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CHAIRMAN'S COLUMN



New year greetings to all !!

It's been a fantastic year 2018 for all of us at Royal Care and I take this opportunity to thank each and every one for their immense contribution towards achieving our common goals.

This first quarter of 2019, we will be equipped with state of the art high precision radiotherapy system -TRUEBEAM RADIOTHERAPY SYSTEM, thus completing the entire spectrum of diagnostic oncology to therapeutic facilities under one roof at Royal Care. We will also be adding full field digital mammogram and SPECT in this quarter. We will keep adding up newer generation diagnostic equipments and other facilities at par with Western/US healthcare systems.

Institute of Critical Care Medicine is fully operational and happy to share that we got approval for starting fellowship courses in critical care medicine (IDCCM). The healthcare environment is changing in the Indian subcontinent and its time for us to face the challenges and move forward.

Regards Dr. K. Madeswaran Founder Chairman





"The way to get started is to quit talking and begin doing".

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- Walt Disney

Relentless hard work by everyone on our team has reaped the dividend, within two years of initiation of healthcare services, royal care hospital has been ranked 6th best hospital in Coimbatore. We shall strive to reach the top 3 within the next couple of years.

The quality control team has done all preparation and has applied for NABH certification already. The inspection shall happen soon and when awarded, Royal care hospital shall be one of the very few hospitals that has done so within such short time of inauguration.

The new ICU with paperless documentation has been inaugurated and it is one of a kind facility in entire Tamil nadu. This unit has received various accolades from doctors and media equally. This shall be a trend setter for the future units that shall come up.

Our hospital doctors and staff participated in various camps, health show programmes, gave talks on the radio for the benefit of the public. Courses were conducted on cardiotocography and a work shop was conducted on parent craft. In this edition, we have special articles on urology, cardio thoracic surgery, Head and neck surgery and nephrology. A special note on our CTVS team who performed the first surgery on a Jehovah witness in Coimbatore without using blood products. Congratulations to them.

We also welcome the new consultants who have joined the Royal care family.



From The Editor's Pen..

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ANOMALOUS ORIGIN OF LEFT CORONARY ARTERY FROM THE PULMONARY ARTERY WITH MITRAL INSUFFICIENCY IN ADULT- A CASE REPORT

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Abstract

Anomalous left coronary artery arising from the pulmonary artery is a rare coronary anomaly, Seldom seen in adults along with severe mitral insufficiency, severe pulmonary hypertension and ongoing heart failure .We report a case of a 35yrs old female who underwent takedown of the anomalous left coronary artery with mitral valve replacement and an arterial bypass graft to the Left anterior descending artery and Obtuse marginal branch.

Keywords - ALCAPA, MR, CABG, LIMA

Introduction

Anomalous left pulmonary artery arisisng from pulmonary artery (ALCAPA) is a common congenital coronary anomaly seen in neonates and seldom seen in adults, reported first in 1908 also known as Bland – White- Garland syndrome (1). The incidence is about 0.25 to 0.5% of congenital heart diseases .(2) The natural history is short that majority of the neonates die by one month and only a few survive more than a year.(3) When seen in adulthood, the presentation is with severe ischemic heart failure and in a decompensated state.

Case report

A 35yrs old lady presented to us with class IV dyspnea and orthopnea of 6 months duration. She had no angina. Clinical examination showed tachypnoea, elevated Jugular venous pressure, and bilateral pitting pedal edema. Her pulse rate was 112 bpm,blood pressure was 100/60 mm Hg and a grade 4/6 pansystolic murmur at the apex. Abdominal examination revealed a tender hepatomegaly. ECG showed left ventricular hypertrophy with left bundle branch block. Chest X-Ray revealed cardiomegaly with congested lung fields. Echocardiogram revealed severe global hypokinesia with EF of 30% and severe mitral regurgitation(MR) due to fixed posterior leaflet, and an eccentric anterior jet. There was no chordal rupture nor annular dilatation. She had mild to moderate tricuspid regurgitation (TR) and severe pulmonary artery hypertension(PAH)and a dilated coronary sinus with no anomalous systemic venous drainage into it. The septal collaterals were so abundant that it mimicked a Swiss cheese Ventricular septal defect (VSD). She had severe left ventricular dysfunction. Biochemical analysis showed an elevated Brain natriuretic peptide level of 3123 and a serum creatinine of 1.3mg/dl, other parameters and hemogram was normal. Her heart failure was managed medically prior to further investigations. Coronary angiogram revealed ectatic and tortuous right coronary system with opacification of the left coronary draining into the pulmonary artery in the levophase. There were no flow limiting lesions in both coronary systems. Right heart study revealed severe PAH with a Qp/Qs of 1.6. and oxygen step up of 20% at the level of main pulmonary artery

The plan of corrective surgery was mitral valve replacement with coronary artery grafting and takedown of ALCAPA. Invasive lines include a radial arterial line, IJV line with a trilumen catheter and an additional femoral arterial line for insertion of IABP catheter if needed.

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After standard midline sternotomy, pericardiotomy was done more towards the right to facilitate a patch if needed. Diagnosis of ALCAPA was confirmed . The left and right coronary systems were grossly dilated and tortuous with abundant collateral coronary arteries connecting left and right systems. LIMA and great saphenous vein were harvested. Both pulmonary artery branches were looped to prevent coronary steal .Standard Cardiopulmonary bypass was instituted using bicaval cannulation. Blood cardioplegia was delivered through aortic root and left coronary ostium after pulmonary arteriotomy and repeated every 20 minutes. Core was cooled to 28oc. Total bypass time was 167 minutes and cross clamp time was 124 minutes. The pulmonary artery was transected and the origin of the left main from the posterior sinus was viewed. Dissection was carried out to isolate the left main that was embedded in fat. Direct transfer to the aorta was contemplated and as the LMCA was too short and originating more laterally it was ligated and transfixed with 4-0 polypropylene sutures. PA was reconstructed. LIMA to LAD was done using 8-0 polypropylene suture and a reversed saphenous venous graft was constructed to the obtuse marginal branch. Mitral valve was approached through Sondergard's groove , the posterior mitral leaflet was densely tethered to the ventricular muscle with deficient leaflet substance. hence replacement of the mitral valve was done with preserving subvalvar apparatus using 25mm TTK Chitra mechanical prosthesis and interrupted 2-0 pledgetted polyester sutures. De-airing was done in the standard way and decalmped. Heart reverted in sinus rhythm, proximal anastomosis was constructed on a partial clamp. She was weaned off CPB uneventfully with a systemic pressure of 107/54 mmHq, RA pressure of 6mm Hg. She needed an inotropic support of Dobutamine at 5mcg/kg/min. Post CPB oximetry of RA and PA samples revealed no significant stepup. She was extubated in 8hrs time and inotropes were weaned off after 24 hours and shifted to wards and discharged on day five.

Discussion

ALCAPA is the most common coronary anomaly seen in children with an incidence of 0.25-0.5%. (2) It is rarely seen in adults and survival with severe MR and heart failure is anecdotal. Soon after the establishment of pulmonary circulation after birth there is a left to right shunt from the right coronary system to the pulmonary trunk with an associated coronary steal and myocardial ischemia (4). Two types have been described, infantile and adult. Coronary collaterals are poorly developed in the infantile group as compared to the adults. Left ventricular muscle is in jeopardy leading to ischemic complications like mitral regurgitation and endocardial fibrosis There is a 90% chance of sudden cardiac death among the adults(5)

Coronary collaterals are well developed in adults with change in pattern of coronary circulation. In our case, the collaterals were so abundant which mimicked Swiss cheese VSD on colour Doppler. MR is due to chronic ischemia of the papillary muscles or infarction associated with annular dilatation. Diagnosis is primarily by Echocardiography in children and in adults it is often missed due to varied presentation. Our patient had a working diagnosis of cardiomyopathy with severe MR until the coronary anatomy was studied prior to surgical intervention. Severe pulmonary artery hypertension render adults as poor surgical candidates.

Diagnosis of ALCAPA is an indication for surgery(6). Establishment of a two coronary circulation is a well-documented technique in children and adults(7,8). Surgical options in children include coronary transfer and intrapulmonary tunnel as described by Takeuchi (9). In adults various techniques have been enunciated like (a) percutaneous coil embolization of anomalous coronary to reduce the shunt and steal (10). (b) Ligation of the left main with a graft to the LAD and circumflex system (c) Direct transfer of the left coronary button to Aorta depends on the sinus of origin on the PA (11) In our patient, a direct transfer was tried with mobilization of the coronary button and as it was technically not feasible it was ligated and transfixed with a graft to LAD and Marginal branch.

Surgical results of children operated early are excellent with a low mortality O-16% (12). Long term results at 20 years is 94.8% (13). Mitral regurgitation normalizes in children following surgical reestablishment of dual coronary system (14). Adults carry a higher operative mortality and long term results are not available. As documented with ischemic MR, a moderate to severe regurgitation needs intervention of the mitral valve. Preference of a mitral repair over replacement has not been well documented in an ALCAPA setting.



We preferred to replace the valve with preservation of sub valvar apparatus in addition to coronary grafting and takedown of ALCAPA. Our post-operative echocardiogram revealed regression of pulmonary artery pressures with significant improvement in global left ventricular function and left ventricular dimensions.

References

- 1. Bland FE, White PD, Garland J. Congenital anomalies of the coronary arteries: report of an unusual case associated with cardiac hypertrophy. Am Heart J. 1933;8:787-801
- 2. Wesselhoeft H, Fawcett JS, Johnson AL. Anomalous origin of the left coronary artery from the pulmonary trunk: its clinical spectrum, pathology and pathophysiology based on a review of 140 cases with seven further cases. Circulation. 1968;38:403-25
- 3. Walid Ben Ali, Oliver Metton. Anomalous origin of left coronary artery from the pulmonary artery: late results with special attention to the mitral valve. Eur. J. cardiothoracic surg. 2009;36:244-49
- 4. Backer CL, Stout MJ,Zales VR et.al. Anomalous Origin of the left coronary. A 20 year review of surgical management. J.Thorac Cardiovasc. Surg. 1992;103:1049-58
- 5. Talwar S, Kiran U, Nath MP, Anomalous origin of left coronary from pulmonary artery in adult; Perioperative concerns, Indian J Thorac Cardiovasc Surg 2010;26:157-61
- 6. Huddleston CB,Blazer DT, Mendeloff EN, Repair of anomalous left main coronary artery arising from the pulmonary artery in infants: long term impact on the mitral valve. Ann. Thorac Surg. 2001;71:1985-9
- 7. Neches WH, Mathews RA, ParkSC et.al., Anomalous origin of the left coronary artery from the pulmonary artery, A new method of surgical repair. Circulation . 1974;50:582-7
- 8. Alexi-Meshkishivili V, Berger F, Weng et.al, Anomalous origin of left coronary artery from pulmonary artery in adults. J card Surg.1995;10:309-15
- 9. Takeuchi S, Imamura H, Katsumoto K, New surgical method for repair of anomalous left coronary artery from pulmonary artery. J.Thorac cardiovasc.surg. 1979;78:7-11
- 10. Collins N, Colman J Benson L Hansen M, Merchant M, Horlick E, Successful percutaneous treatment of anomalous left coronary artery from pulmonary artery. Int J Cardiol. 2007;122:e29-31

CONGRATULATIONS TO CTVS TEAM Cardiac surgery without blood transfusion

53 yrs old lady (An Orthodox Christian - Jehovah's, who do not admit blood transfusion nor allow their blood to be circulated in machines like Heart Lung and Dialysis machines) presented to our hospital for cardiac ailment and needed coronary artery bypass surgery (CABG). It was a challenge to attempt it as the procedure and post-operative period needs to be tide over without blood transfusion. She was well prepared and taken up for CABG and on successful completion, discharged on day four of surgery with a total loss of only 200ml blood.

Royal Care Hospital is proud to announce that such a procedure is the first of its kind in this subset of patients in Coimbatore. Worldwide reports claim that such surgeries in this subset of population is risky and carries a significant mortality.

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ARGE MULTILOCULAR CYSTIC RENAL MASSES – DILEMA



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Background:

Cystic renal neoplasms of the kidney can be benign or malignant. Multicystic nephroma (MCN) represents a rare benign cystic lesion of the kidney, which usually presents as a unilateral multicystic renal mass without solid elements. According to the World Health Organization (WHO) classification of the renal neoplasms, it is grouped along with mixed epithelial–stromal tumor of the kidney.

Introduction

Multicystic nephroma (MCN) is a rare, benign cystic neoplasm of the kidney. It was first described in 1892 as cystic adenoma of the kidney and over 200 cases have been reported in the literature so far.[1] Its etiology and histogenesis is debatable, and in the past they were considered to be developmental lesions with malignant potential. The name multicystic nephroma was first proposed in 1951 and later modified and further subdivided into cystic nephroma and cystic partially differentiated nephroma depending on the absence and presence of blastemal element, respectively.[2]

MCN is an uncommon, benign cystic lesion of the kidney with bimodal age distribution, occurring in both infants and adult population. Although it has been described in neonates, MCN is more commonly seen in the age group of 2-4 years (of which 73% are in males). Below 4 years of age, male to female ratio is 3:1 and boys are affected more than girls. In adults, it is seen in the 4th–6th decade with its male to female ratio being 1:8.[3–6] Adultonset cystic nephroma is histogenetically and morphologically different from pediatric cystic nephroma.[7–9] According to the World Health Organization (WHO) classification of renal neoplasms, MCN is grouped with the mixed epithelial and stromal tumors (MEST).[10] The term renal epithelial and stromal tumor (REST) can be used to encompass both MCN and MEST.[11]

Surgical intervention is the effective method at present to exclude malignant cystic lesions of kidney. However, nephron-sparing surgery can be an option depending on the site and size of the lesion.

Materials, Methods And Result

We report one such case, 48 year old female

patient, who presented to us with incidentally detected Cystic Left Renal mass lesion of 12.1cm x 9.7cm x 7.1cm, suggestive of Left Cystic RCC (On USG). Computed tomography (CECT) scan suggested a Bosniak III type cyst, of size 10.5 x 9.6 x 9.0 cms in the upper pole of left kidney causing displacement of left kidney, closely abutting left PCS. No evidence of extracapsular extension or infiltration into adjacent organs. Left renal vein and IVC were normal.

With high degree of suspicion for malignancy, patient underwent Laproscopic Left Radical Nephrectomy.

Histopathologically report turned out to be Multilocular cystic Nephroma. Microscopically, section from lesion showed well encapsulated, circumscribed, multiloculated cystic lesion. Cysts lined by flat/ cuboidal cells with occasional hobnailings separated by fibrous stroma. No nuclear atypia, invasion or blastemal elements noted.

Post-operative period was uneventful and patient recovered well and discharged in stable condition.

Discussion

MCN has at least 20–25 synonyms, which include multilocular cystic renal tumor, benign multilocular cystic nephroma, polycystic nephroblastoma, and so on.

The pathogenesis of MCN is controversial and its classification is confusing. There are numerous proposed theories indicating the etiology as a developmental defect.[12] It has also been postulated that it could be neoplastic in origin, probably arising from the ureteral bud.[12] Various pathological criteria have been proposed in the past to differentiate and classify this entity.

First diagnostic criteria were formulated in 1951[2] and later modified in 1956[13] and include the following: (a) lesion must be multilocular, (b) the cyst for the most part lined by epithelium, (c) the cyst must not communicate with renal pelvis, (d) the residual renal tissue should be essentially normal, except for pressure atrophy, and finally (e) no fully developed nephrons.

The terminology was modified in 1989, which emphasized neoplastic rather than developmental origin.[9] It was broadly segregated into MCN and cystic partially differentiated nephromablastoma.[9] In MCN, multiple cysts showed septa consisting of mature, welldifferentiated tubules among the fibrous tissue. Lesions which were predominately cystic without nodular or solid components and containing blastemal elements were noted as cystic partially differentiated nephroma, especially those occurring in the age group less than 2 years. Further subclassification of cystic partially differentiated nephroma was attempted to predict the aggressiveness of this entity based on the content of septal stromal elements, and presence of more than 50% of mature septal element was classified as grade I and less than 50% as grade II.[9]

Patients usually present with nonspecific symptoms. Abdominal pain, hematuria, and urinary tract infection are common in adults. Hematuria can be seen in all age groups and is thought to be due to extension of tumor into the renal pelvis.[14,15] Loin pain was the commonest presentation in this study and only two patients presented with hematuria. Presentation can sometimes be with severe colicky abdominal pain due to spontaneous rupture of the cyst, which can lead to a clinical diagnosis of urinary stone disease. In the present series, case no. 6 had a

Ultrasound	Multiple anechoic spaces traversed by thin septa No solid elements A beak or "claw" of normal renal parenchyma around the periphery of a well-defined mass
Computed tomography	Splaying or displacement of the renal collecting system A sharpy circumscribed, multiseptated renal mass Attenuation value of the cyst contents equal to or slightly higher than that of water Septations enhance following contrast material administration, but contrast material does not accumulate within individual loculi
	Well defined margins Multicystic architecture Herniation into the renal collecting system
Magnetic resonance imaging	Low signal intensity of the tumor capsule
Angiography	Multilocular cystic renal tumors usually appear hypovascular
Pathology · macroscopic	Unilateral and solitary lesion - sharp interface with normal renal parenchyma
features	Multilocular cysts of varying sizes with thin septa and no solid elements
	Cysts are non-communicating with each other or the renal pelvis
	Cysts contain serous fluid
Pathology · microscopic features	Cysts lined by tubular epithelium which may be columnar or flat, and is characteristically "hobnail" in apearance Fibroblastic stroma between cysts
	Smooth and skeletal muscle tissue and undifferentiated metanephric bastema may be present

similar presentation and an intravenous urogram was nondiagnostic. It usually affects single kidney, although rarely bilateral MCN has been reported.[3,6,] Lower pole of kidney is the most favored site and the upper pole is the least favored; however, it can arise from any portion of the renal parenchyma. Interestingly, in the present series, upper pole was the most common site of presentation.

Distinct radiographic features have been described, but are not universally present in all cases. Ultrasound is often the first investigation used in evaluating abdominal masses, confirmed by CT scan. The sonographic findings depend on the amount of stromal tissue and size of locules. Cysts usually show up as hypoechoic lesions delineated by hyperechoic septae [Figure 1], and this feature can be suggestive of MCN but not diagnostic. The mass is often easily demonstrable at ultrasound, with an average diameter of approximately 10 cm. If the cysts are small, the mass may demonstrate internal scattered echoes but without distinct loculations.[3] Calcification has been described as a rare feature of MCN,[6] and curvilinear calcifications may be seen on ultrasound within the septa. Both needle-guided aspiration and color Doppler ultrasound have been proposed to help differentiate between benign and malignant multilocular cystic lesions.



Radiological and pathological features of multicystic nephroma

CT image features suggestive of MCN are multicystic architecture [Figure 2] with welldefined margins, enhancing septae and herniation into renal pelvis. This herniation often causes hematuria. However, it is interesting to note that the two cases in this series presenting with hematuria did not show any herniation in CT imaging. Calcification is rarely seen; occasionally, however, ossification can occur within septa or within the renal capsule. Central or small peripheral curvilinear calcifications can occasionally be seen at the edge of the herniated pelvic portion. In addition, dense calcium rings in multiple cysts have been reported.[4] If present, calcification may lead to confusion with malignancy.





Plain And Contrast Ct Images showing large multilocular cystic mass lesion of Left kidney.

Magnetic resonance imaging (MRI) is rarely indicated, but imaging features include usually hypointense signal on TI-weighted sequences (although this may vary) and hyperintense signal on T2-weighted sequences. Septa are usually hypointense on all sequences due to fibrous content.

The pathological features of the multilocular cystic renal neoplasms including cystic nephroma and cystic hamartoma, which show cystic growth pattern, are very difficult to separate radiographically. Fine needle aspiration cytology (FNAC) has been attempted in order to establish a preoperative diagnosis. Papanicolaou staining of the cyst fluid showed markedly atypical cells forming papillary clusters. However, though not very conclusive, low cellularity, absence of necrosis, and paucity of single cells should be viewed with suspicion of MCN. Hence not routinely done.



Cut section of multilocular cystic nephroma

It is widely believed that it is impossible to clinically or radiologically distinguish between the benign entity of MCN and renal cell carcinoma.

Although the MCN is not a pre-malignant condition, there are case reports of co-existing foci of renal cell carcinoma in the lining of the cyst wall. MCN is considered to be of benign nature in adults; tumor recurrences have been observed and whether these recurrences are related to the missed foci of malignant area or sarcomatous degeneration is not clear. These recurrences are more often seen after partial nephrectomy. So far, only four cases of local recurrences have been reported, all following partial nephrectomy. However, in a series of 24 patients who underwent partial nephrectomy for MCN, no recurrences were found after a mean follow-up of 39 months.[6] More recently, a case of percutaneous treatment has been described in treating MCN. Percutaneous endoscopic resection of a portion of the cyst protruding into the renal parenchyma was performed, and follow-up CT 4 weeks later revealed complete resolution. However, 3 years later, the authors reported failure with percutaneous approach.

Conclusion

Multilocular cystic nephroma is an uncommon cystic lesion of the kidney and should be considered in the differential diagnosis of malignant cystic renal tumors in both children and adults. Whilst it is important to consider the diagnosis of MCN for any multicystic mass, a definitive diagnosis can only be made following surgical treatment with total or partial nephrectomy depending on the size and location of the lesion in the kidney.

References

- Edmunds W. Cystic adenoma of the kidney. Trans Pathol Soc Lond. 1892;43:89–90.
- 2. Powell T, shackman R, Johnson HD. Multilocular cysts of kidney. Br J Urol. 1951;23:142–52. [PubMed]
- Madewell JE, Goldman SM, Davis CJ, Jr, Hartman DS, Feigin DS, Lichtenstein JE. Multilocular cystic nephroma: A radiographic-pathologic correlation of 58 patients. Radiology. 1983;146:309–21. [PubMed]
- Banner MP, Pollack HM, Chatten J, Witzleben C. Multilocular renal cysts: Radiologic-pathologic correlation. AJR Am J Roentgenol. 1981;136:239–47. [PubMed]
- Mukhopadhyay S, Valente AL, de la Roza G. Cystic Nephroma: A histologic and immunohistochemical study of 10 cases. Arch Path Lab Med. 2004;128:1404–11. [PubMed]
- Castillo OA, Boyle ET, Jr, Kramer SA. Multilocular cysts of kidney. A study of 29 patients and review of literature. Urology. 1991;37:156–62. [PubMed]
- Eble JN, Bonsib SM. Extensively cystic renal neoplasms: Cystic nephroma, cystic partially differentiated nephroblastoma, multilocular cystic renal cell carcinoma, and cystic hamartoma of renal pelvis. Semin Diagn Pathol. 1998;15:2–20. [PubMed]
- 8. Bisceglia M, Galliani CA, Senger C, Stallone C, Sessa A. Renal cystic diseases: A review. Adv Anat Pathol. 2006;13:26–56. [PubMed]
- Joshi VV, Beckwith JB. Multilocular cyst of the kidney (cystic nephroma) and cystic, partially differentiated nephroblastoma. Terminology and criteria for diagnosis. Cancer. 1989;64:466–79. [PubMed]
- 10. Bonsib SM. Cystic nephroma. Mixed epithelial and stromal tumor. In: Eble JN, Sauter G, Epstein JI, Sesterhenn IA, editors. World Health Organization classification of tumours. Pathology and genetics of tumors of the urinary system and male genital organs. Lyon: IARC Press; 2004. p. 76.
- Turbiner J, Amin MB, Humphrey PA, Srigley JR, De Leval L, Radhakrishnan A, et al. Cystic nephroma and mixed epithelial and stromal tumor of kidney: A detailed clinicopathologic analysis of 34 cases and proposal for renal epithelial and stromal tumor (REST) as a unifying term. Am J Surg Pathol. 2007;3I:489–500. [PubMed]
- 12. Sacher P, Willi UV, Niggli F, Stalmach T. Cystic Nephroma: A rare benign renal tumour. Paediatric Surg Int. 1998;13:197–9. [PubMed]
- 13. Boggs LK, Kimmelsteil P. Benign multilocular cystic nephroma: Report of two cases of multilocular cyst of kidney. J Urol. 1956;76:530–41. [PubMed]
- Bouhafs A, Cherradi N, Lamaalmi N, Beklachem R, Barahioui M. An unusual case of multicystic Nephroma with prominent renal pelvis involvement. Int J Urol. 2006;13:436–8. [PubMed]
- Kural AR, obek K, Ozbay G, Onder AU. Multilocular cystic Nephroma: An unusual presentation. Urology. 1998;52:897–9. [PubMed]

GLIMPSES



Chairman's Address @ Hospital 2nd Anniversary Celebration



Class on NABL



Parent Carft Workshop at Ardra Hall, Cbe



New ICU Inauguration



CME Programme - ICU

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Medical Camp @ Ooty



Medical Camp @ Pollachi



Ortho camp @ COSIEMA



Face to Face Programme World Heart Day 2018



CME @ Tirupur



CME Programme - PET CT



Breast Cancer Awareness @ Karunya University

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"Patients Meet" who underwent successful bronchial thermoplasty treatment



CME @ Karur



CME @ Palakkad



Cardiotocography Masterclass



Health Talk Programme @ Quantum KPR Mills

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ODONDOGENIC MYXOMA - A CASE REPORT AND LITERATURE REVIEW

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Introduction

The term odontogenic myxoma (OM) was first coined by Thoma and Goldman in 1947.(1) The reported incidence of this tumour is 0.04–3.7%. OM represents 3-6% of all odontogenic tumours and appears to originate from dental papilla, follicle, or periodontal ligament. According to the World Health Organization (WHO), OM is classified as benign tumour of mesenchymal or ectomesenchymal origin with or without odontogenic epithelium.(2)

OM is characterized grossly by mucoid or gelatinous greyish-white tissue.(3) Clinically, it is a slow-growing, expansile, painless tumour, which may cause root resorption, tooth mobility, bone expansion, cortical destruction and facial distortion. Most frequently, OM occur in the second or third decades of life, (4,5,6) and has a slight female predilection.

OM remains asymptomatic for long time and hence the lesions may reach a considerable size before patient perceives its existence and seeks medical attention. If the treatment is neglected, it can result in complications with an increased possibility of morphological and functional damage.

In this article, we present a case of OM occurring in the left maxilla of a 20-year-old man focusing on brief review of clinical, radiological, histological features and treatment options.

Case report

A 20 year old male patient presented with swelling of left cheek, left nasal block and pain over left cheek. He had swelling of left maxilla for five years which was slow growing. There was no history of trauma. Past medical and dental histories were non-contributory

Clinical examination revealed a firm, non-tender, large smooth swelling of left maxilla without any colour change in the skin. Endoscopic examination of left nasal cavity was completely blocked by an expansile mass of left maxillary



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sinus and the nasal septum was pushed to the right side. On intra-oral examination, a bony hard expansile swelling was present at left buccal as well as lingual vestibular region. The swelling extended from left lateral incisor to last molar tooth without any erythema or ulceration in the overlying mucosa. His eye movements were normal. The left first molar was loose. Rest of ENT, and systemic examinations were normal. No lymphadenopathy was observed.

Routine haematological investigations were normal. A contrast CT imaging showed expansile mass of 4.3 X 4.0 X 4.8 cm arising from left maxilla extending into left maxillary sinus, completely occupying left maxillary sinus. Fig 1.



Fig 1: CT scan of left maxillary OM

There was thinning, scalloping and irregularities of the overlying cortex with break of the overlying cortex at places. The roots of left maxillary molar teeth were seen along the lower part of the mass. There was widening of the left maxillary sinus. This mass lesion was extending into the left maxillary ostia and protruding into the left nasal cavity intending left middle and inferior turbinates. It was also protruding into the left ethmoid air cells.

He underwent enucleation of the left maxillary mass, curettage and removal of involved tooth through crevicular incision made from left upper incisor to second molar under general



anaesthesia. Angled sinus endoscopes was used to dissect tumour from difficult corners thereby the tumour mass was completelyremoved and sent for histology.

On macroscopic examination the tumour appeared soft and gelatinous. Histological examination of the specimen revealed benign neoplasm composed of spindle to stellate cells with moderate eosinophilic cytoplasm scattered in myxoid matrix. No increase in mitosis/nuclear atypia found. Focal areas of bony fragments and odontogenic epithelium within myxoid matrix were seen. These features were consistent with odontogenic myxoma.Fig 2



Fig 2 : Histopathological aspect of maxillary odontogenic myxoma

Follow up at eight months after the surgical procedure, there were no clinical signs of recurrence. The left maxillary swelling completely settled, the expanded medial wall of maxillary sinus remodelled and left nasal airway become patent. Patient was happy with cosmetic appearance of face and improvement in nasal breathing.

Discussion

OM is a rare tumour of the jaws, and generally occur in the second to fourth decades of life. OM involves the mandible more commonly than the maxilla. When the maxillary sinus is involved, the odontogenic myxomas often fill the entire antrum and expands it as is in our case. Displacement of teeth has been registered in 9.5% of the cases. OM most commonly were located in the premolar and molar areas of mandible and maxilla. In the present case, the lesion was located in the premolar and molar area of the left maxilla.

OM is generally a slow growing benign neoplasm. However, cases of rapidly growing, locally aggressive tumours showing local infiltration have been described.(6) The tumour is nonencapsulated and hence can cause significant infiltration into the adjacent medullar bone, which contribute to their high recurrence rate.(8)

Lesions of the maxilla have also been described as being more aggressive than those of the mandible. While the tumour expands, it remains asymptomatic for most patients. Ulceration of the overlying oral mucosa only occurs when the tumour interferes with dental occlusion. When present, the symptoms, as reported in the literature, can be classified according to their site of expansion as summarized by S. Nguyen et al.(9)

Palate and oral cavity

Mass Pain, erythema, oedema Unerupted or absent tooth Tooth displacement or mobility Malocclusion Paresthesia Bone perforation Facial or jaw asymmetry Trismus Dysphagia Dysphonia Erosion or ulceration of mucosa (rare)

Sinuses and nasal cavity

Nasal obstruction Sinus or paranasal pain Recurrent epistaxis Nasal discharge Sinusitis **Orbit** Exophthalmos Diplopia Lacrymal duct obstruction

Asymptomatic patients may delay the time of diagnosis and result in bigger expansion of the tumour before presentation. Our patient remained asymptomatic for long time before presented with pain, swelling of cheek and nasal obstruction.

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Numerous other lesions of the jaw are considered as differential diagnosis of OM such as simple cysts, ameloblastoma, odontogenic keratocyst, intraosseous haemangioma, osteosarcoma, metastatic carcinoma and giant cell granuloma.

The radiographic aspects of OM are markedly variable.(7) It can be unilocular. Often they show multilocular radiolucencies representative of honey comb or soap bubble appearance. They may appear as strings of tennis racket or a step ladder due to elongated, straight, thin, and lacy trabeculae intersecting at a right angle. A 'sun-ray' or 'sunburst' appearance has also been reported in the literature with significant expansion, breakout of the tumour and perforation of the cortex. The bony trabeculations or bony septa formed by the OM helps them to distinguish it from malignant tumours arising centrally within the jaw bones as the latter usually cause massive bone destruction without formation of compartments.(10)

In our case CT imaging showed expansile mass arising from left maxilla extending into left maxillary sinus, completely occupying left maxillary sinus. There was thinning, scalloping and irregularities of the overlying cortex with break of the overlying cortex at places.

On gross examination, OM appear as a pale brownish gelatinous substance (tender coconut appearance).(11)Microscopically, the myxoma is made up of loosely arranged spindle-shaped and stellate cells, many of which have long fibrillar processes that tend to intermesh. The loose tissue is not highly cellular, and these cells do not show evidence of significant activity (pleomorphism, prominent nucleoli or mitotic figures). The intercellular substance is mucoid. The tumour is usually interspersed with a variable number of fine capillaries and occasionally strands of collagen.(12) Presence of relatively large quantities of hyaluronic acid in the ground substance explains its neoplastic behaviour. Depending upon the pattern of differentiation, the histological nature of the tumour varies. If the myxomatous element predominates, it is designated as odontogenic fibromyxoma and with predominance of fibrous tissue as odontogenic myxofibroma.

The typical features of the case reported here is in conformity with an OM due to typical age of presentation, characteristic soft, slippery and gelatinous nature of the specimen on macroscopic examination and the histopathological findings. OM is generally considered a slow-growing neoplasm. However it may be infiltrative and aggressive, with high recurrence rates. There are no clear surgical management guidelines for odontogenic myxoma, and a variety of approaches may be used. Surgical treatment of OM varies from local excision, curettage or enucleation to radical resection and maxillectomy. When associated with teeth, their removal is usually necessary. Recurrence is considered to be directly related to the extent of surgery. The recurrence rate OM ranges between 10–33% with an average of 25%. Hence close postoperative follow-up and long term follow up has been advocated to confirm disease free status.(13)

In case mandible, radical resection with peripheral margin and reconstruction of the defect offers reasonable cosmetic and functional outcome. However in case large maxillary lesions this may result in maxillectomy which produces significant cosmetic and functional defect. Enucleation and curettage has proved an effective approach in several cases but the risk of recurrence appears to be higher.(14) In the present case the patient had enucleation, curettage and removal of involved teeth. Nevertheless angled sinus endoscope was judiciously used to remove the tumour completely. On eight month follow up patient had no clinical evidence of recurrence and he is pleased with preservation of cosmetic appearance of the face and improvement in nasal block. As advocated in the literature, patient is placed under long term follow up to monitor for any recurrence.

Conclusion

We presented a case of a 20-year-old man who came with a slow growing OM of the left maxilla, with expansion of the maxillary sinus. On the basis of common site of occurrence, usual age of occurrence, clinical features and radiological findings one should consider OM as a differential diagnosis in patients presenting with jaw tumours. Our case has fulfilled all the criteria for OM.

Resection of malignant or benign tumours of the jaw causes emotional, functional, and aesthetic complications if not diagnosed and treated properly. There is no unanimous consensus exists concerning the extent of surgery. In our case, use of sinus endoscope helped in complete removal of the tumour with extraction of involved teeth thereby avoiding more radical surgery and preservation of cosmesis and function.

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References

- 1. Thoma, K.H. and Goldman, H.M. (1947) Central Myxoma of the Jaw. American Journal of Orthodontics and Oral Surgery, 33, B532-B540.
- G. Sivakumar, B. Kavitha, T. Saraswathi, and B. Sivapathasundharam, "Odontogenic myxoma of maxilla," Indian Journal of Dental Research, vol. 19, no. 1, pp. 62–65, 2008.
- 3. Lo Muzio L, Nocini P, Favia G, Procaccini M, Mignogna MD. Odontogenic myxoma of the jaws: A clinical, radiologic, immunohistochemical, and ultrastructural study. Oral Surg Oral Med Oral PatholRadiolEndod. 1996;82:426–33.
- 4. Peltola J, Magnusson B, Happonen RP, Borrman H. Odontogenic myxoma-a radiographic study of 21 tumours. Br J Oral Maxillofac Surg. 1994;32:298–302.
- 5. Bast BT, Pogrel MA, Regezi JA. The expression of apoptotic proteins and matrix metalloproteinases in odontogenic myxomas: J Oral Maxillofacial Surg. 2003;61:1463–6.
- 6. Brannon RB. Central odontogenic fibroma, myxoma (odontogenic myxoma, fibromyxoma), and central odontogenic granular cell tumor. Oral MaxillofacSurg Clin North Am. 2004;16:359–74.
- 7. Friedrich, R.E., et al. (2012) Radiographic Findings of Odontogenic Myxomas on Conventional Radiographs. Anticancer Research, 32, 2173-2177.
- 8. Boffano, P., et al. (2011) Surgical Treatment of Odontogenic Myxoma. Journal of Craniofacial Surgery, 22, 982-987.
- 9. Sally Nguyen1, Simon-Pierre Charbonneau2, Julien E. Ghannoum2, Eric Bissada .Extensive Odontogenic Myxoma of the Maxilla: A Case Report and Review of Literature.
- 10. Kawai T, Murakami S, Nishiyama H, Kishino M, Sakuda M, Fuhihata H. Diagnostic imaging for a case of maxillary myxoma with a review of the magnetic resonance images of myxoid lesions. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 1997;84:449–54.
- 11. Neville BW, Damn DD, Allen CM, Bouquot JE. 2nd ed. Philadelphia: WB Saunders Co; 2002. Oral and maxillofacial pathology.
- 12. Rajendran R, Sivapathasundaram B, editors. 5th ed. New Delhi: Elsevier; 2006. Shafer's Textbook of Oral Pathology.
- 13. Cankaya, M.A.E., Erdem, M.A., Bilgic, B. and Firat, D. (2013) Myxofibroma of the Maxilla, Current Concepts, and Differential Diagnosis. Journal of Dental Sciences, 1-4.
- 14. Yoko Kawase-Koga, Hideto Saijo, Kazuhito Hoshi, Tsuyoshi Takato and Yoshiyuki Mori, BMC Research Notes20147:214, Surgical management of odontogenic myxoma: a case report and review of the literature.

INTERESTING CASE OF ADULT NEPHROTIC SYNDROME



Dr. S. Murugananth MD, DM (Nephro) Consultant Nephrologist

Case report

A 48 year old male with no previous co-morbids sustained right knee injury after fall from two wheeler and had pain in Rt knee. For above complaints he resorted to alternative medicines for six months after which his symptoms subsided.

He presented to us six months later with gradually progressive pedal edema, puffiness of face and decreased urine output. On examination he was volume overloaded and normotensive. On evaluation he had features of full blown nephrotic syndrome. His renal function was normal.





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Investigations at initial presentation :

Parameter	Value
Hb%	14 gm/dl
Blood Urea	24 mg/dl
Serum Creatinine	0.6 mg/dl
Urine Albumin	4(+)
24 hours urine protein	8000 mg/day
Serum Albumin	2.0 gm/dl
Total Cholestral	706 mg/dl
Low density lipo protein	486 mg/dl

Course

Patient was counselled regarding nature of the disease. He was started on diuretics, statins and prophylactic anticoagulation to prevent thrombotic events due to nephrotic syndrome.

Patient underwent kidney biopsy for definite histological diagnosis. Immunoflourescence microscopy showed IgG(+3) and C3 (+1), granular positivity over capillary walls. Light microscopy showed spikes and pinhole leisons over glomerular basement membrane. Histo pathological diagnosis of membranous nephropathy was made.

Evaluation

Membraneous nephropathy can be primary or secondary (drugs, connective tissue disorders, infection, malignancy). Serum M-type anti phospholipase A2 receptor antibody (MPLAR) which is a sensitive test for primary membranous nephropathy was negative in our patient. This denoted a secondary cause in our patient. We persumed that the alternative medicine he had taken would have been the causative agent. Mercury is a common contaminant of alternative medicine which can induce membranous nephropathy. In our patient serum mercury level was 11.24mg/I (normal range 0.21-1.3mg/I) and 24 hours urine mercury leval was 70.58mg/L (normal range <20mg/L).

The sample of alternative medicine which the patient had consumed was collected and subjected to heavy metal analysis. It contained 52mg/kg of mercury which is very much higher than the permissible levels

Management & further course

Our patient was continued on conservative management with diuretics, statins and oral coagulants. Further course of our patient is depicted in following graphs





At presenta

Serum albumin & Urine Protein



Discussion

Mercury has strong affinity for the renal tissue. Nephrotoxicity is commonly manifested as nephrotic syndrome and tubular injury. Mercuryassociated nephrotic syndrome is reversible after elimination of the source of intoxication. Literature data from 1950 to 2010 concerning toxic mercury exposure and nephrotic syndrome described 4 cases of minimal change disease and 15 cases of membranous nephropathy. It is due to idiosyncratic reactions or an abnormal immune response to the heavy metal. Mercury components can have immune modulating activity. Experimental data showed that rats injected with mercuric chloride develop autoantibodies which immune localize along the glomerular basement membrane.

The levels in the blood or urine do not reflect the true degree of chronic mercury poisoning. Mercury tends to deposit in the tissues.

In mild disease, withdrawal of mercury-containing medicines is enough. In severe disease with clear evidence of mercury poisoning, chelation would facilitate the removal of mercury. A few authors have reported successful use of chelating agents such as DMPS(Di Mercapto Propane Sulphate). It will chelate mercury and is excreted in urine. Increased urine concentration to many fold initially indicates effectiveness of therapy. Six months therapy is effective in inducing remission.

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AN UNUSUAL CASE OF RECURRENT CHEST PAIN

Dr. Arunkumar Panneerselvam MD, DNB (Cardio)

Consultant Interventional Cardiologsit

A 41-year-old male, nonsmoker, presented with recurrent paroxysmal episodes of retrosternal chest discomfort, not related to exertion, and lasting for few minutes. His clinical examination during the episode was normal except for tachycardia and sweating. The electrocardiogram showed sinus tachycardia with a heart rate of 128 beats per minute (bpm). No ST-T changes were observed. Serial troponin T levels were normal. Baseline blood investigations including lipid profile were normal. He was subjected to treadmill exercise electrocardiogram to ascertain the cause of chest pain. He exercised very well on treadmill, achieving 12 metabolic equivalents. Although he complained of nonspecific pricking pain during the exercise, no ST-T changes were observed [Figure 1]. Echocardiography showed normal left ventricular systolic function with no wall motion abnormality. In view of no major cardiovascular risk factors, atypical nature of chest pain, and normal exercise stress test, he was treated with antacid and anxiolytics. However, despite treatment, he continued to have similar episodes with significant chest discomfort. In view of recurrent episodes of undiagnosed chest discomfort, he was subjected to a coronary angiogram. During coronary angiography, immediately after radial access, he started having chest discomfort with sinus tachycardia. Coronary angiogram was performed, which revealed grade III[1] mid-left anterior descending coronary artery myocardial bridging [Figures 2 and 3]. No plaque was observed. He was started on extendedrelease metoprolol and dose was titrated to 100 mg/day to achieve a resting heart rate of 60–70 bpm. After initiation of the beta-blocker therapy, he became completely asymptomatic. Myocardial bridging is a condition where a segment of coronary artery travels through the myocardium. This can be diagnosed noninvasively by multidetector computed tomogram.



Figure 1: Peak exercise electrocardiogram showing no ST-T changes

But more often, it is an incidental finding in coronary angiogram and does not require treatment in asymptomatic patients. In symptomatic patients, beta-blockers and calcium channel blockers are cornerstone of the therapy. If the patient has refractory symptoms, then myotomy is the treatment of choice if feasible. In our case, it was odd that the patient did not develop any chest discomfort or ST-T changes during the exercise test, despite achieving very good level of exercise. Yet, he continued to have significant symptoms on minimal exertion or even at rest. This case suggests that myocardial bridge should be one of the differential diagnoses in cases of atypical, recurrent, and unexplained chest pain.



Figure 2: Coronary angiogram diastolic frame showing normal left anterior descending coronary artery



Figure 3 : Systolic frame showing grade III mid-left anterior descending artery bridging



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