case Reports in Clinical Pathology
Case Reports in Clinical Pediatric Emergency Medicine
Case Reports in Clinical Periodontology
Case Reports in Clinical Pharmacology & Toxicology
Case Reports in Clinical Pharmacy and Therapeutics
Case Reports in Clinical Physiology and Functional Imaging
Case Reports in Clinical Practice and Epidemiology in Mental Health
Case Reports in Clinical Psychology and Psychotherapy
Case Reports in Clinical Psychology in Medical Settings
Case Reports in Clinical Radiology
Case Reports in Clinical Rehabilitation
Case Reports in Clinical Research and Regulatory Affairs
Case Reports in Clinical Research in Cardiology
Case Reports in Clinical Respiratory
Case Reports in Clinical Rheumatology
Case Reports in Clinical Simulation in Nursing
Case Reports in Clinical Sleep Medicine
Case Reports in Clinical Techniques in Small Animal Practice
Case Reports in Clinical Therapeutics
Case Reports in Clinical Toxicology
Case Reports in Clinical Transplantation
Case Reports in Clinical Trials
Case Reports in Clinical Ultrasound
Case Reports in Clinical Virology
Case Reports in Complementary Therapies in Clinical Practice
Case Reports in Consulting and Clinical Psychology
Case Reports in Contemporary Clinical Trials
Case Reports in Controlled Clinical Trials
Case Reports in Diabetes Research and Clinical Practice
Case Reports in Evaluation in Clinical Practice
Case Reports in Fundamental & Clinical Pharmacology
Case Reports in Hematologic Diseases
Case Reports in Hereditary Cancer in Clinical Practice
Case Reports in Human Psychopharmacology: Clinical and Experimental
Case Reports in Innovations in Clinical Neuroscience
Case Reports in Laboratory and Clinical Medicine
Case Reports in Neurophysiologie Clinique/Clinical Neurophysiology
Case Reports in Nutrition in Clinical Practice
Case Reports in Pacing and Clinical Electrophysiology
Case Reports in Psychiatry in Clinical Practice
Case Reports in Therapeutics and Clinical Risk Management
Case Reports in Veterinary Clinical Pathology
Case Reprots in Obstetrics and Gynecology

Website and E-Mail

http://www.scirp.org/journal/crcm

Email: crcm@scirp.org

What is SCIRP?

Scientific Research Publishing (SCIRP) is one of the largest Open Access journal publishers. It is currently publishing more than 200 open access, online, peer-reviewed journals covering a wide range of academic disciplines. SCIRP serves the worldwide academic communities and contributes to the progress and application of science with its publication.

What is Open Access?

All original research papers published by SCIRP are made freely and permanently accessible online immediately upon publication. To be able to provide open access journals, SCIRP defrays operation costs from authors and subscription charges only for its printed version. Open access publishing allows an immediate, worldwide, barrier-free, open access to the full text of research papers, which is in the best interests of the scientific community.

- High visibility for maximum global exposure with open access publishing model
- Rigorous peer review of research papers
- Prompt faster publication with less cost
- Guaranteed targeted, multidisciplinary audience

Scientific Research Publishing

Website: http://www.scirp.org

Subscription: sub@scirp.org

Advertisement: service@scirp.org