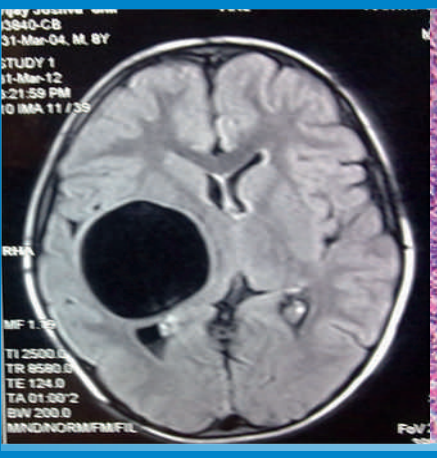
pISSN NO. 2277 - 8845
eISSN NO. 2278 - 2044

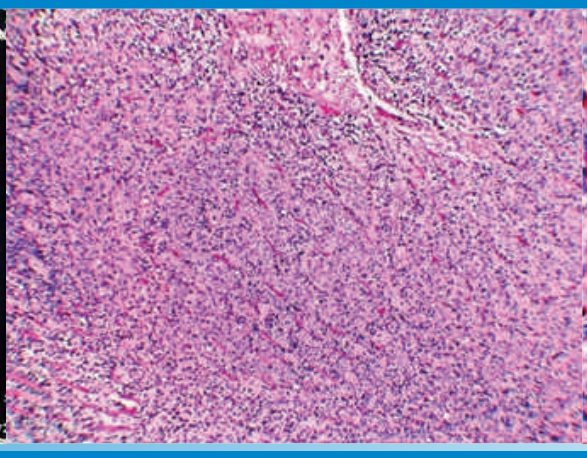
Chettinad Health City

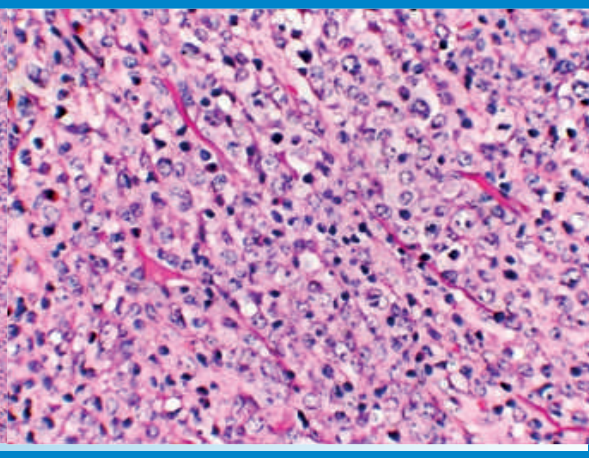
Volume - 2, Number - 1,
January- March 2013

Chettinad Health City
MEDICAL JOURNAL

In this issue







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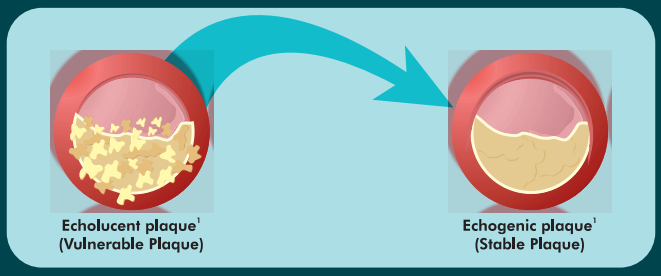

1. Atherosclerosis 215(2011) 440-445. * Coronary Artery Disease MA/CV/SXL/ADV/019/Mar 2012 Not to be used beyond Feb 2014 For the Use only of a Registered Medical Practitioner or a Laboratory or a Hospital

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EDITORIAL OFFICE

Dr. N. Pandiyan
Chief Consultant,
Department of Reproductive Medicine,
Chettinad Health City,
Rajiv Gandhi Salai, (OMR, Chennai),
Kelambakkam, Kanchipuram Dist.,
Tamil Nadu - 603 103
India
T. +91 (0)44 4742 8300
F. +91 (0)44 4741 3343

Email:
chettinadhealthcityjournal@gmail.com

PUBLISHED BY
Chettinad Academy of Research and Education

WEBSITE
www.chettinaduniversity.com/journal

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pISSN NO. 2277 - 8845
eISSN NO. 2278 - 2044

Chettinad Health City

MEDICAL JOURNAL

Contents

Editorial

Vanakkam.

The journal enters the second year with this issue. This issue carries several interesting and informative articles, a perspective article, commentaries and case reports.

Screening has been the mantra of the last century, which is carried well into this century. Several screening methods were advocated for many chronic conditions - Diabetes, Cancer, and Preeclampsia. Many of these screening methods have now come under careful scrutiny and critical thinking. A perspective article deals with prostate specific antigen screening for prostate cancer.

Health care planning for the fastest growing country in the world is not an easy task, with majority of the country's population living in the rural areas. Health care infrastructure in the rural areas leaves much to be desired. A commentary article deals with the problems in rural health care infrastructure in India.

An original article reports on a series of cases with hand, foot and mouth disease.

An original article reports on a series of cases with hand, foot and mouth disease.

This issue also carries several interesting case reports. A case report describes surgical approach to solitary giant neuro cysticercosis in a child with combined immunodeficiency. Another case report presents T-cell lymphoma arising from the gluteal muscle. A dysmorphic child with aniridia and Sydney crease is described in a case report, emphasizing the need for early diagnosis.

A case report on lingual thyroid describes the clinical presentation. The issue also presents several useful medical updates. The pages of history traces the origin of Hippocratic oath. An interesting ECG triggers your thinking brain into action.

We hope you will enjoy going through this issue; do give us your valuable feed back.

Dr. N. Pandiyan
Chief Editor : Chettinad Health City Medical Journal

E-mail : pandiyan1@yahoo.com

Perspective Article

To do or Not to do!

Dr. V.J. Raj Kumar

V J Rajkumar, M.S; FRCSI; FRCS (Glas), Cons Urological Surgeon, ABM University Health Board, Morriston Hospital, Swansea, UK

Corresponding author - Dr. V.J. Raj Kumar (rajneath@hotmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 2 - 3

Introduction

Prostate Specific Antigen (PSA) is a protein produced by the cells of prostate gland. It is present in small quantities in the serum of men with healthy prostate but is often elevated in presence of prostate cancer and other prostate disorders.

A Blood test to measure PSA is considered the most effective test currently available for early detection of prostate cancer but this effectiveness has also been questioned[1].

However PSA is neither specific for prostate nor cancer. Although present in large amount in prostatic tissue, semen and serum in men, it has also been detected in other body fluids and tissues including female ejaculate, breast milk, amniotic fluid, endometrium, normal breast tissue and salivary gland tissue[2].

Normal/Reference Range

Defining a normal range is difficult. Rather than attempting to define a normal range, it would probably more appropriate to provide the clinician with an appropriate PSA cut off level that affords a reasonable yield of cancer. Even more appropriate, perhaps, would be to establish additional criteria e.g. age, race, digital rectal examination results that would provide a risk assessment of prostate cancer being present.

Various factors such as benign hyperplasia, inflammation, ejaculation, cycling, prostatic massage and instrumentation have all been known to alter the PSA level. Even though there is no specific normal or abnormal PSA level, 4.0ng/ml it is generally taken as a cut off level. However prostate cancer was diagnosed in 15.2% of men with PSA level below 4.0ng/ml[3]. In another study 65-75% of men of PSA between 4.1- 9.9ng/ml did not have prostate cancer[4].

Limitation Of PSA Test

- Detecting tumour does not always mean saving lives- finding a small tumour does not necessarily reduce the chances of dying from prostate cancer. PSA testing may identify very slow growing tumours that are unlikely to threaten life.

- False positive test – False positive test occurs when the PSA level is elevated but no cancer is actually present. Only 25-35% of men who have a biopsy due to an elevated PSA actually have prostate cancer. Hence a false positive test may lead to additional medical procedures that have potential risk and significant financial cost and can create anxiety for the patient and for the family

- False negative test – False negative test occurs when the PSA level is normal range even though prostate cancer is actually present. Most prostate cancers are slow growing and may exist for decades before they cause symptoms.

Factors Enhancing Perfomance Of PSA

The major efforts to improve PSA testing have addressed enhancement of specificity. The question of whether to improve sensitivity or specificity is important as they are generally inversely related parameters. Efforts to enhance specificity would appear to be more logical because with serial testing, a false negative result is of less consequence. By increasing the PSA cut off level, specificity improves but at the cost of decreasing sensitivity. False positive test are exceedingly expensive as they mandate further testing with attendant increase in expenses and morbidity

Number of approaches have been widely used to enhance PSA performance

Age specific PSA cut off point have been used, taking into account that prostate grows with age and PSA gradually increase with age.

40-49yrs ----------2.5ng/ml

50-59yrs----------3.5ng/ml

60-69yrs----------4.5ng/ml

70-79yrs----------6.5ng/ml

The age specific ranges have not been generally favoured because their use may lead to missing or delaying detection of prostate cancer in as many as 20% of men in the 60's and 60% of men in their 70's.

- PSA velocity - change in PSA overtime may be greater in men with prostate cancer. It is generally agreed that rise of PSA over 0.7ng/ml over a period of 12 months may indicate prostate cancer
- Prostate density – Adjusts serum PSA with respect to prostatic volume, as a larger prostate may be associated with higher PSA level even though the gland is benign.
- Free/Total PSA ratio may be helpful in differentiating between benign and malignant prostate in men with PSA between 4-10ng/ml
- Alteration of PSA cut off level – reducing the cut off level will increase the chance of detection of cancer but may also increase over diagnosis and false positive results and lead to unnecessary medical procedures.

PSA In Prostate Cancer Screening

The use of PSA to screen men for prostate cancer is controversial because it is not yet known for certain whether it actually saves lives. Moreover it is not clear that the benefits of PSA screening outweigh the risk of follow up diagnostic test and cancer treatment. The PSA test may detect small cancers that would never become life threatening. This puts men at risk of complications from unnecessary treatment.

The benefits of screening for prostate cancer are still being studied. Two large trials are ongoing at the moment to look at the benefits of prostate cancer screening ie PLCO trial and ERSPC trial[1,5]. In the ERSPC trial it has been estimated that 1410 men would have to be screened and 48 additional cancers would have to be detected to prevent one death from prostate cancer.

Key Points

- It is generally accepted to use 4.0ng/ml as the cut off level.
- Many factors affect PSA levels in serum
- Single PSA test is not used as a diagnostic test
- PSA test should always be used along with digital rectal examination
- PSA should offered to well informed men aged 50 and over who have a life expectancy of more than 10years
- Decision to biopsy the prostate should take into account other additional factors, PSA velocity, PSA density, age, family history and co-morbidities[6].
- PSA on its own is more useful as a prognostic tool

References

1) Schroder FH et al'' Screening and Prostate Cancer Mortality in a Randomised European Study''. New Eng J.Med. 2009; 360: 1320 - 1328.

2) Dale L. Laux et al. Detection of PSA Using Membrane Based Test. Midwestern Assoc of Forensic Scientists 2008. http://mafs.net/pdf/forensicdetectionsemen3.pdf

3) Thompson IM et al. Prevalence of prostate cancer among men with a PSA less than 4.0 New Eng J.Med. 2004; 350 (22): 2239 - 46.

4) Smith DS et al. Early Detection of Prostate Cancer with PSA. Cancer. 1997; 80 (6): 1852 - 1856

5) Andriole G et al. Mortality results from a randomized Prostate Cancer screening trial (PLCO trial). New Eng J.Med.2009; 360 (13) : 1310 - 1319.

6) AUA (American Urological Association) Guidelines and NICE (National Institute of Health and Clinical Excellence UK) Guidelines

Belly Buddy

Not all bacteria are malevolent and harmful. Billions of those that colonise our gut maintain a relationship with us that is at least commensal and at best, mutually beneficial. One such organism goes by the name, Akkermansia muciniphila and it accounts for nearly 3-5% of gut microbial population. It is a resident of the mucus layer and it appears to degrade the mucus. Dr. Patrice D. Cani and his colleague from Belgium, have discovered that its numbers are significantly reduced in obesity and type 2 diabetes resulting in increased inflammation and defective gut barrier. When the researchers (working with rats) restored its numbers through prebiotic feeding, metabolic status of the host improved with reversal of fat-mass gain and reduction in metabolic endotoxaemia, adipose tissue inflammation, and insulin resistance. Akkermansia muciniphila apparently achieves this by increasing the intestinal levels of endocannabinoids that control inflammation, the gut barrier, and gut peptide secretion. Of course it is effective only when it is alive. Not all our true friends are easily recognisable. This one is microscopic with an unpronounceable name. But does it matter? Keep it alive in your gut and it is your friend for life. The study is published in the latest issue of Proceedings of the National academy of Sciences (PNAS 2013 ; published ahead of print May 13, 2013, doi:10.1073/pnas.1219451110)

- Dr. K. Ramesh Rao

Commentary

An Objective View of Problems in Rural Healthcare Infrastructure in India

Dr. Syed Meraj Ahmed

Associate Professor, Department of Community Medicine, Chettinad Hospital & Research Institute, Chennai, India

Dr. S M Ahmed completed his MBBS and MD in Community Medicine from Jawaharlal Nehru Medical College and Hospital, Aligarh Muslim University. He also holds the DLSHTM and MSc in Public Health degree from London School of Hygiene and Tropical Medicine, University of London, UK. He qualified in the PLAB (UK) licensing examination in 2005 and was working in a GP surgery clinic in UK for more than a year. He is an active member of the Indian Medical Association (IMA) and IAPSM. He is currently working as an Associate Professor in the Department of Community Medicine, Chettinad Hospital & Research Institute.

Corresponding author - Dr. Syed Meraj Ahmed (merajahmed10@hotmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 4 - 6

Abstract

Presently it seems that adequate rural healthcare in India is a virtual pot of gold at the end of a long winding road. While we are making great strides in upgrading our healthcare infrastructure and resources to world standards in the metropolises and other cities, the gap in accessibility to these resources between the urban and rural population within the country is ever increasing.

Overall we still can only compare our medical successes with other developing and poorly developed countries and are still far away from the kind of health changes that has been brought about in developed countries. Previous studies have suggested that majority of the rural deaths which are preventable, are due to communicable, parasitic, respiratory diseases and infections. Easily accessible basic interventions can help in minimizing the sufferings.

Problem areas that needs to be looked into while comparing rural and urban healthcare services are inequality and inadequacy; misallocation of public money and inadequate rural public health expenditure; flagrant commercialization of healthcare and crippling hold of drug manufacturing companies on distribution and pricing of life saving medicines.

The selective, institutionalized, centralized and top – down method of healthcare service delivery needs to be dismantled and a decentralized medical service which can be easily accessed by the people is required for the majority of the rural population. Small changes along with some drastic ones by the people who develop policies are required like the concept of rural medical colleges, family physicians, integration of Indian System of Medicine Practitioners into the registered medical practitioner category. Most important is appropriate allocation of funds and budgets to upgrade/ develop the healthcare infrastructure among the rural population that is actually utilized and shown through regular audits.

Key Words: Rural healthcare, Public private partnership, Stakeholders, Healthcare planning

Problem Statement

Innumerable data have shown that there is a dearth of even appreciable quality healthcare as far as the population who are poor, lives in underdeveloped/ remote areas or even the suburban slums are concerned [1,2,3,4]. An article rightly published that access to health care is defined as the use of healthcare by those who need it and studies have shown that gender, social geography, social groups and class influence access5.

Even when the infrastructure and resources are available for the select few it becomes a harrowing experience to get medical attention since it all becomes too expensive for the common man. A recent article on a major newspaper stated that for a patient around 70% of total health spending is out of pocket, and around 70% of that is on drugs[6]. India faces an acute shortage of over 64 lakh skilled human resource in the health sector with Uttar Pradesh alone accounting for a shortfall of 10 lakh allied healthcare professionals, according to a latest study by the Public Health Foundation of India[7].

Overall we still can only compare our medical successes with other developing and poorly developed countries and are far away from the kind of health services access at the population level that has been brought about in developed countries.

Previous studies have suggested that majority of the rural deaths which are preventable, are due to communicable, parasitic, respiratory diseases and infections. Easily accessible basic interventions can help in minimizing the sufferings.

Roadblocks To Essential Healthcare Coverage

- Expanding middle class population with severely limited access to healthcare services. (Table -1 & 2)
- Communicable diseases once thought to be under control, such as dengue fever, viral hepatitis, tuberculosis, malaria, and pneumonia etc. have returned in force or have developed a stubborn resistance to drugs.

- Over the next 5-10 years, lifestyle diseases are expected to grow at a faster rate than infectious diseases in India, and to result in an increase in cost per treatment.
- Infiltration of unregulated private healthcare services even up to the tertiary secondary and primary health centre and sub centre levels have increased the financial burden on the individual patients as ~80% of the cost is borne out of their own pocket. (Table – 3)
- Inability to counter commercial interests of pharmaceutical companies with the broader social objective of curing disease and preventing epidemics that could literally ravage the Indian subcontinent.

Health Sector Reforms being undertaken

- Strengthening management structures by recruiting health workers on a temporary/contractual basis.
- Getting staff on a contractual basis wherever there is a dearth of nursing and allied health staff.
- Strengthening infrastructure by upgrading health centres and introducing better treatment protocols.
- Public private partnership on a built-own-operate-transfer basis.
- Encouraging outreach activities by NGOs and utilizing their close proximity with the population to improve the healthcare delivery.

Interventions that can really help

- The rural healthcare system to be modelled on the basis of the requirement at the local level rather than on the vision of the central or state government.
- Performance based incentives to the healthcare institutions both governmental and private in rural areas.
- To vigorously promote the concept of medical colleges having 4-5 villages under them and to provide preventive, curative and promotive health.
- Affordable healthcare insurance that allows access to essential healthcare services[8,9,10].
- A compulsory rotatory posting of new medical graduates to rural areas and attractive incentives provided depending on the performance[11,12].
- A universal mobile number that can be dialled to access a trained healthcare specialist for information on any health related problems at the district level.
- Promotion by the Government of local manufacturers to make medical equipments and diagnostic equipments that is more affordable.

In Conclusion

Policy makers and other stakeholders need to sift through the gargantuan pile of mistakes, failures, lost opportunities and myopic ideas. That means huge investments in healthcare infrastructure well utilized in the right areas and not well spent. Universal healthcare delivery should imply access to it by everyone who needs it irrespective of caste, colour, creed or social status.

Table 1

Per lakh population	beds	hospitals	Dispensaries
Urban	178.78	3.6	3.6
Rural	9.85	0.36	1.49

Source: Review of healthcare in India, 2005. Can be accessed at http://www.cehat.org/publications/PDf 20files/r51.pdf

Table 2

India: Health Workforce and Capacity (2005 – 2010)	
Physicians (per 10000 pop.)	6
Nurses & Midwife (per 10000 pop.)	10
Community Health Workers (per 10000 pop.)	7
Births attended by skilled health personnel (%)	58%
Hospital beds (per 10000 pop.)	9

http://www.who.int/gho/publications/world_health_statistics/2012/en/index.html

Table 3

INDIA: Funding, financing and expenditure	
Health expenditure per capita (PPP; $) (2009)	$124
Total expenditure on health (as percent of GDP) (2009)	4.2%
General Govt. expenditure on health (as % of General Govt. expenditure) (2009)	3.7%
General Govt. expenditure on health (as % of Total expenditure on health) (2009)	30.3%
Private expenditure on health (as % of total expenditure on health) (2009)	69.7%
Out of pocket expenditure (as % of private expenditure on health) (2009)	86.4%

http://www.who.int/gho/publications/world_health_statistics/2012/en/index.html.

References

1) De P et al; Efficiency of health care system in India: an inter-state analysis using DEA approach; Soc Work Public Health. 2012; 27(5):482-506.

2) Purohit BC; Inter-state disparities in health care and financial burden on the poor in India. J Health Soc Policy. 2004; 18(3):37-60.

3) Mathew JL; Inequity in childhood immunization in India: a systematic review; Indian Pediatrics. 2012 Mar; 49(3):203-23.

4) De Costa A et al; Where are healthcare providers? Exploring relationships between context and human resources for health Madhya Pradesh province, India Health Policy. 2009 Nov; 93(1):41-7.

5) http://www.cehat.org/publications/PDf%20files/r51.pdf.

6) http://www.thehindu.com/health/article3850103.ece;

7) http://southasia.oneworld.net/resources/from-paramedics-to-allied-health-sciences-landscaping-the-journey-and-way-forward#.UOVXm28zpJc

8) James CD et al; To retain or remove user fees? Reflections on the current debate in low- and middle-income countries; Applied Health Econ Health Policy. 2006;5(3):137-53

9) Dalinjong PA et al; The national health insurance scheme: perceptions and experiences of health care providers and clients in two districts of Ghana Health Econ Rev. 2012 Jul 23; 2(1):13.

10) Jehu-Appiah C et al; Household perceptions and their implications for enrolment in the National Health Insurance Scheme in Ghana; Health Policy Plan. 2012 May; 27(3):222-33

11) Saini NK et al; what impedes working in rural areas? A study of aspiring doctors in the National Capital Region, India Rural Remote Health. 2012; 12:1967.

12) Lee YH et al; Initial evaluation of rural programs at the Australian National University: understanding the effects of rural programs on intentions for rural and remote medical practice; Rural Remote Health. 2011; 11(2):1602.

American Kids and Mental Health

According to a new report released by Centre for Disease Control (CDC), 20% of all American kids (i.e. 1 in 5) between the ages of 3 and 17 have some sort of a mental health problem. The report was developed in collaboration with the Substance Abuse and Mental Health Services Administration (SAMHSA), National Institute of Mental Health (NIMH), and Health Resources and Services Administration (HRSA). The commonest mental disorder appears to be ADHD (6.8%), followed by behavioral problems (3.5%), anxiety (3%), depression (2.1%), autism related conditions (1.1%) and Tourette syndrome (0.2%). Adolescents aged 12 to 17 years in addition had history of illicit drug use disorder in the last year (4.7%), alcohol use disorder in the last year (4.2%) and cigarette dependence in the last month (2.8%). Most of these problems are found more frequently in males; however, depression and alcohol use disorder were more common in girls. Report also noted that more boys in the age group of 12 - 17 are likely to commit suicide. It is truly alarming! It would be interesting to know how the Indian kids fare http://www.cdc.gov/mmwr/preview/mmwrhtml/su6202a1.htm?s_cid=su6202a1_w

- Dr. K. Ramesh Rao

Original Article

Hand, Foot and Mouth Disease

*Dr.Anitha Elango, **Dr.Rathinasamy, **Dr.Umadevi, ***Dr.Jaishree, ***Dr.Suresh, ****Dr.Shilpa Jha

* Senior Resident, ** Professor, *** Associate Professor, **** Junior Resident, Department of Paediatrics, Chettinad Hospital and Research Institute, Chennai, India.

Dr.Anitha is working as a Paediatrician in Chettinad Health City from 2012. She finished her postgraduation from Sri Ramachandra University. General paediatrics is her chief interest.

Corresponding author - Dr.Anitha Elango (dr.anishan@gmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 7 - 8

Introduction

Hand, foot and mouth disease (HFMD) is a common illness of infants and children caused by Coxsackie viruses. It occurs in children less than 10 years, commonly less than 4 years. It is common to have outbreaks in summer. Clinical examination of children reveals vesiculopustular lesions over the throat, tonsils, hands(especially palms), feet(especially sole), and buttocks.

What Made Us Suspect Hand Foot Mouth Disease

In Paediatric OPD mothers of children complained of maculopapular rashes over the the hands,feet and buttocks with the history of mild fever and pruritis.On examination they were found to have vesiculopustular lesions over the body (commonly in soles,palms,oral cavity and buttocks). These lesions made us think of hand foot mouth disease. For example:

1 yr old child came with complaints of

- rashes all over the body-2 days
- excessive cry-1 day
- Decreased appetite-1 day
- Uneasiness-1 day

Parents noticed that the child had macular lesions over the body 48 hrs ago which turned to papular/vesicular lesions 24hrs ago before coming to the opd.They had also seen 30-40 lesions in buttocks .On examination the child was found to have vesiculopustular lesion over the buttocks, knee (Fig 1&2), forearm, tongue and palate .With such typical history and clinical presentation we made a diagnosis of hand foot mouth disease and the patient was treated symptomatically. We saw about 20 similar cases during the period of Aug 2012-Mar 2013.

Data analysis:

Table -1 Age group:

Age	no.of cases	percentage
<1 year	4	20
1-3 years	12	60
3-6 years	0	0
6-12 years	4	20
>12 years	0	0

Table-2 Clinical manifestation:

Symptoms/signs	no.of cases	percentage
Fever(low grade)	14	70
Malaise/tiredness	10	50
Irritability/frequent cry	6	30
Decreased appetite	14	70
Eruptions	20	100
Cough/cold	4	20
Loose stools	2	10
Itching	12	60

Investigations: 1.Complete Blood Count 2.Xray chest
Diagnosis Is Primarily Clinical.Virus isolation was not done since it is costly and not available at regional centres.

Treatment:

- Reassurance
- Prevention of transmission
- Fluid intake
- Antipyretics
- School exclusion-until symptoms resolve, blisters dry

Discussion

The most common causes of Hand, Foot and Mouth disease(HFMD) are coxsackie virus A16 and enterovirus 71 (EV71) which belong to picornaviridae family[1]. Outbreaks of HFMD occur worldwide, more frequently in summer and early autumn.But we have seen cases in winter season.

Incubation period is about 3-5 days and the infectivity is 7 days from the incubation period.It mainly spreads via faeco oral route[2].Other modes of transmission are by direct contact of secretions [nasopharyngeal(droplet) spread] or fluid in blisters and by means of vertical spread.

In our cases vesicles were present mostly in the buttock area compared to the limbs,whereas literature says the most common area affected is soles and palms[3].All cases recovered completely within 10 days.

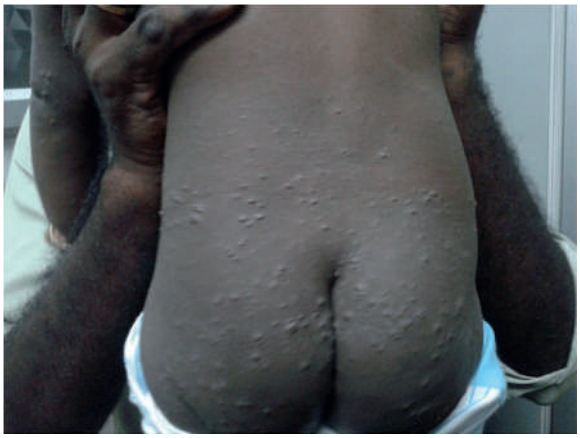

Fig1: Vesiculopapular lesions in the gluteal region

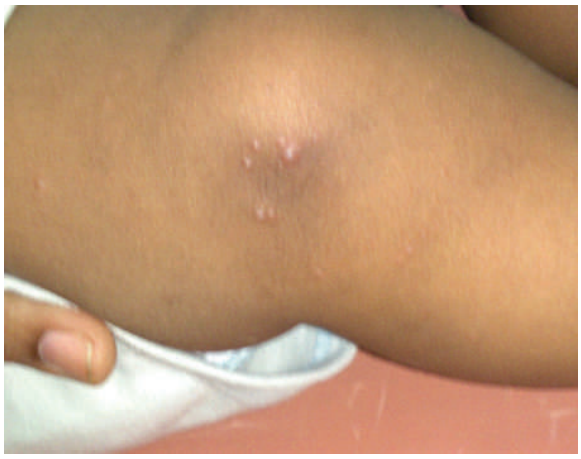

Fig2: Vesiculopapular lesions in the Knee

Diagnosis is primarily clinical[4]. The organisms can be isolated from NPA(nasopahryngeal aspirate) or throat swab for EV PCR,Other fluids (vesicle, CSF) for EV PCR,Stool for EV isolation/ culture,but it may take weeks to permit the characterisation of viruses.In various outbreaks worldwide CA16 and EV71 viruses were identified as the causative agent for this outbreak[1,5-9].

Differential diagnosis includes herpangina(limited to posterior oral cavity with no skin lesion),herpes simplex &herpes zoster virus,chicken pox,viral pharyngitis, scabies.

Coxsackie virus A16 infection is a mild disease and patients will recover within 7 to 10 days. Enterovirus EV71 leads to neurological complications such as aseptic meningitis, encephalitis,acute flaccid paralysis,fatal neurogenic pulmonary oedema, dehydration, secondary bacterial infection[9].

In one of our cases,the child developed bronchopneumonia due to secondary bacterial infection which resolved with treatment. Another case had severe pruritis even after the vesicles got resolved and was treated symptomatically.

Prevention is mainly by staying away from crowded places such as shopping centres when we are unwell and following good hygienic practices such as frequent hand washing to limit the spread of the disease.

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References

1) Li L He Y,Yang H,Zhu J,Xu X,Dong J,Zhu Y,et al.Genetic characteristic of human Enterovirus 71 and Coxsackie virus A 16 circulating form 1999 to 2004 in Shenzhen,Peoples' Republic of China.J Clinical Microbiol.2005;43:3835-9.

2) Kar BR,Dwibedi B,Kar SK.An Outbreak of Hand Foot Mouth Disease in Bhubaneshwar,Odisha.Ind Ped.2013;50(1):139-142.

3) Mark J. Abzug.Hand-Foot-Mouth Disease,Nonpolio Enteroviruses,Kleigman,Stanton,St.Geme,Schor, Behrman,Nelson Textbook of Pediatrics,19e;242:1090.

4) Sarma N,Sarkar A,Mukherjee A,Ghosh A,Dhar S,Malakar R.Epidemic of hand foot mouth disease in West Bengal,India in August,2007:A multicentric study.Indian J Dermatol.2009;54:26-30.

5) Podin Y,Gias EL,Ong F,Leong YW,Yee SF,Yusof MA,et al.Sentinel surveillance for human Enterovirus 71 in Sarawak,Malaysia:Lessons from the first 7 years.BMC Public Health.2006;6:180.

6) Ho M,Chen ER,Hsu KH,TwuSJ,Chen KT,Tsai SF,et al.An epidemic of enterovirus 71 infection in Taiwan.N Engl J Med.1999;341:929-35.

7) McMinn P,Stratov I,Nagarajan L,Davis S.Neurological manifestations of enterovirus 71 infection in children during an outbreak of Hand ,Foot, and Mouth disease in Western Australia.Clin Infect Dis.2001;32:236-42.

8) Cardosa MJ,Perera D,Brown BA,Cheon D,Chan HM,Chan KP,et al.Molecular epidemiology of human enterovirus 71 strains and recent outbreaks in the Asia-Pacific region:Comparative analysis of the VP1 and VP4 Genes.Emerg Infec Dis.2003;9:462-8.

9) Dwibedi B,Kar BR,Kar SK.Hand,foot and mouth disease(HFMD):A newly emerging infection in Orissa,India.National Med J India.2010;23:313.

Case Report

Solitary Giant Neurocysticercosis In A Child With Combined Immunodeficiency

Dr.K.V.Karthikeyan* Dr.V.G.Ramesh**

*Consultant Neurosurgeon, **Professor &HOD , Department of Neurosciences, Chettinad Super Speciality Hospital, Chennai, India

Dr.K.V.Karthikeyan is currently working as Consultant Neurosurgeon at Chettinad Super Speciality Hospital. He completed his graduation in 2001 from the prestigious Madras Medical college and completed his MCH (Neurosurgery -5 yrs) from the Madras Institute of Neurology at Madras Medical college in 2007.He is very well trained in Micro neurosurgery and vascular neurosurgery .He joined our institute in 2010.He has special interest in Vascular Neurosurgery and Endoscopic Neurosurgery.

Corresponding author - Dr.K.V.Karthikeyan (surgenn@gmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 9 - 10

Abstract

Neurocysticercosis is one of the most common CNS infections in both adult and paediatric age group. Giant Neurocysticercosis is a rare phenomenon in brain. Few cases have been reported in adult population . Solitary giant cystic neurocysticercosis in paediatric age group have not been reported in literature. Here, an 8 yr old boy, already a known case of combined immunodeficiency since birth with frequent chest, skin and mucocutaneous infection presented with large intracranial cystic lesion. CT and MRI imaging showed a non contrast enhancing cystic lesion . Intra operatively a cystic lesion with live worm and scolex removed and confirmed with histopathology as Neurocysticercosis. Giant neurocysticercosis has not been reported in severe combined immunodeficiency patients. Hence we present this interesting case.

Key words : Giant – Cysticercosis – Combined Immunodeficincy

Case Report

An 8 year old boy presented with history of progressive weakness of left upper limb for the past 4 weeks duration and headache for 2 weeks duration. Patient had history of repeated chest, skin and mucosal infection since birth and he was recently diagnosed as Combined immunodeficiency in the form of both B cell and T cell dysfunction. Biochemical and pathological tests confirmed it. On examination the boy was conscious, oriented, and had early papilloedema. He had left hemiparesis of grade IV power. MRI Brain showed a well circumscribed non contrast enhancing 8x 7 cm single cystic lesion in the right temporo parietal region with mass effect. Patient underwent a right temoporo parietal craniotomy and trans sulcal approach. At a depth of 3 cm a solitary cyst along with wall was removed completely without any spillage. The post operative period was uneventful and the hemiparesis recovered completely during 6 week follow up. Histopathology confirmed as cysticercus with scolex.

Discussion

Neurocysticercosis is the most common parasitic infection of the CNS caused by T.Solium. Neurocysticercosis is further divided into parenchymal and extraparenchymal disease. Parenchymal disease is characterized by infection with cysticerci within the brain parenchyma. Extraparenchymal disease develops when cysticerci migrate to the CSF of the ventricles, cisterns, and subarachnoid space or within the eyes or spinal cord.

Approximately 10-20% of patients with neurocysticercosis present with extraparenchymal disease, often with concomitant parenchymal disease. Subarachnoid neurocysticercosis may form in the gyri of the cerebral convexities or in the fissures of the brain, especially the sylvian fissures. These forms of neurocysticercosis are associated with parenchymal inflammation and resemble parenchymal disease in manifestations and pathogenesis.

Oncospheres that invade the brain may lodge in the brain parenchyma, subarachnoid space, ventricular space[1], or spinal cord. Cysticerci develop after 2 months and may or may not stimulate an appreciable inflammatory response.

In the brain parenchyma, cysticerci form a thin capsule of fibrous tissue that thickens with time. After several years, the parasite dies or is killed and is replaced by an astroglial and fibrous tissue granuloma due to the immune reaction, that becomes calcified. Cysts that grow in the sylvian fissure and in the subarachnoid space at the base of the skull may enlarge to 10 - 15 cm in diameter. Meningeal and spinal cord cysticercosis occurs if the oncospheres enter via the choroid plexus and hatch in the arachnoid membranes along the neural axis.

Most of the cystecercosis infection are small lesion and they elicit strong immune mediated inflammatory reaction which in turn causes extensive surrounding edema and they present with seizure or neurological deficits. But in immunodefiency patients due to the lack of immune reaction they remain asymptomatic until they become big in size and present with increased Intra Cranial Pressure (ICP) features[2,3].

The number of cysticerci present ranges from one to several hundred. But solitary giant cysticercosis without any immune reaction has been reported only in very few instances. Most of the cysticercosis are treated with medical management. Only giant lesions causing ICP features require surgical excision[4].

Conclusion

Neurocysticercosis present mostly as single or multiple lesions with strong immune reaction like surrounding edema. Solitary giant cysticercosis are rare in children.

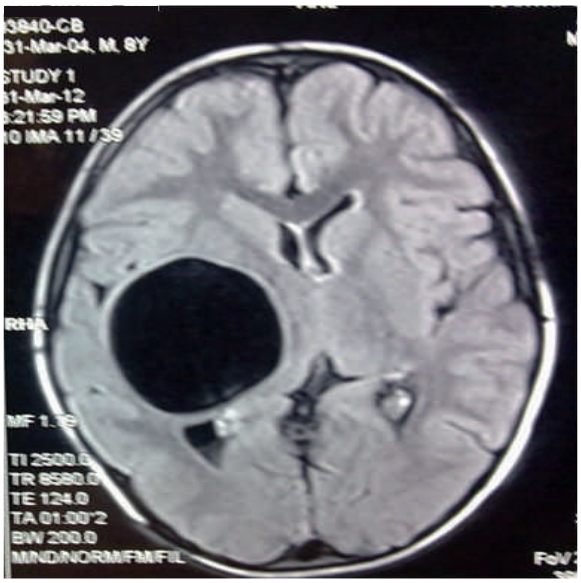


Fig 1: MRI T1 Showing Large Cystic Lesion In Right Temporoparietal Region

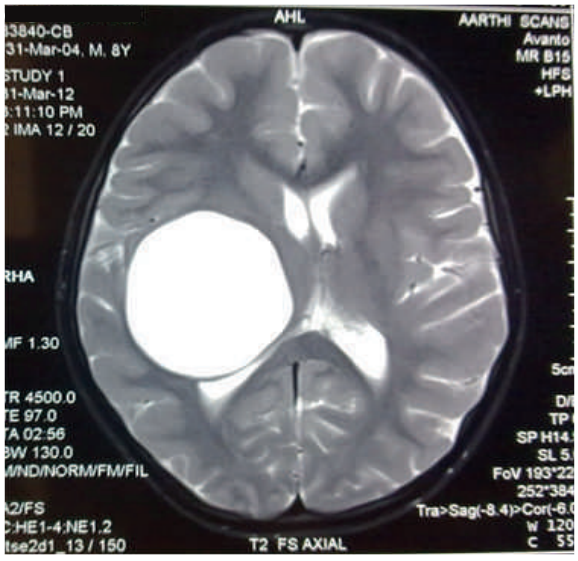


Fig 2: MRI Brain T2 Sequence Showing Large Cyst In Right Temporoparietal Region

That too occurring with background of Combine Immunodeficiency has not been reported so far.

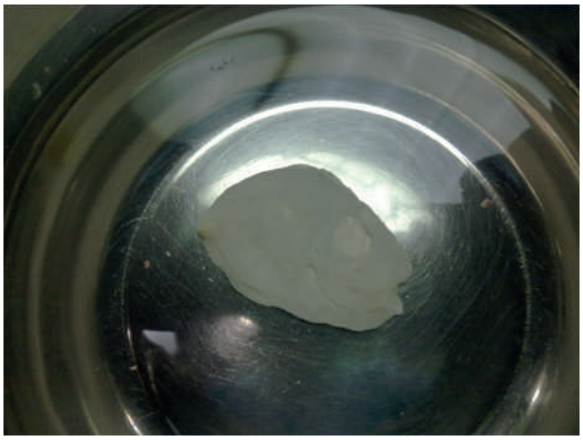


Fig 3: Resected Cyst along with worm

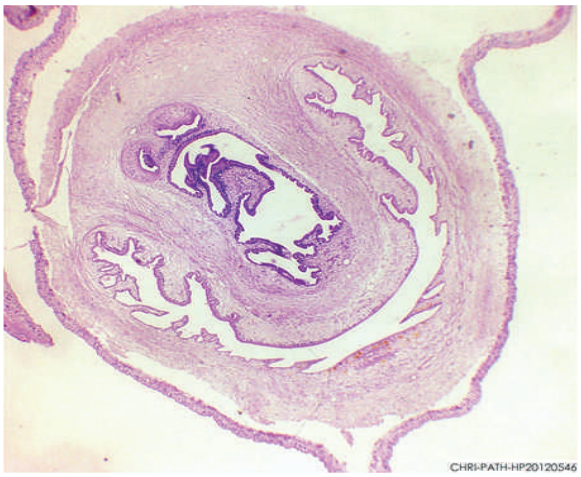


Fig 4: Histopathology showing scolex with wall lined by eosinophilic and mononuclear infiltrates

References

1) Bansal KK, Gupta C, Goel D, Singhal A, Bansal R. Giant fourth ventricular cyst : diagnostic and therapeutic dilemmas. J Assoc Physicians India. 2006 Apr;54:289.

2) Ramesh VG, Parthiban A. Giant parenchymal cysticercosis with unusual imaging features. J Clin Neurosci. 2008 Dec;15(12):1404-6. doi: 10.1016/j.jocn.2007.09.026.

3) Umredkar A, Singla N, Mohindra S, Bal A, Gupta SK. Giant intraparenchymal neurocysticercosis: report of surgical aspects two cases. Neurol India. 2009 Nov-Dec;57(6):800-2. doi: 10.4103/0028-3886.59483.

4) Brandão RA, Nunes TW, Dellaretti Filho MA, Tótola PV, Fonseca VS, Souza WC. Giant Neurocysticercosis: diagnosis and treatment. Rev Assoc Med Bras. 2010 Jul-Aug;56(4):395-6.

Case Report

T- Cell Lymphoma Arising From Gluteal Muscle –A Rare Presentation

Dr.K.Senthil Kumar* Dr. R. Anantharamakrishnan** Dr. R. Karunanithi***

*Assistant Professor, **Associate Professor, ***Professor, Department of Surgery, Chettinad Hospital and Research Institute, Chennai,India

Dr.K.Senthil Kumar M.S.,FMAS.,Dip in Lap, is an Assistant Professor in the Department of Surgery, Chettinad Hospital and Research Institute. He did his M.B.B.S in PSG Institute of Medical Sciences and Research (1999 batch) and postgraduation in Coimbatore Medical College (2006 batch). He joined Chettinad Hospital and Research Institute in 2009. He has done Fellowship in minimal access surgery. He has attended many state and national conferences and presented interesting clinical cases and case studies in surgery. He is a member of Asia Pacific Hernia Society and his field of interest is Urology and minimal access Surgery.

Corresponding author - Dr. K. Senthil Kumar (drsenthil_psg@yahoo.co.in)

Chettinad Health City Medical Journal 2014; 2(1): 11 - 13

Abstract

Non-Hodgkin Lymphomas (NHLs)are diverse group of blood cancers that include any kind of lymphoma except Hodgkin's lymphomas[1]. NHLs arise from the lymph nodes or extranodal sites. B cell lymphomas are common and T cell lymphomas account for only 10 to 15 % of NHL. Extranodal sites comprise 24% – 48% of NHL cases, commonly occuring sites are GI tract, skin and bone. Rare localizations have also been reported like skeletal muscles comprising 0.5% of Extranodal NHLs[2]. There is an increasing incidence of these Extranodal lymphomas during the past decades. Sometimes, Lymphomas present as refractory cellulitis and is diagnosed after failure to respond to antimicrobial therapy. Here we report a rare case of T cell type of NHL arising from gluteal muscle which presented with features mimicking cellulitis.

Key Words: T- Cell Lymphoma, Non-Hodgkin Lymphomas (NHLs), Gluteal swelling

Case Report

A 60 yr female presented to surgical opd with complaints of pain and swelling in the left gluteal region and hip for 2 weeks. She had fever and difficulty in walking for 3 days.There was no history of weight loss and loss of appetite. No significant past medical or surgical history. On examination patient was moderately built and well nourished. On examination patient was febrile,no pallor, left inguinal nodes palpable.Left side pitting pedal edema was present.No signs of DVT. Vitals were stable. CVS, RS examination was normal. Per abdomen examination : No hepatosplenomegaly Examination of the left gluteal region and thigh revealed Swelling in the lt gluteal region, thigh and lower limb(Fig1). Tenderness present in the lt hip, lt iliac fossa, lt gluteal region. Warmth was present, and no fluctuation. Blood investigations were normal. USG left gluteal region was suggestive of cellulitis, with no evidence of loculation. Hence a diagnosis of gluteal cellulitis was made and the patient was started on intravenous antibiotics. Patient was treated conservatively for 1 week but her symptoms never improved. She had progression of pain and swelling. MRI showed features suggestive of inflammatory pathology in gluteal muscle (Fig 2). Hence Incision and drainage was planned under spinal anaesthesia. Intraop findings : Gluteal muscle was found to be unhealthy & grey in colour, intermuscular layer had fluid, no pus found. Hence muscle biopsy was taken and sent for histopathology, and fluid was sent for culture & sensitivity. Fluid showed no bacterial growth. Inguinal node biopy showed reactive hyperplasia.Muscle biopsy report was positive for malignancy-suggestive of high grade NHL(Fig 3a,3b). Immunohistochemistry showed strong positivity for CD 45,CD 3 than CD 20 (Fig 4a,4b,4c)confirming a diagnosis of diffuse large T cell lymphoma. Hence a diagnosis of primary T cell lymphoma arising from gluteal muscle was made. Patient was started on chemoradiation and responded well to treatment.

Discussion

The patient in this case presented with features suggestive of cellulitis and was found not responding to antimicrobial therapy. Patient was investigated further and MRI was done which also suggested features of cellulitis. Finally, muscle biopsy clinched the diagnosis of NHL - T cell type.

- Peripheral T cell Lymphoma generally affect 60 years and older and are common more often in men than in women[3]. The signs and symptoms vary according to the site, subtype and grade of lymphoma. Common signs and symptoms include fatigue, painless swelling in lymph nodes, fever, weight loss, night sweats. Primary extranodal lymphomas were defined as those presenting with extranodal sites and no or only minor lymph node involvement[4]. Almost any organ can be affected by NHL, the most common extranodal sites being stomach, Waldeyer ring, spleen, central nervous system, bone, lung, skin and skeletal muscles[5]. Primary muscle lymphoma is even less common. Although primary skeletal muscle NHL is very uncommon[6,7] clinical presentation of refractory cellulitis, as seen in this case is extremely rare.

- The extranodal NHLs are difficult to treat, the main modality of treatment being anthracycline based chemotherapy regimes[8] as follows: CHOP(cyclophosphamide, hydroxydoxorubicin, oncovin, prednisone) EPOCH (etoposide, prednisone, oncovin, cyclophosphamide, hydroxydoxorubicin) Hyper - CVAD (cyclophosphamide, vincristine, adriamycin, dexamethasone) "hyper" refers to "hyperfractionation of the dose". Locoregional radiation therapy is the treatment option for limited-stage disease.

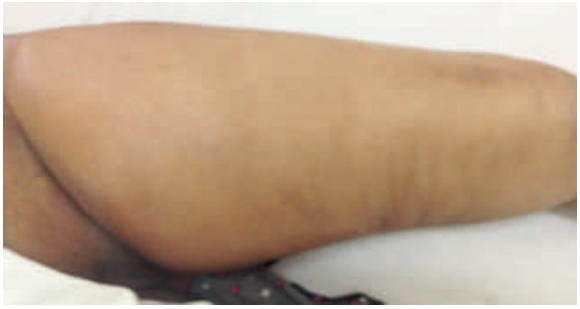


Fig 1: Clinical examination showing swelling left thigh and gluteal region

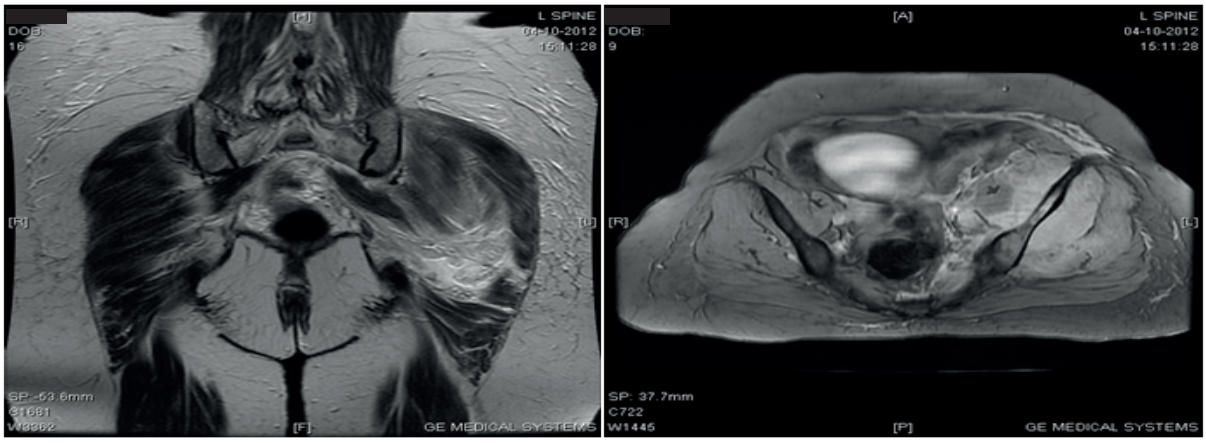


Fig 2: MRI left gluteal region and thigh showing muscle edema and fluid in the intermuscular space

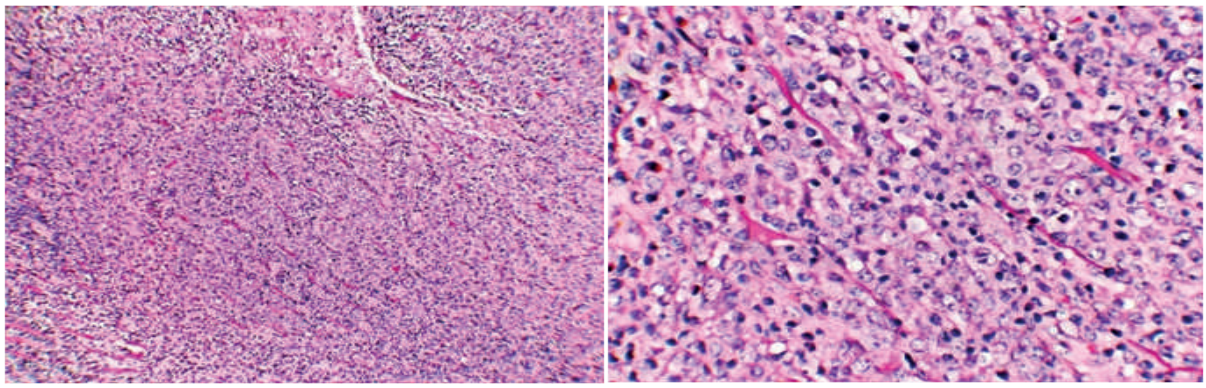


Fig 3: 3a&b: 3a:Muscle histology showing bundles of skeletal muscle infiltrated by atypical cells with eosinophilic cytoplasm and large nucleus (H&E,100X) / 3b:Mitosis and extensive areas of necrosis suggestive of high grade NHL.(H&E,400X)

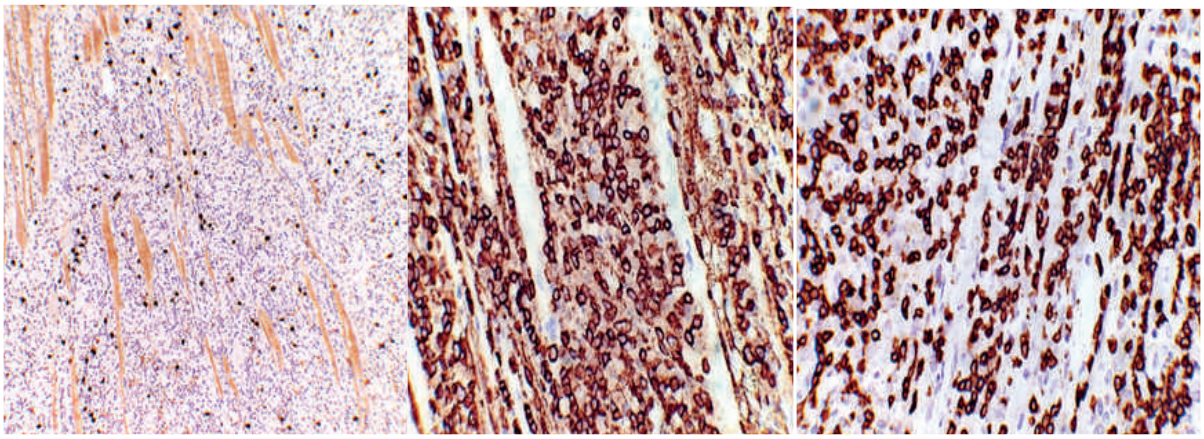


Fig 4a,4b,4c:Immunohistochemistry showing 4a(100X): B cell marker CD 20+ / 4b(400X): LCA-leucocyte common antigen CD 45 +++ / 4c(400X):T cell marker CD 3 ++ positivity suggesting diffuse large cell - T cell lymphoma

Conclusion

Malignancies are an important, although rare, cause of back pain which must be a consideration in patients with certain factors, or in patients who do not respond to treatment. This case report emphasizes the importance of performing a thorough examination of any unexplained complaint of low back, buttock or hip pain,the need for continual re-evaluation and modification of the initial diagnosis, the importance of diagnostic Ultrasound &MRI when clinically indicated and the importance of tissue biopsy in suspected cases.

References

1) Wikipedia-http://en.wikipedia.org/wiki/Non Hodgkin lymphomas - Dorland's medical dictionary

2) Laura Bourdeanu, Rashmi Menon, and George Somlo. Diffuse Large B-Cell Lymphoma with Calf Muscle Localization. Case Reports in Hematology (Internet) 2011[cited 2011]; doi:10.1155/2011/292494: (about 3 pages)

3) Muller A,Ihorst G,Mertelsmann R.Epidemiology of Non Hodgkin's lymphoma (NHL):trends,geographic distribution and aetiology. Ann Hematol.2005;84:1-12.

4) Lopez-Guillermo A,Colomo L,Jimenez M,et al. Diffuse B cell lymphoma:clinical and biological characterization and outcome according to nodal and extranodal primary origin.J Clin Oncol. 2005;23:2797-2804.

5) Paes FM ,Kalkanis DG ,Sideras PA , Serafini AN. FDG PET/CT of extra nodal involvement in Non Hodgkin lymphomas and Hodgkin disease. Radiographics.2010 30(1):269-291.

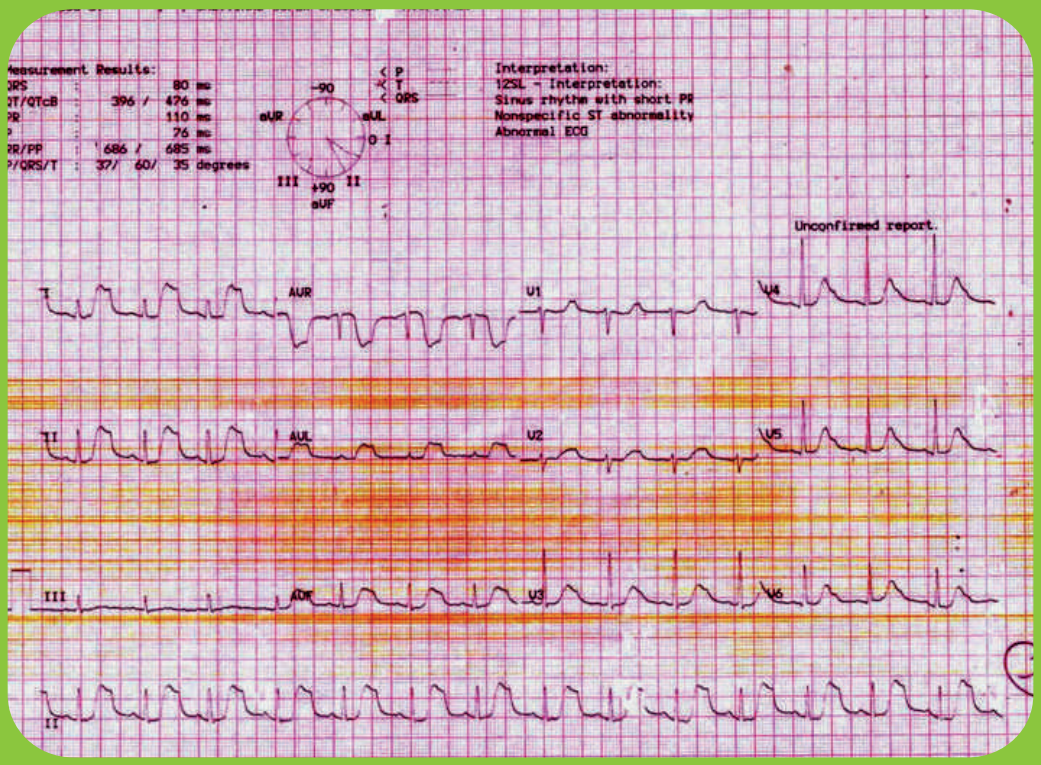
6) De Giorgi S,Piazzolla A,De Giorgi G,Cimmino A,Parisi G,Ricco R :Non hodgkin's lymphoma in the gluteal region-a case report ; Chir Organi Mov:2004 Oct-Dec;89(4):329-38

7) Baddour LM,Haden KH,Allen JW .Primary skeletal muscle lymphoma presenting as refractory cellulitis. Cutis.2001 Sep;68(3):223-6.

8) Zain JM ,O'Connor O,. Targeted treatment and new agents in peripheral T cell lymphoma. International journal of Hematology. 2010;92(1):33-44.

Diagnose the condition

60 year old chronic smoker presented with chest pain radiating to both shoulder ridge for 3 days. ECG was taken



Answer in page no : 16

- Dr. N. Ganesh, Consultant Cardiologist, Chettinad Super Speciality Hospital.

Case Report

Aniridia with Sydney Crease

Dr Srinivasan K*, Dr Thayumanavan**, Dr Padmapriya***

*Prof of Paediatrics and Consultant Neonatologist Chettinad Super Specialty Hospital, ** Prof of Paediatrics and Head of the Dept. of Paediatrics, *** Compulsory Resident Rotatory Intern Chettinad Hospital & Research institute, Chennai, India.

Professor Dr Srinivasan Kitchanan M.D, D.C.H, PGD (NEO) is Consultant Neonatology at Chettinad Health City. He has done his Fellowship in Neonatology from Australia. He headed the Neonatology unit Madras Medical College, at Institute of Child Health, Egmore. He has a special interest in the field of pre-term intensive care and nutrition.

Corresponding author - Dr. Srinivasan. K (srini_10@yahoo.com)

Chettinad Health City Medical Journal 2014; 2(1): 14 - 16

Abstract

Aniridia is abnormal hypoplastic iris reduced to rim of tissues and appears as though there is total absence of iris. It is usually bilateral and is a rare condition bound to be missed during neonatal period due to normal blepherospasm of newborns. Such detection requires protocol guided routine systematic examination aided by a check list. Early detection helps in screening for associated ophthalmic conditions, counseling parents for further investigations, management and to avoid medico legal conflicts. In this case report, bilateral aniridia a rare anomaly is associated with Sydney crease another recognized dysmorphology.

Keywords: Aniridia, Neonatal examination, Sydney crease, Nuclear cataract.

Introduction

The following case has been reported as there are no reports of association of aniridia with Sydney crease and highlights the importance of routine systematic examination based on checklist for newborns in eliciting clinical findings. Missing such a rare condition can have long term diagnostic and prognostic implications. Eye examination in a new born is routinely done but difficulty in opening their eyes results in avoiding further detailed examination. In this case, rare finding Sydney Crease is associated with another rare condition Aniridia.

Case History

A female child, Infant of Gestational Diabetes Mother (IGDM) born to non consanguineous parents delivered by emergency caesarian section to a mother on Insulin, was transferred to nursery for glucose monitoring. Baby's cry, color and activity were good and on enteral feeds. On day 2 of life a routine examination revealed large pupil which lead to further careful examination and diagnostic finding of bilateral Aniridia with minimal iris in the periphery. Further examination revealed the presence of hairy forehead and Sydney's line in the baby's both palms. Other system examination revealed no clinically detectable anomalies. TORCH screening, Ultrasound examination of cranium and abdomen did not reveal any abnormality. Further eye examination by ophthalmologist did not show any other abnormalities and intraocular pressures were normal. Parents were counseled for regular review at child development clinic and for renal ultra-sonography every 3-6 months till 5 years of age and less frequently till 16 years or till genetic test confirms non involvement of extragene[1]. She was also advised routine screening for detection of developing corneal opacity, cataract and glaucoma and avoidance of direct bright lights. Infant on ophthalmic follow up was found developing nuclear cataract at nine months of age.

Discussion

Aniridia is a misnomer because iris tissue is usually present although it is hypoplastic (Fig.1). The condition is bilateral in 98% of all patients regardless of the means of transmission and is found in approximately 1 in 50,000 persons[2]. Aniridia represents a defect of neural crest cell development. In addition to striking absence of iris tissue the other ocular abnormalities frequently seen are nystagmus, fibrovascular corneal pannus, refractive errors, glaucoma, cataract, foveal hypoplasia and optic nerve hypoplasia. In a study of Korean patients, Cataract (82.5%), glaucoma (51.6%), keratopathy (71.6%), and foveal hypoplasia (81.8%) commonly accompanied aniridia. Thirty-four (60.7%) eyes had Visual Acuity less than 20/200 and 20 eyes (35.7%) had Visual Acuity between 20/200 and 20/60[3]. Relative frequencies of the age of patients with aniridia at time of glaucoma diagnosis are as following[4]: from birth to 9 years, 10-19, 20-29, 30-39 were 15%. In the fifth decade i.e. 40-49: 35%, and in 50-59: 5%. Aniridia is an autosomal dominant condition caused by mutation in the PAX6 homeobox gene 11p13 and usually (66%) has no systemic manifestation and classified as Aniridia I[1]. About 30% cases are sporadic, with deletion at 11p13 and classified as Aniridia II. This type has high incidence of associated abnormalities including Wilm's tumour when contiguous oncogene WT1 gene deletion occurs[5] which is known as WAGR syndrome[6], genitourinary abnormalities and mental handicap. Another form of Aniridia classified as Aniridia III is autosomal recessive with mental

retardation, Cerebellar ataxia. In the molecular diagnosis of aniridia, ocular malformation may be regarded as a group of heterogeneous disorders, rather than a single disease entity, associated with mutations in PAX6 a gene responsible for eye development and/or other genes located elsewhere in the human genome as suggested by the fact that there is variability of the phenotype in the presence or absence of PAX6 mutations[7]. Abdominal palpation and/or abdominal ultrasound study should be performed in all new cases and as a part of our follow.

Sydney crease which is proximal transverse crease extending to the ulnar border of the palm in association with Aniridia has not been reported (Fig.2). Prevalence of Sydney creases is 0.19% in a study of South Nigerian population[8]. There is general pattern of increase in prevalence rate in Caucasian when compared to the Orients[8]. Ravindranath et al., reported 3.8% a high occurrence of Sydney crease in their normal control in central India[9] with female preponderance. Dar et al.,[10] reported that dermatoglyphic polymorphism results from the co-operation of genetic, ethno-historic and environmental factors and reported that Sydney crease was significantly present in at risk neonates10, female congenital rubella[11] Leukemia[12,13,14] Trisomy21.[15] The finding was also significantly present in developmental delay and in hyperactive children[16].

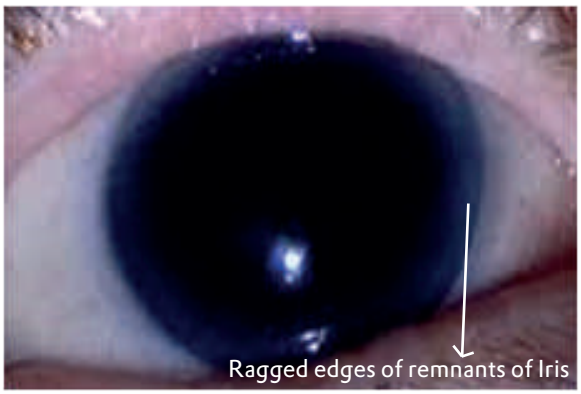


Fig 1: Illustration of Aniridia in newborn

Fig 2: Illustration of Sydney Crease - Two transverse creases extending to ulnar border

There are also reports of Sydney crease in Williams Beuren Syndrome, Fragile X-syndromes, Marfans, Rubeinstein Taybi Syndrome, and Achondroplasia In a follow up study the Sydney crease disappeared in 58.8% of infants by 10 months of age[17].

Studies do not explain the lack of homogeny and in this neonate aniridia and Sydney crease were randomly present and needs further genetic studies. This case report highlights the significance of routine check list based neonatal examination and of follow-up.

References

1) Jack J Kanski: Clinical Ophthalmology; 6th edition 2007: pg 75

2) Tremblay F, Gupta SK, De Becker I, et al. Effects of PAX6 mutations on retinal function: an electroretinographic study. Am J Ophthalmol. 1998;126:211–218.

3) Shin Hae Park, Young Gun Park, Mee Yon Lee, and Man Soo Kim Clinical Features of Korean Patients with Congenital Aniridia Korean J Ophthalmol. 2010 October; 24(5): 291–296.

4) Gramer E, Reiter C, Gramer G. Glaucoma and frequency of ocular and general diseases in 30 patients with aniridia: a clinical study. Eur J Ophthalmol. 2012 Jan-Feb; 22(1):104-10.

5) Forfar and Arneil's textbook of pediatrics, Elsevier; 7th edition. pg 390

6) Kleigman Nelson textbook of pediatrics, Saunders. 17th edition, pg 1712.

7) Lim HT, Seo EJ, Kim GH, Ahn H, Lee HJ, Shin KH, Lee JK, Yoo HW. Comparison between aniridia with and without PAX6 mutations: clinical and molecular analysis in 14 Korean patients with aniridia. Ophthalmology. 2012 Jun; 119(6):1258-64.

8) CA Oyinbo MSc; HB Fawehinmi MD Ph.D; Prevalence of simian and Sydney creases in the Ijaws of South- South Nigeria; The Internet Journal of Biological Anthropology ISSN: 1939-4594

9) D. K. Sharma & Vandana Sharma Prevalences of Simian, Sydney and Suwon Creases and their Association with Each Other, Body Sides, Handedness, Sex and Anomalies, Diseases, Syndromes in a Population of Central India. Int. J. Morphol., 2011, 29(3):1069-1075.

10) Dar H, Schmidt R, Nitowsky HM. Palmar crease variants and their clinical significance: a study of newborns at risk. Pediatr Res 1977; 11:103-8.

11) S.G. Purvis-Smith B.Sc. New England , MargaretA. Menser M.B. Sydney, M.R.A.C.P. Dermatoglyphics In Adults With Congenital

Rubella, The Lancet. 1968; 292 (7560) : 141 - 143.

12) Oorthuys AM, de Vaan GA, Behrendt H, Geerts SJ.; Palmar flexion creases in childhood neoplasia; Cancer. 1979 Feb; 43(2):749-59.

13) Menser MA, Purvis-Smith SG. Dermatoglyphic defects in children with leukaemia. Lancet. 1969 May 31;1(7605):1076-8.

14) Menser MA, Purvis-Smith SG. Dermatoglyphics in leukaemia. Lancet. 1972 Apr 29;1(7757):956-7.

15) Rajangam S, Janakiram S, Thomas IM. Dermatoglyphics in Down's syndrome. J Indian Med Assoc. 1995 Jan;93(1):10-3.

16) Johnson CF, Opitz E.; The single palmar crease and its clinical significance in a child development clinic.; Clin Pediatr (Phila). 1971 Jul;10(7):392-403.

17) Berger, A., Dar, H., Borochowitz, Z. and Winter, S. T. Changes In The Sydney Line During The First Year Of Life. Developmental Medicine & Child Neurology, 1983; 25: 490–492.

Answer to :
Diagnose the condition

ECG was showing diffuse Concave upward ST elevation in limb and chest leads. There were no reciprocal ST changes. No Q waves in any of the leads. Acute MI is the most common diagnosis; but absence of Q waves with diffuse ST elevation without any localization and absence of reciprocal ST depression goes against the diagnosis; patient was diagnosed as having acute pericarditis with clinical history, normal cardiac enzymes and echocardiography. High index of suspicion is required in such cases; or thrombolysis or even anticoagulation in such cases can lead to fatal pericardial bleeding and tamponade.

- Dr. N. Ganesh, Consultant Cardiologist, Chettinad Supert Speciality Hospital.

You can't keep a good guy down

Marihuana (cannabis) is like a good friend with a bad reputation. For most of human history (going back to 7000 BCE), its use has been legal. Even now it has remained the fourth most popular recreational drug (only behind alcohol, caffeine and tobacco). But in 1937, USA decided to make its use illegal for non-medical reasons (even AMA did not fully agree with the decision). But it is bouncing back. A spate of recent studies have re-established its beneficial effects. Even the Food and Drug Administration (FDA) acknowledges that "there has been considerable interest in its use for the treatment of a number of conditions, including glaucoma, AIDS wasting, neuropathic pain, treatment of spasticity associated with multiple sclerosis, and chemotherapy-induced nausea." Now in the latest study published in American Journal of Medicine (May 2013, Vol. 126, No. 5doi:10.1016), Murray A. Mittleman and his colleagues, have claimed potential benefits in patients with obesity and diabetes mellitus. In a study involving 579 current marihuana users and 1975 past users, the authors found that current marijuana use was associated with 16% lower fasting insulin levels (95% confidence interval [CI],), 17% lower HOMA-IR (homeostasis model assessment of insulin resistance; 95% CI) and significant associations between marijuana use and smaller waist circumferences. Getting treated for obesity or diabetes may become a recreation.

Subcellular stress is the key to obesity

Obesity is a major health problem in many parts of the world. Fat, unlike money, is difficult to lose when once it accumulates. Until now, intractable obesity is considered to be due to the development of progressive insensitivity to fat sensing hormone, leptin. But Eduardo A. Nillni, professor of medicine at Brown University, in an earlier study, had also observed low levels of MSH (alpha-Melanocyte-stimulating hormone) in obese rats, particularly after heavy meal. Alpha-MSH has two functions in hypothalamus region of the brain: one is to suppress hunger; the other is to facilitate the production of hormone TRH, which promotes the thyroid mediated calorie burning in the body. Now, in a new study published in Journal of Biological Chemistry, Nillni and his colleagues decided to examine the cause for low levels MSH in rats with diet induced obesity. They found that in obese rats, the endoplasmic reticulum (ER) is stressed and fails to properly assemble the enzyme proprotein convertase 2 (PC2), which is required for the synthesis of POMC, a precursor of MSH. So, low levels of MSH are seen even when leptin levels are adequate and the gene expression for the precursors is normal. Further, if ER stress is treated by giving Tauroursodeoxycholic acid (TUDCA) or, PBA (4-phenyl butyric acid) to the obese rats, MSH levels recovered, So, the root cause of self-perpetuating obesity may be the breakdown of ER protein processing. This explanation is novel and intractable obesity may become treatable in near future (http://news.brown.edu/pressreleases/2013/05/obesity)

- Dr. K. Ramesh Rao

Case Report

Management of Lingual Thyroid by Suprahyoid Approach

*Dr.S.Ramanujam **Dr.R.Karunanithi **Dr.R.Ganesan ***Dr.M.Loganathan

*Assistant Professor, **Professor, ***Associate Professor, Dept. of General Surgery, Chettinad Hospital and Research Institute, Chennai, India.

A General surgeon with excellent academic credentials, graduated from PSG Institute of Medical Sciences, Coimbatore and Thanjavur Medical College respectively for MBBS and MS, and with four and half years of experience as Assistant Professor since April 2009 in General Surgery Department in Chettinad Hospitals, interested in teaching and cricket, passionate about updating regularly in surgical knowledge and skills.

Corresponding author - Dr.Ramanujam S (docramanujam@rediffmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 17 - 18

Abstract

Management of lingual thyroid, a very rare anomaly is often associated with risks of incomplete excision and bleeding by the common intraoral approach. The suprahyoid approach of excision provides clear visualization for complete removal and efficient control of bleeding. After reviewing the literature, we report a case of Lingual thyroid with obstruction managed by suprahyoid approach, a safer technique.

Key words: Lingual thyroid, Surgical techniques, Suprahyoid approach.

Introduction

Embryological complete failure of migration of the median anlage from its origin in the baseof the pharynx resulting in thyroid tissue located at the base of the tongue between the epiglottis and the circumvallate papillae is called as Lingual thyroid[1]. Lingual thyroid is the most frequent ectopic location of thyroid gland. Prevalence rates vary from 1 in 100,000 to 1 in 300,000. Review of literature reveals that only about 400 symptomatic cases have been reported so far. Here we report a case of lingual thyroid excised by suprahyoid approach which is a safer approach.

Case History

A ten year old female child presented with history of severe dysphagia to solid foods. No history of any other significant symptoms of hypothyroidism. Physical examination revealed a midline mass at the base of the tongue (Fig1). Her Ultrasonogram neck revealed non-visualisation of thyroid in its normal anatomical location. TSH (Thyroid Stimulating Hormone) levels were elevated at 8.72 uIU/ml (Reference range: 0.32-6.82uIU/ml). FT4 (Free Thyroxine4) was borderline low at 0.9 ng/dl (Reference range: 0.8- 2ng/ Other biochemical parameters were non-contributory. A radionuclide scan was carried out using I131 suggesting an ectopic thyroid tissue corresponding to swelling in the posterior third of tongue and abse thyroid tissue in its normal location (Fig2). She wa diagnosed as a case of Lingual thyroid with hypothyroidism and placed on L-Thyroxine and brough euthyroid state. She was taken up for complete surgical excision of lingual thyroid by suprahyoid midline approach in view of its safety under general anesthesia. By transverse skin crease incision, skin and subplatysmal flaps raised and dissection continued till hyoid bone. Sub periosteal elevation of muscles attached to hyoid bone was done and suprahyoid muscles split and oral cavity was entered. Using a finger in oral cavity, the mass was pushed through the suprahyoid incision and removed in toto (Fig3). Oral mucosa closed meticulously, muscles repositioned and wound closed. Ryle's tube was placed and retained for 48 hours. Oral feeds started after 2 days and replacement dose of L thyroxine started immediately in postoperative period. Histopathology revealed a nonencapsulated collection of mature thyroid follicles. Patient was followed up for two years needing dosage adjustment of L thyroxine with no wound or surgery related complications.

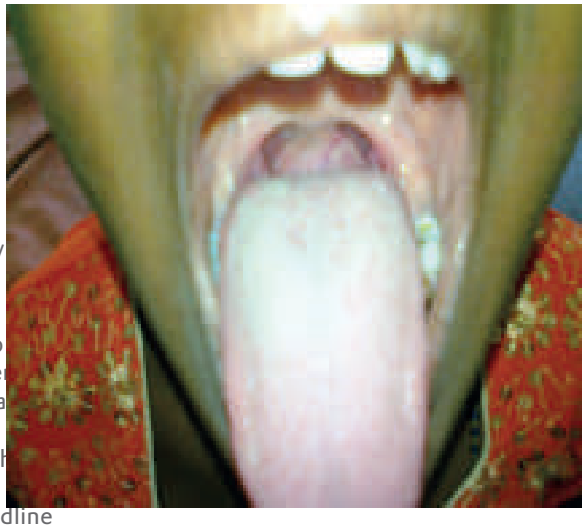


Fig. 1: Preop picture showing a midline mass in the base of tongue

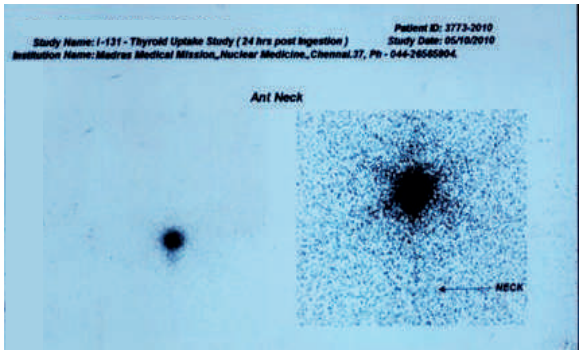


Fig. 2: A radionuclide I 131 scan showing an ectopic thyroid tissue corresponding to the swelling in the posterior third of tongue and absent thyroid tissue in its normal location

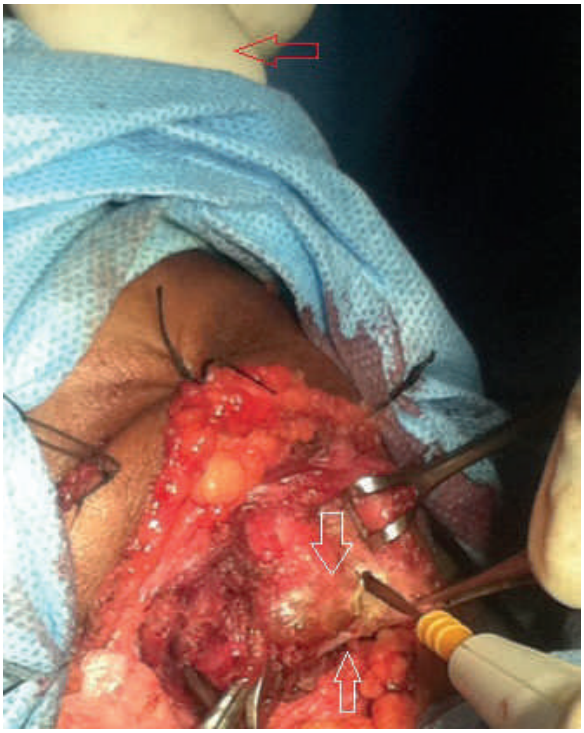


Fig. 3: Intraoperative picture showing Lingual thyroid mass (⇓)pushed into suprahyoid space using a finger guide in oral cavity(⇐). (⇑- hyoid bone)

Discussion

Lingual thyroid is defined as the presence of thyroid tissue in the midline at the base of the tongue anywhere between the circumvallate papllae and the epiglottis. The condition arises from the embryonic failure of normal thyroid tissue to descend from the foramen cecum area of the tongue base through the lower neck, presenting as a lobular midline mass in the mucosal surface of the tongue base[2]. Larger lesions can interfere with swallowing and breathing, but most patients are unaware of the mass at the time of diagnosis. The ectopic thyroid secretions are not adequate to maintain a euthyroid state. Upto 70% of patients with lingual thyroid have hypothyroidism and 10% suffer from cretinism[3]. Diagnosis is established by a justifiably strict criterion which includes either histologic confirmation of the lesion or the development of hypothyroidism after removal[4]. Now as an alternative diagnostic test the concentration of I131 by the tumor and its absence in neck is used[5]. At least one of these criteria must be met before a tumor may be classified as lingual thyroid. Levothyroxine therapy corrects hypothyroidism and also induces shrinkage of lingual thyroid[6]. Occassionally large blood vessels are present on the surface of lingual thyroid tissue, predisposing to ulceration or hemorrhage. When symptoms of bleeding or obstruction appear, therapy by means of surgery or radioiodine ablation is warranted. Surgical excision is an effective treatment for lingual thyroid in patients with obstructive symptoms. The surgical treatment approachesof the lingual thyroid described are transoral, transmandibular- translingual, Lateral pharyngotomy and suprahyoid approaches[7].

Transoral approach is the commonest but with higher complications, inadequate exposure leading to inadequate removal and bleeding being the most common. Trans mandibular/translingual and lateral pharyngotomy approaches are more morbid and has restricted indication[8]. Suprahyoid approach is a safer alternative inspite of external scar and extensive dissection. With the surgical knowledge of neck dissection this approach makes complete excision easier which in turn prevents recurrence and bleeding. In our patient once the diagnosis of Lingual thyroid was established and after bringing her to euthyroid state, complete excision of the lingual thyroid was done by suprahyoid approach with L thyroxine supplementation and two years follow up.

References

1) Ulrich, Henry F. Lingual thyroid.Ann. Surg.1932;95(4):503-7.

2) Williams JD, Sclafani AP, Slupchinskij O, Douge C. Evaluation and management of the lingual thyroid gland. Ann Otol Rhinol Laryngol 1996;105:312-6.

3) Neinas FW, Gorman CA, Devine KD, Woolner LB. Lingual thyroid: Clinical characteristics of 15 cases. Ann Intern Med 1973;79:205.

4) Montgomery ML. Lingual thyroid: A comprehensive review. West J Surg 1935;43:661, 44:54,122,189,238,301,373,442.

5) Kansal P, Sakati N, Rifai A, Woodhouse N. Lingual thyroid. Diagnosis and treatment. Arch Intern Med. 1987;147(11):2046-48.

6) Elprana D, Manni JJ, Smals AG. Lingual thyroid.ORL J Otorhinolaryngol Relat Spec 1984;46:147-52.

7) Grant E Ward, James RCantrell,Warde B. Allan. The surgical treatment of lingual thyroid. Ann. Surg. 1954;139(5):536-44.

8) Vairaktaris E, Semergidis T, Christopoulou P, Papadogeorgakis N, Martis C. Lingual thyroid: A new surgical approach—A case report. J Craniomaxillofac Surg 1994;22:307.

Case Report

A Case of Central Giant Cell Granuloma Involving the Maxillary Sinus Clinically Masquerading as a Malignant Neoplasm

*Ramesh V, **Sriram K , ***Arunprasad G

*M.D.S, Dean, Professor & HOD 1Dept of Oral pathology and Microbiology, Mahatma Gandhi Post Graduate Institute of Dental Sciences (MGPGI) Puducherry, India. , **M.D.S, Senior Lecturer, Dept of Oral and Maxillofacial Pathology, Chettinad Dental College and Research Institute (CDCRI)Chennai, India. ***M.D.S, Senior Resident, Jawaharlal Nehru Institute of Post Graduate Medical Education and Research (JIPMER), Puducherry, India.

Professor Dr. V. Ramesh is currently serving as a Dean and HOD (Dept of Oral Pathology and Microbiology) in Mahatma Gandhi Post Graduate Institute of Dental Sciences (MGPGI), Pondicherry, Pondicherry University. He is one of the senior most and well known Oral & Maxillofacial Pathologists in India. He completed his under graduation and post graduation from Govt Dental College, Chennai, Tamil Nadu. He has many publications to his credit in international and national journals and has delivered many orations as an invited guest speaker in various dental and medical conferences. He served as the President of Indian Association of Oral and Maxillofacial Pathologists (IAOMP 2009-2010) and organised the First International Conference of IAOMP at Chennai.

Corresponding author - Dr.K.Sriram (ksriramds@gmail.com)

Chettinad Health City Medical Journal 2014; 2(1): 19 - 22

Abstract

Central giant cell granuloma is traditionally considered to be a non neoplastic bone lesion. They usually present as a slow growing asymptomatic lesion involving the jaw bones i.e. maxilla and mandible. This paper presents an interesting case of a central giant cell granuloma in a 23 year old female that clinically presented as a swelling in the left side posterior hard palate region and radiographically with complete obliteration of the left maxillary sinus, with extension to infratemporal fossa region mimicking a malignant neoplasm. Controversy surrounding the pathogenesis, histopathological differential diagnosis and evolving differences in potential treatment modalities for this interesting pathology has also been discussed.

Key words: Giant cell granuloma, Jaw bone, Maxillary sinus, Corticosteroids

Introduction

Central giant cell granuloma (CGCG) is a localised benign but sometimes aggressive osteolytic lesion, basically consists of fibro vascular connective tissue with actively proliferating fibroblasts related spindle shaped cells and multinucleated giant cells as its primary cellular components[1]. Most of the CGCGs are asymptomatic slow growing lesion, usually diagnosed during routine radiographic examination or painless expansion of the bone in patients less than 30 years of age. Females are more often affected than males and approximately 70% cases arise in the mandible, followed by the maxilla. Lesion involving the maxillary sinus is extremely a rare occurrence[2]. This article reports an extensive case of CGCG involving the left posterior maxillary alveolus, maxillary sinus and infratemporal fossa region and its management with discussion on the controversies surrounding the pathogenesis, histopathological differential diagnosis or closely related entities and different current potential treatment option available for this entity.

Case description

A 23-years-old female patient reported with a chief complaint of swelling associated with intermittent pain in the left palatal region, approximately for two months. Swelling was initially smaller in for and had progressed to the present size. No history of numbness or abnormal sensation was reported. Clinical examination revealed a facial asymmetry due to a mild, diffuse swelling involving the left malar region. The texture and colour of the overlying skin was normal.

Intraoral examination revealed an obvious swelling involving the left side posterior hard palate region, of size approximately 5X4 cm, extending from second premolar to maxillary tuberosity region and medially upto the mid palatal raphae. The colour of the swelling was normal as that of adjacent mucosa except for the focal redness at the posterior aspect in an otherwise smooth lesion (Fig1). Generally the consistency was soft and boggy with mild tenderness on palpation.

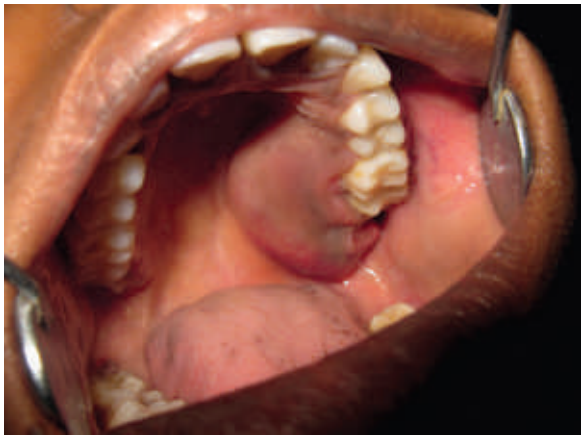


Fig 1 : Well defined smooth surface swelling involving left posterior palate

Thermal vitality test was positive in all maxillary posterior teeth. Panoramic radiograph (OPG) showed a diffuse haziness on the left side posterior maxilla and maxillary sinus region without any teeth displacement and root resorption. (Fig2)

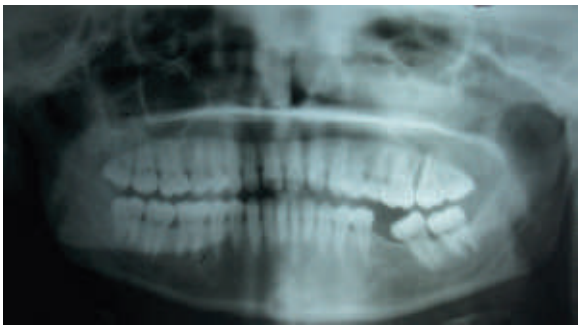


Fig 2 : OPG showing diffuse haziness on the posterior left maxilla and maxillary sinus region (Note – No root resorption or displacement is evident)

CT scan showed an expansile lytic lesion with a thin ossified rim measuring 40×34×38 mm involving the left maxillary antrum and the alveolar process of left maxilla with extra osseous component in the left infratemporal fossa region (Fig3). Deviation of the nasal septum was also noticed towards the right side.

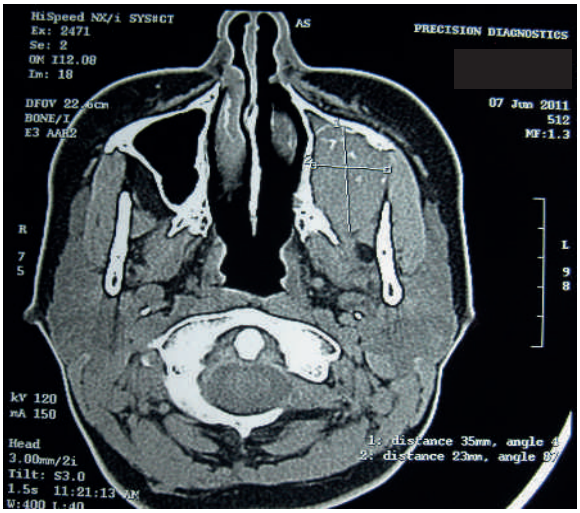


Fig 3 : CT scan showing obliteration of left maxillary sinus with destruction of posterior wall of the sinus (Before steroid injection)

Based on the clinical and radiographic presentation, a provisional diagnosis of primary malignancy of left maxillary sinus region or a salivary gland malignancy extending to involve the maxillary sinus and infratemporal fossa region was made. The differential diagnosis includes ameloblastoma, odontogenic myxoma, odontogenic keratocyst, ossifying fibroma and hemangioma.

An incisional biopsy under local anaesthesia was taken from the left maxillary sinus by creating a small window through the anterior wall. Profuse bleeding was encountered during biopsy and the hemostasis was achieved with the surgipack. Histopathological examination showed proliferating plump spindle cells and unequally distributed multinucleated giant cells in the fibrous stroma (Fig4). The giant cells were varying in shape, size, and consists of varying no of nuclei usually ranging from 10-15. Focal hemorrhagic areas and peripheral reactive bone were present. No evidence of pleomorphism, abnormal nuclear cytoplasmic ratio, and atypical mitotic figures were noticed.

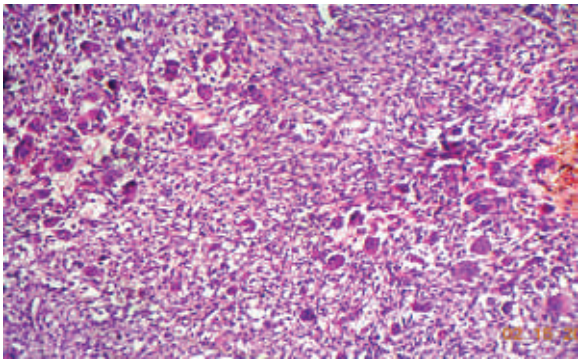


Fig 4 : Numerous unevenly distributed multinucleated giant cells within a background of plump proliferating spindle cells (H&E × 20)

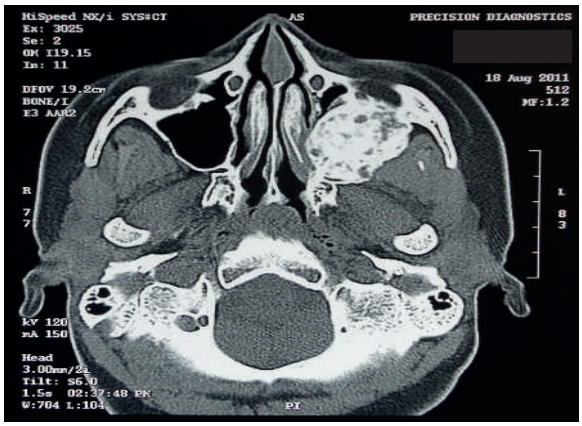


Fig 5 : CT scan showing diffuse radio opaque areas within the lesion after intralesional corticosteroid injection

Patient was referred for serum calcium and parathormone level to rule out hyperparathyroidism and the report was found to be within the normal range.
Based on the histopathology and serum biochemical investigation, a diagnosis of central giant cell granuloma was given.

Considering the posterior extension of the lesion till the infratemporal fossa region and the macroscopic nature of CGCG i.e. it does not grow as a uniform solid mass, with the stroma composed of loose fibrous tissue intermixed with abundant hemorrhagic areas, the initial treatment plan was to consolidate the lesion and possibly decrease lesion size using intralesional corticosteroids followed by the surgical removal of the lesion.

Hence the patient was started on with the initial treatment of intralesional corticosteroids as recommended by Terry and Jacoway[3] i.e. equal parts of Triamcinolone acetonide (10mg/1ml) and local anaesthetic (2% lignocaine with 1 in 200,000 adrenaline) 2ml per 2cm of the lesion was given as weekly regimen for 6 weeks.

Patient was carefully monitored for steroid induced side effects and the course was fairly uneventful. CT scan was taken one week after the last injection (7th week from starting) and on interpretation revealed diffuse radio opaque areas within the lesion which indicate consolidation of the lesion compared to the initial presentation (Fig5). Patient was informed about

the improvement, surgical removal of the lesion was planned and through an intraoral approach the lesion was removed thorough surgical curettage (Fig 6&7) Patient is under regular follow up and till to date there is no recurrence.

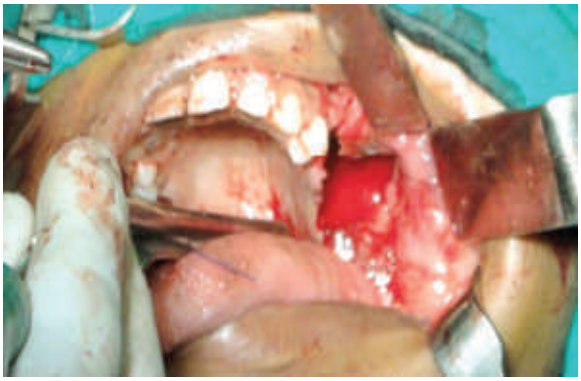


Fig 6: Intraoral surgical curettage of the lesion

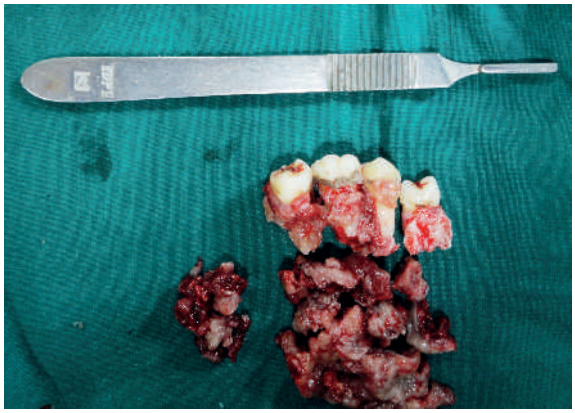


Fig 7 : Gross specimen after surgical removal of the lesion

Discussion

The term central giant cell reparative granuloma was initially coined by Jaffe in 1953 to describe a tumor of the jaw bones that had previously been diagnosed as giant cell tumor of bone. In 1962, Ackerman and Spjut described the first two cases involving the small tubular bones of hand, for which they coined the term "giant cell reaction"[4]

The most interesting aspect of this pathology is that its etiopathogeneis which still remains elusive. Earliest theories has suggested that the lesion may be derived from the odontoclasts that were responsible for the resorption of the deciduous teeth based on the facts that they occur more commonly in the deciduous teeth bearing regions of the jaws and in most cases the period of onset was found to be either during the time of exfoliation or few years after the exfoliation of deciduous teeth.

Traditionally it has been hypothesized that the giant-cell-rich areas represent a reaction to recent haemorrhage due to trauma and the fibroblastic component represents the older or the healing part of the lesion which lead to its description by the term called "Central giant cell reparative granuloma". But the fact that almost every lesion does not regresse without an intervention, lead to the removal of the term "reparative" from its original description [5]

J.A Regezi et al has speculated that there could be separately a reactive and neoplastic form or a subset of tumours that behave as a neoplasm developing from a reactive lesion through an epigenetic event in spindle mesenchymal cells[6]. Recently, cytogenetic abnormalities have been identified in a giant cell granuloma, raising the possibility that this tumor may indeed be neoplastic[7].

Though little is known about the exact etiology and the nature of CGCG, recent molecular studies have shown that the active proliferating component in the CGCG is the fibroblast related spindle cells which secrete cytokine such as monocyte chemoattractant protein (MCP) that recruits monocytes from the blood vessels which fuses to form the multinucleated giant cells i.e osteoclasts[6].

And also it has been shown that osteoclastogenesis is under the influence of osteoprotegerin and its antagonist receptor activator of nuclear factor of kappa B (RANK) ligand via an osteoclast receptor known as RANK[6].

From a differential diagnosis standpoint, severel lesions have to be considered when entertaining a diagnosis of CGCG.

Aneurysmal bone cyst (ABC) tends to occur in the same age group and also has slight female predilection but the most striking feature in the ABC is the presence of large blood filled spaces and thrombosis. These blood filled spaces are typically bordered by fibrous septa of cellular tissue that may consist of osteiod or woven bone which are oriented along its long axis.

The microscopic and radiographic feature of brown tumor of hyperparathyroidism is nearly identical and is therefore necessary always to rule out primary and secondary hyperparathyroidism viz due to parathyroid disease and chronic renal failure by determining the serum calcium, phosphorus and parathormone level.

A diagnosis of cherubism should be entertained whenever evaluating central giant lesion of the jaw but the classical multifocal involvement and the age of occurrence in a childhood usually between 2-7 years old, allows easy distinction of this entity from CGCG.

Though surgical curettage, excision or resection were considered as the conventional treatment modalities for CGCG, several medical treatment options are now available, mainly due to the current understanding about the molecular biology of the cellular components of the lesions.

One of the potential medical treatment options that have been tried in CGCG either alone or in combination with surgery and reported with a reasonably good success rate is intralesional corticosteroids. The rationale of using steroid in the CGCG is based on the fact that the giant cells express glucocorticoid receptors and it has been hypothesized that steroid inhibits the production of extracellular bone resorption mediating

lysosomal proteases by giant cells and also induce apoptosis of osteoclast (giant) like cells[8].

Though few authors[9] have reported, complete regression of the CGCG with intralesional steroid alone, in the present case the steroid was given mainly to consolidate and decrease the lesion size to facilitate its complete surgical removal.

The option between steroid alone or combined surgical and steroid treatment for CGCG entirely depends on the individual case, and how well the patient responds to the initial course of steroid injection.

Adolescent patient, moderately sized lesion in the site that can be evaluated with the simple radiograph (less exposure & cost effective), good patient compliance, with good treatment response, intralesional corticosteroid alone may be a potential option and can be given till the complete regression of the lesion. Practically the most important problem with this steroid alone option is the constant follow up that may extend for 3-6 years with the associated chance for lack of patient compliance.

The fact that the multinucleated giant cells in CGCG are basically osteoclasts and expresses calcitonin receptors forms the basis for the use of calcitonin in CGCG to inhibits the giant cell function. Harris M[10] reported four cases of CGCG treated by calcitonin where a complete remission was achieved. However the literature evidence shows that therapeutic response to calcitonin is variable and is influenced by mode of administration i.e. intravenous, subcutaneous or as nasal spray.

Presuming CGCG as a vascular lesion Interferon α also has been used in the treatment of CGCG[1]. Another promising treatment modality for CGCG in future may be administration of osteoprotegerin which is an antagonist for RANK ligand and by binding to RANK receptor on osteoclasts (giant cell) potentially inhibits its function. i.e. bone resorption[11]. In future further research should focus on gene and protein expression in CGCG to develop new medical therapeutic agents with predictable results.

Conclusion

It is essential to be aware of the fact that CGCG rarely can present as an extensive lesion involving maxillary sinus, infratemporal fossa region mimicking malignant neoplasm, this possibility should also be considered in the differential diagnosis for similar clinical presentation.We also favour the use of intralesional corticosteroids as an initial treatment option for CGCG especially for an extensive lesion to consolidate and decrease the size of the lesion to facilitate the surgical removal and to reduce the post surgical morbidity.

References

1) Neville BW, Damm DD, Allen CM, Bouquot JE. Central giant cell granuloma. In: Oral and maxillafacial pathology. 3rd ed.Philadelphia: Saunders, An imprint of Elsevier; 2009.pp 626-9.

2) C Leon Barnes, John W Eveson, Peter A Reichart, David Sidransky. Central giant cell lesion. Jundt G. In: World health organisation classification of tumours, Pathology and genetics, Head and neck tumours. 1st ed. Lyon, France: IARC Press; 2005.pp 324.

3) Terry BC, Jacoway JR. Management of central giant cell lesions. Oral and Maxillofac Surg Clin North Am. 1994; 6:579-600.

4) Dorfman HD, Cerniack B. Giant cell lesions. In:Bone tumors, 1st ed.Missouri: Mosby publications;1998.pp598-603.

5) Silverberg S.G, Delellis R.A. Frable W.J. Central giant cell granuloma. In Principle and practice of surgical pathology and cytopathology. 3rd ed. New York :Churchhill Livingstone; 1997.pp1445-1446.

6) Regezi J.A, Pogrel M.A. Comments on the pathogenesis and medical treatment of central giant cell granuloma. Journal of oral and maxillofacial surgery 2004; 62(1):116-118.

7) Buresh CJ,Seemayar TA , Nelson M , Neff JR, Dorfmann HD, Bridge JA . t (X;4) (q22; q31.3) in giant cell reparative granuloma Cancer gene Cytogenet 1999;115(1):80-81

8) Al-Ahmad HT, Anabtaubi M, Salfiti F,Eid RA. Combination of surgery followed by intralesional steroids in treatment of aggressive mandibular giant cell granuloma. A case report . Jordan medical journal. 2009; 43(3): 231-236.

9) Mohanty S , Jhamb A. Central giant cell lesion of mandible managed by intralesional triamcinolone injections. A report of two cases and literature review. Med Oral Patol Oral Cir Buccal. 2009;14(2):98-102.

10) Harris M. Central giant cell granuloma regresses with calcitonin therapy. Br J Oral Maxillofac Surg 1993; 31:89-94.

11) Fili S, Karalaki M, Schaller B. Therapeutic implications of osteoprotegerin. Cancer Cell International 200 9; 9:26.

From the Pages of History

Hippocrates and his Oath

Dr. Ramesh Rao, Professor and HOD, Dept. of Pathology, Chettinad Hospital and Research Institute, Chennai, India

Chettinad Health City Medical Journal 2014; 2(1): 23 - 24

Hippocrates (460-370 BCE) "At least, do no harm"

The Hippocratic Oath recited at the medical graduation ceremonies the world over is probably the oldest extant rite of passage. Although the exact date of its composition is not known, it is at least, 2400 years old. According to Orr et al. (1997) , the content of the traditional Hippocratic Oath can be divided into 12 items:Pledge to God: "I swear by Apollo the physician…" Pledge to teachers:promise of collegiality and financial support; Commitment to students: promise to teach those who swear the Oath; pledge to patients:promise to use "ability and judgment." Appropriate means: use of standard "dietary" care; Limits on means: originally prohibited surgery for renal stones, by deferring to those more qualified; Appropriate ends: the good of the patient &not the physician; Limits on ends: originally prohibited abortion and euthanasia; Justice: "avoiding any voluntary act of impropriety or corruption." Chastity: originally prohibited sexual contact with patients; Confidentiality: not to repeat anything seen or heard; Accountability: Prayer that the physician be favored by the gods if the Oath is kept, and punished if it is not kept.

As little is known about the original Oath, it is not clear how widely it was used in its time.Because of its supplication to pagan gods (Apollo, Asclepius etc.) at the opening, it did not become popular in western world until the middle ages, when it was rediscovered and modified to conform to monotheistic Christian doctrines. The first documented use of the oath was at the University of Wittenberg, Germany, in 1508 . The Oath was finally translated to English only in eighteenth century.

Origin of the Oath

Although it is attributed to Hippocrates, he might not have been its author. After a scholarly analysis, Ludwig Edelstein (1902-1965), a History of Medicine Professor at Johns Hopkins University, showed that the Hippocratic Oath may actually have been the work of the followers of Pythagoras of Samos, who lived a generation before Hippocrates (Orr et al., 1997). There is another reason why it may not be his work. Hippocrates was opposed to religion based medical practice and he would not have authored an Oath that begins by swearing to the gods of medicine (Roger Bulger in Hippocrates Revisited).

Why Retain His Name?

Very little authentic information is available about Hippocrates. Most of what we know come from legends that began to circulate after his death. These suggest that he was renowned physician during his own lifetime and had

many admirers, who praised and respected him and his work. Even Plato and Aristotle who came after him spoke of him with great respect. But he also had many detractors who accused him of burning down the medical library in Cos in order to eliminate competing medical traditions. But we get true understanding of his greatness from his works and not from these legends. These works consisting of more than fifty texts and essays display an altogether different outlook, to the prevalent one at that time, towards the practice of medicine - one that emphasizes nature over philosophy, observation over theory, and the patient over the physician's self-interest. Hippocrates rejected the medical systems based on philosophy and religion and promoted a system based on empirical observation. He insisted on patient-oriented medicine and recommended treatment modality that caused the least damage (His motto: At least, do no harm!). It is because of these progressive ideas that he is regarded as "Father of Medicine" and rightly so.

Its Relevance

In the last century, the traditional Hippocratic Oath has been extensively criticised for being outdated and failing to incorporate many of the new ideals such as societal or legal responsibilities, research ethics, and accountability in group practice. While in many cases, the traditional Oath is suitably updated to address these concerns, other professional medical oaths are also beingused including"the Declaration of Geneva" (written in1948—and revised in 1983—in response to the medical crimes committed during the Nazi regime in Germany) and the oath written in 1964 by Louis Lasagna , Academic Dean of the School of Medicine at Tufts University.

References

1. Hulkower R. (2010). The History of the Hippocratic Oath: Outdated, Inauthentic, and Yet Still Relevant. The Einstein Journal of Biology and Medicine25: 41-44

2. Orr RD, PangN, PellegrinoED, SieglerM. (1997). Use of theHippocratic Oath: A review of twentieth century practice and a content analysis of oaths administered in medical schools in the U.S. and Canada in1993. J Clin Ethics 8:377-88.

3. Smith L. (2008). A brief history of medicine's Hippocratic Oath, or how times have changed. Otolaryngol Head Neck Surg 139:1-4.

http://en.wikipedia.org/wiki/Declaration_of_Geneva

http://ethics.ucsd.edu/journal/2006/readings/Hippocratic_Oath_Modern_Version.pdf

Two rights, when combined, may be wrong

Simvastatin, the second most widely prescribed drug worldwide, is often recommended for obese diabetics to lower their serum cholesterol and prevent heart disease. Obese diabetics also benefit from regular exercise which improves their overall fitness. So, it makes a lot of sense to combine these two therapeutic approaches to amplify the benefit. But only, it doesn't!. In a study carried out in University of Missouri, John Thyfault and his colleagues discovered that simvastatin hindered the positive effects of exercise for obese and overweight adults. The study was done on 37 sedentary obese individuals between the ages of 25 and 59. All the participants were made to go through the same exercise regimen for 12 weeks; however, 18 of them also received 40 mg of simvastatin. When the cardiopulmonary fitness and skeletal muscle mitochondrial content were measured at the end of 12 weeks, the improvement was significantly less in those who also received statin (1.5%) compared to exercise only group (10%). Statin seems to adversely influence the exercise outcomes in these patients. The Authors, while acknowledging the need for additional study, caution against combining these two therapeutic options. They however concede that the sequential use of these options needs to be evaluated. The study is published in the latest issue of Journal of the American College of Cardiology, (2013; DOI:10.1016/j.jacc.2013.02.074)

- Dr. K. Ramesh Rao

Instruction to Authors

Chettinad Health City Medical Journal 2014; 2(1): 25 - 26

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Lace your junk food with fish oil!

Considerable research during the last decade has shown that junk food (rich in saturated fats and refined sugars) could disrupt neurogenesis and turn your brain into a junk yard. Hormones, which normally protect neurons and stimulate their growth, are prevented from reaching the brain by the high levels of triglycerides and pro-inflammatory molecules that occur following consumption of high-fat diet. Dr. Lucy Pickavance and colleagues, from Institute of Ageing and Chronic Disease, the University of Liverpool, wanted to find out if omega-3 fatty acids (from fish oil) can minimise the harmful effects of junk food. So, they did a meta-analysis of 185 research publications on the subject and found that although the omega 3 fatty acids did not have a direct impact on either neurogenesis or weight loss, they appear to restore normal function by interfering with the production of the inflammatory molecules, suppress triglycerides, and return the nerve growth factors to normal. Omega 3s seem to mimic the effects of calorie restrictive diets. It makes a lot of sense to include them in your regular diet, particularly if you are a junk food addict (British Journal of Nutrition, 2013; 109 (09): 1573 DOI: 10.1017/S000711451200579X)

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