Infectious Diseases (ID) & Microbial Newsletter

BHARATI HOSPITAL & RESEARCH CENTRE

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From the Editor's desk...

Infectious disease and microbiology are two sides of a coin with one complementing the other.This newsletter is an attempt to promote awareness about infectious diseases amongst students and medical professionals. It also gives microbiological data inputs that would aid clinicians in treatment of various infectious diseases.

We are proud and privileged to have Dr Rajeev Soman a renowned authority on infectious diseases join the team as an editor to guide and mentor all those who are interested in the subject.

Interesting cases with their discussion will be followed by microbiology data.

Dr K K Lahiri

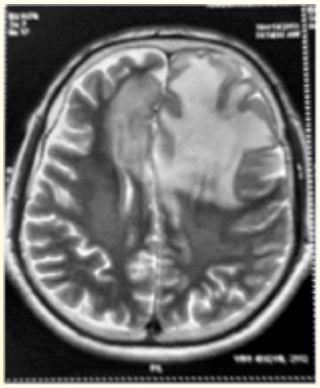
INTERESTING CASE

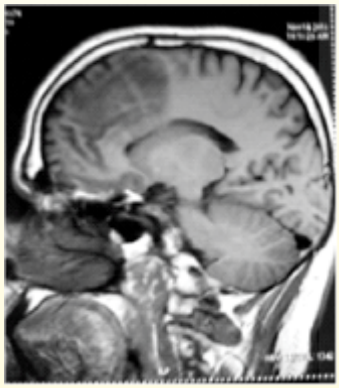
CNS mass lesion in HIV-1 patient : Persistence pays !

Dr. ShwetaPanchakshri (DNB Family Med, Fellow in infectious diseases at Bharati Hospital)
Dr. Bharat Purandare (Asso. Prof, Department of infectious diseases, Bharati Hospital)
Dr. Vishal Rokade (Asso. Prof, department of Neurosurgery, Bharati Hospital)

Mr SK, 46 years/ male, was being treated elsewhere for- altered sensorium, reduced speech, fecal and urinary incontinence, headache, vomiting, loss of appetite for 3 weeks, gradually progressive in nature. In past history- He was a known case of HIV-1 from 2011 was on TDF+3TC+EFV. CD4- initially 100 gradually increased to 700 with viral load undetectable. It again declined to 237 despite full adherence to treatment and with viral load remaining undetectable. Also had abdominal TB in 2011-Completed ATT for 18 months. Recently detected DM with HBA1c 9.3%- presently on insulin

Outside workup in 7 days - MRI brain with contrast -





Extensive lesion involving periventricular and subcortical white matter of left frontal lobe, Left temporal lobe along the external capsule, with scalloping.Involvement of corpus callosum seen.Significant edema present. Mild mass effect present. Post contrast images showed irregular, nodular, predominantly peripheral enhancement. CSF studyshowed- Clear, Glucose- 104, protein- 60.8, Total nucleated cells- 13 (100% lymphocytes). CSF was negative for HIV-1 RNA PCR/ Gene Xpert/ Cryptococcal antigen/ JCV DNA quantitative/ VZV PCR. CBC- Hb- 12, WBC- 11720, CD4- 237(21.5%), CD8- 558.

On admission to our hospital, the differential diagnosis was kept as follows-

		Altered sensorium	Unexplained CD4 decline	Signs of raised ICP	MRI findings contrast enhancement	CSF findings
1	HAND-CNS escape	✗	✗	✗	✗	✗
2	PML	✗	✗	✗	✗	✓
3	Toxoplasma Encephalitis	✓	✗	✓	✓	✗
4	TB Encephalitis	✓	✓	✓		
5	Primary CNS Lymphoma	✓	✓	✓	✓ (with corpus callosum involvement)	✓

Taken altogether lymphoma was suspected. Serum LDH WAS 2113 and Brain biopsy was asked for. Patient underwent left frontal partial lobectomy. Initial histopathology report showed inflammatory lesion suggestive of encephalitis which was not consistent with the clinical diagnosis and radiological findings. Hence deeper cuts of the specimen were asked for which revealed perivascular infiltrates with mononuclear cells, which were found CD20 positive by IHC. Diffuse large B cell lymphoma was found (non GCB type: Hans algorithm). This confirmed the diagnosis of HIV associated lymphoma and was referred to the oncologist.

Differential diagnosis of CNS mass lesion in HIV

HIV TYPE-1 ASSOCIATED NEUROCOGNITIVE DISORDER-

- Clinical triad-
  1) Cognition- Forgetfulness, mental slowing, decreased concentration
  2) Behavioral- Apathy, Social withdrawal, lack of spontaneity
  3) Motor- Gait instability, Poor co-ordination, Leg weakness
  Altered mental status is generally late finding.
- Risk factors- AIDS illness, increased age, survival duration, lower nadir of CD4 count(<200), Higher viral load
- CSF- non specific, mild lymphocytic pleocytosis
- MRI- subcortical atrophy
- Brain biopsy- not indicated. (postmortem examination shows-encephalitis with multinucleated giant cells, mononuclear infiltrates.
- Treatment- ART with good CPE (CSF penetration effectiveness score

PML- PROGRESSIVE MULTIFOCAL LEUCOENCEPHALOPATHY-

- Clinical presentation- Bilateral LL weakness, visual symptoms, gait ataxia, altered mental status
- Risk factors- CD4 count <200,
- CSF- nonspecific, CSF- JCV PCR +
- MRI- no post contrast enhancement unless PML IRIS, no perilesional edema
- Brain biopsy- demyelination in subcortical region, large hyper-chromatic nuclei stain positively for JCV on IHC.
- Treatment- no specific treatment benefit compared to ART

TOXOPLASMA ENCEPHALITIS

- Clinical presentation- headache, confusion, fever, seizures, other focal neurological signs. Altered sensorium usually not seen.
- Risk factors- CD4 count <200 (uncommon if CD4 count >200)
- Sr Anti toxoIgG can be detected
- MRI-multiple ring enhancing lesions with surrounding edema and can produce mass effect
- Brain Biopsy- usually not required. Usually shows necrotic abscesses with blood vessel thrombosis and necrosis, cysts containing bradyzoites with numerous active tachyzoites.-

TUBERCULOMA-

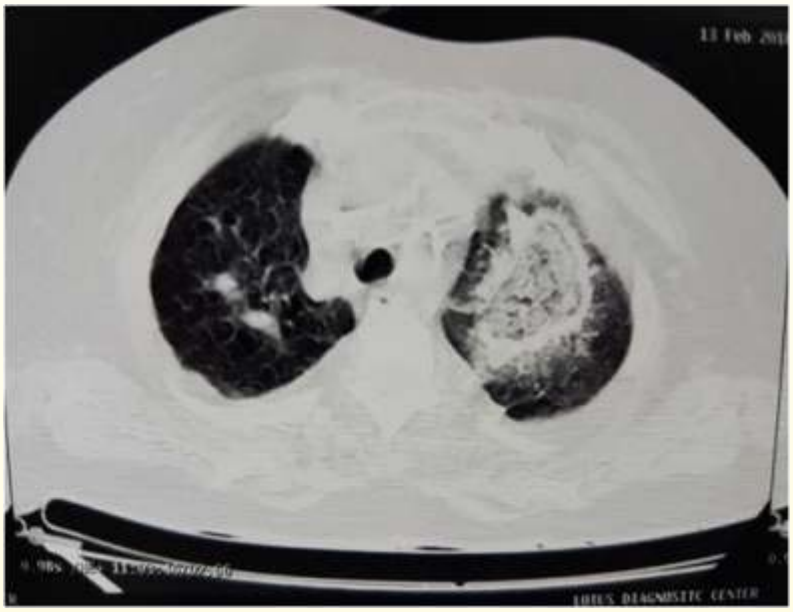
- Clinical Presentation- seizures
- MRI- solitary or multiple, avascular masses with ring enhancement after contrast, with moderate perilesional edema. MR Spectroscopy-lipid lactate peaks seen
- Biopsy- granulation tissue with central necrosis

CNS LYMPHOMA-

- Clinical presentation-Alerted mental status, features of encephalopathy.
- Risk factors- Immunocompromised state, prolonged corticosteroids (>6 months), low CD4 count. EBV association.
- MRI- Involvement periventricular white matter with corpus callosum. Perilesional edema present. Homogeneous post contrast enhancement, with diffuse infiltration.
- CSF atypical cells seen.

PICTORIAL CME

IMAGE A: Identify the radiologic sign.



Case courtsey: Dr. Anita Anokar (Asst prof, Departement of Pulmonary Medicine, Bharati Hospital)

(Answers to be found elsewhere in this issue of Bharati Hospital ID newsletter)

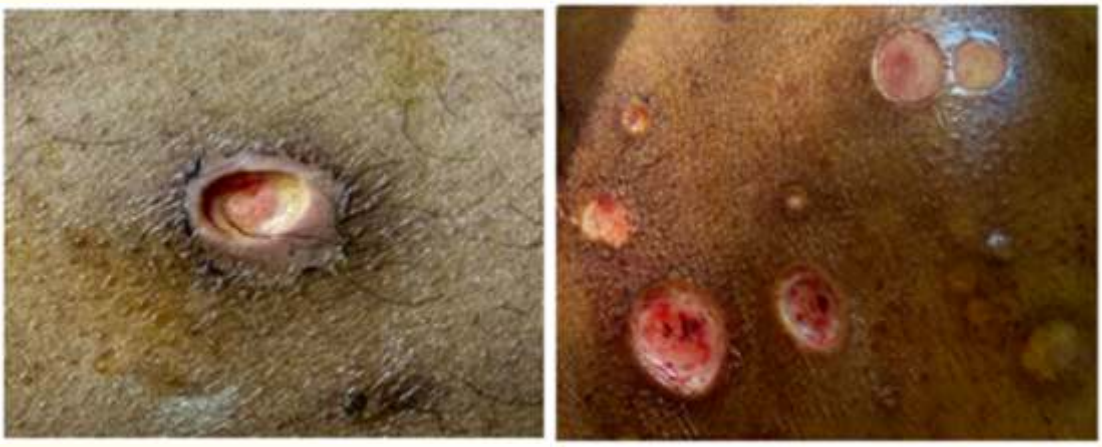
INTERESTING CASE

MSSA, MRSA and GNB nibbling at the same patient!

Dr. Sujata Rege (DNB Family Med, Former Fellow in infectious diseases at Bharati Hospital)
Dr. Nitin Gadkari(Asso. Prof, Department of Medicine, Bharati Hospital)
Dr. Maheshkumar Lakhe (Asso. Prof, Department of Infectious Diseases, Bharati Hospital)
Dr. Deepak Bhosle (Prof, Department of Medicine, Bharati Hospital)

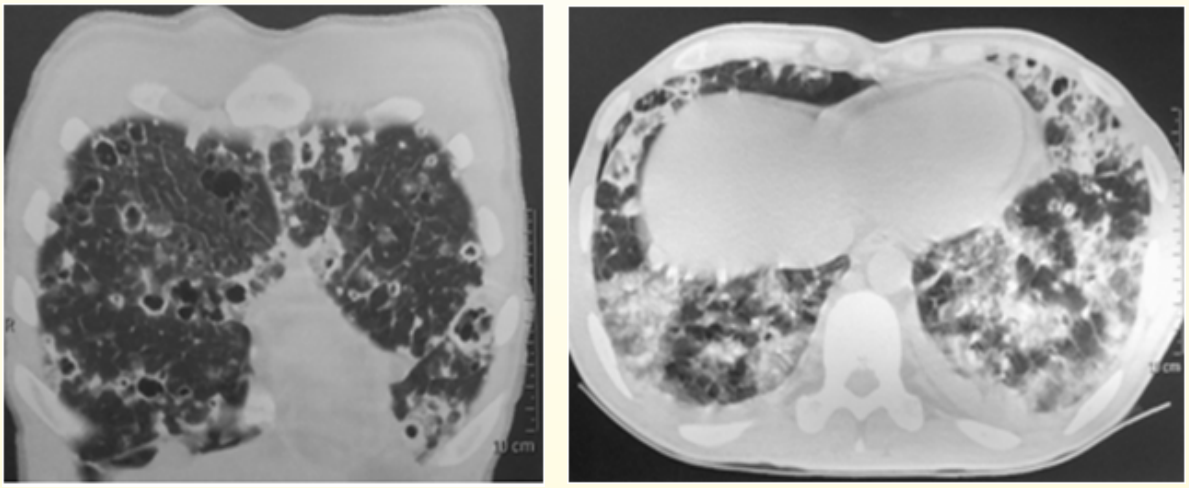
26/M who had no comorbidities was diagnosed with Non-compressive myelopathy& received multiple immunosuppressive agents over 4 years. In February 2018 he received 2 doses of Rituximab& was on Azathioprine 100mg/day thereafter.

In March, he developed a wound over sacral area (due to a toilet seat), which worsened, for which he underwent local debridement of the wound.



Gluteal lesions- de-roofed blisters

In April, he was readmitted with complaints of fever, dry cough, dysphagia and dyspnea. His sacral wound was unhealthy and he had developed additionalgluteal lesionswhich were discharging pus. HRCT thorax showed bilateral peripheral cavitary nodules.



CT Thorax at admission- bilateral, peripheral>perihilar, thin walled cavities

2 sets of Blood cultures grew MSSA. TTE was normal. Tissue cultures from gluteal lesions grew MRSA, Carbapenem- resistant –E. coli, Klebsiella pneumoniae and Acinetobacterbaumannii. He underwent bronchoscopy with BAL, which grew MRSA.

Discussion-

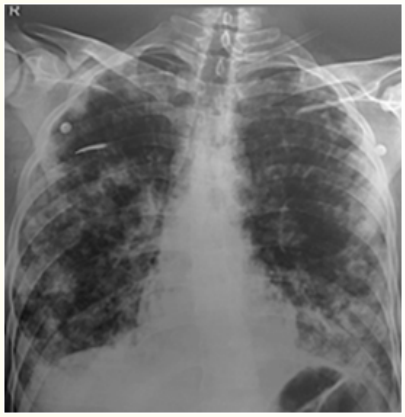
1) Etiology of lung abscesses, which determines treatment duration:
Since the lung lesions were bilateral, peripheral thin-walled cavities, they were likely hematogenous abscesses. Therefore, the organism producing bacteremia (MSSA) was the likely cause of the lung lesions, rather than MRSA which was found in BAL and could be a colonizer.

2) Etiology of Skin and soft tissue infection: The lesions appeared like de-roofed blisters leading to punched-out ulcerations, which are typical of Staphylococcus aureus. Therefore, MRSA could be the true etiologic agent for these lesions, rather than GNB (E.coli, Klebsiella, Acinetobacter) which could be colonizers of the ulcerated lesions.

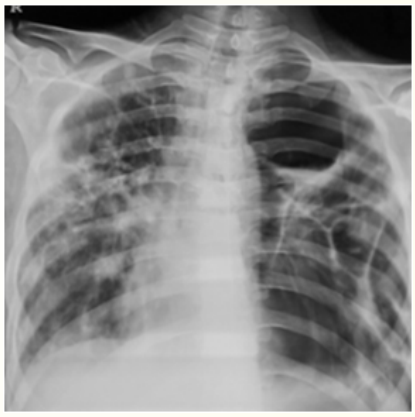
Treatment advised: Flucloxacillin 8g/24h for at least 6 weeks and Vancomycin 1gm q12h for 2 weeks (with Therapeutic Drug Monitoring), repeated blood cultures to check for clearance of bacteremia.

In conclusion:

a) MSSA was the cause of bacteremia and hematogenous lung abscesses, which needed treatment with Cloxacillin for 4-6 weeks.
b) MRSA was the cause of gluteal lesions which needed treatment with Vancomycin for 2 weeks.
c) GNB were considered colonizers and were not treated at all.



CXR At admission:
Peripheral nodular and cavitating opacities



Post-treatment:
Residual pneumatocele and fibrosis

PICTORIAL CME
IMAGE B:What does the following rash indicate?

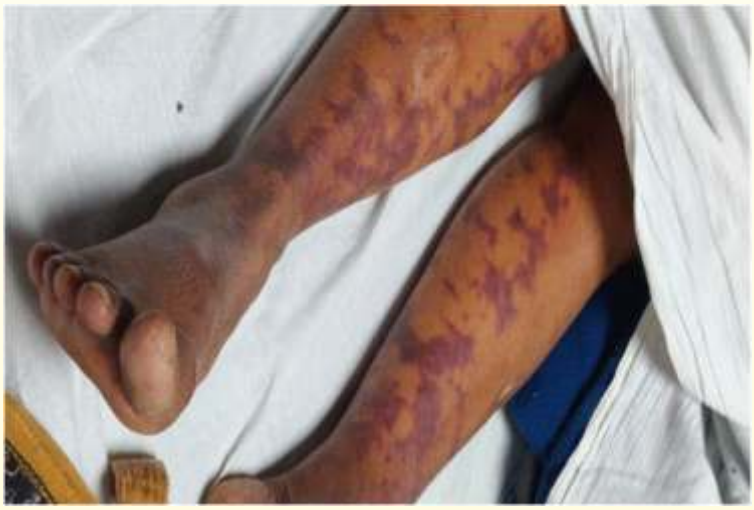


Image Courtesy: Dr. Prajakta Mane and Dr. Shivakumar Iyer (Department of Critical care, Bharati Hospital)

(Answers to be found elsewhere in this issue of Bharati Hospital ID newsletter)

MICRO TALK(From the Microbiology Department)

Challenges in Colistin susceptibility testing:

Dr. Kalpana Suryawanshi (Asst. Prof and Fellow In Clinical Microbiology, Dept. Of Microbiology)

Colistin (Polymyxin E) is a cationic cyclic Lipopeptide. They were isolated from Paenibacilluspolymyxa& have been used since 1050s, for Multi Drug resistant Gram Negative infections except few having intrinsic resistance to it like Proteus, Providencia, Serratia, Neisseria, Brucella, Chromobacterium, Burkholderia spp. But chromosomal & Plasmid mediated resistance due to mcr-1, mcr-1.2, mcr-2, mcr-3 resistance to this last resort antibiotic is already been reported so Judicious use with appropriate antibiotic susceptibility testing is warranted.

Clinical & laboratory Standards Institute (CLSI), & European Committee on Antimicrobial Susceptibility testing (EUCAST) has recommended broth Microdilution (BMD) without surfactant, as a reference method for determination of Colistin minimum Inhibitory concentrations.Commonly used other methods like E test, agar dilution have become obsolete, due to suboptimal reproducibility andaccuracy. Following are highlights of issues in antibiotic susceptibility testing of colistin in laboratories.

1) Colistin is multicomponent (A& B) molecule, variation in brand to Brand (manufacturing), & (Batch to Batch) preparation creates error in MIC reading.
2) Being a large molecule it diffuses poorly in agar medium giving small zone of inhibition &erroneous results in disc diffusion method which also fails to detect colistin heteroresistance. Disc diffusion test is removed from CLSI guidelines since 2017.
3) Variation of cationic concentrations(Calcium & Magnesium concentration) in Mueller-Hinton medium in different commercial brands leads to false susceptibility (very major error up to >32%), a false resistant (major error) in Colistin Gradient Epsilometer test, (E test). A few studies have also demonstrated a high rate of very major errors (false susceptibility) of up to 32%.This may be explained by their inability to accuratelydetect MIC by one or two folds dilutions for concentrations >2µg /ml.
4) Cationic composition increases the propensity of colistin to adhere to laboratory ware made up of polystyrene, polypropylene and glass. This effect couldn't be alleviated by polysorbate-80 a surfactant used for prevention of this binding to microtiter plates.
5) Agar dilution also gives unreliable results because of different cation concentrations present in Mueller Hinton agar, & studies have reported essential agreement of about 94% between BMD & agar dilution methods. However, these are not recommended by any of the guidelines.
6) Automated systems such as BD Phoenix, VITEK 2Compact, use limited number of colistin dilutions and may produce false susceptible results due to the skipped well phenomenon.

Essential agreement (EA-defined as MIC results within a twofold dilution from the Reference BMD results), for colistin testing by VITEK 2 Compact,was 91% & Categorical agreement(CA-defined as Agreement in the interpretation of MIC between the reference BMD method &Vitek) was <90%. Very major rate in colistin susceptibility was up to 36%.

ARTICLE CORNER

Interesting infectious disease articles recently published

(1) Implications for Diagnosis and Treatment of Infective Endocarditis: Eight year Experience of an Infectious Disease Team in a Private Tertiary Care Centre (JAPI April 2018 Vol 66)

Rajeev Soman, Neha Gupta, PiyushChaudhari, Ayesha Sunavala, Anjali Shetty, Camilla Rodrigues

Background: The profile of Infective endocarditis (IE) has been evolving continuously. Like other infectious Diseases (ID) syndromes, IE has not escaped from antibiotic resistance issues. The aim of this study was to determine the implications for diagnosis and treatment by studying the clinical profile and outcome of patients admitted with IE in a tertiary care centre in Mumbai during the period from 2007-2015.

Methods: 53 patients having definite or possible IE as per Modified Duke's Criteria (MDC), that were referred to the ID division, were included in this study.

Results: 44 (83%) patients had definite IE and 9 (17%) patients had possible IE. 77.4% of the patients were above 40 years of age. 3 patients presented as euthermic IE. Vegetations were not seen on transthoracic echocardiography (TTE) in 3 patients and were seen only on transesophageal echocardiography (TEE).

15 patients had prosthetic valve IE. 7 patients had rheumatic heart disease. 3 patients had bicuspid aortic valve and 4 had ventricular septal defect (VSD). The rest had no apparent underlying heart disease (45.3%).

41 patients (77.3%) had culture-positive IE and 12 patients (22.6%) had culture-negative IE. Streptococcus spp. was found in 14 (26.4%) patients, Enterococcus spp. in 9 patients (17%). Other organisms isolated were methicillin-sensitive S. aureus (3), Methicillin Resistant S. aureus (1), Eikenellacorrodens (1), B. cepacia (2), Salmonella Typhi (1), P. aeruginosa (1), M. abscessus (2) and other rapidly growing mycobacteria (RGM) (5), Candida parapsilosis (1), Candida pelliculosa (1) and Aspergillus fumigatus (1). Notably there was only one case of MRSA.

Among the Streptococcus spp., Penicillin MIC testing was done in 11 cases of the 14 cases of Strep spp. 3 of them showed intermediate resistance and 2 were resistant. Among enterococcal IE, 3 had high level aminoglycoside resistance (HLAR) and 2 had β-lactamase producing enterococci with HLAR and 1 had Vancomycin resistance. These were successfully treated with combinations of Ampicillin with Ceftriaxone, Ampicillin-Sulbactam with Imipenem and Daptomycin respectively. The only case of MRSA prosthetic valve endocarditis was successfully treated with Vancomycin and Rifampicin in addition to surgery. Surgery for IE was performed in 26 out of 53 (49%) patients. Early valve surgery (within 15 days of hospital admission) was performed in 6 of these 26 patients.

Conclusion
43.5% patients had no predisposing factors for IE and blood cultures were negative in 22.6% cases. In our study, PVE was the most common predisposing condition for IE. VGS followed by enterococci were found to be the commonest cause for IE in our setting. Both organisms show variable drug resist patterns. Vancomycin may not be required as empiric treatment in our settingas MRSA is rare. Surgery, whenever indicated, helps in improving outcome in these patients thus reiterating the need for a team approach for optimal management of this complex, challenging condition.

Comment:
This study gives us an overview of infective endocarditis epidemiology in India and makes us aware of the challenges involved In treatment. Nevertheless, if managed appropriately, the prognosis of the condition can be expected to be good.

(2) EARNEST Trial Substudy: Evolution of Protease Inhibitor Resistance in Human Immunodeficiency Virus Type 1 Infected Patients Failing Protease Inhibitor Monotherapy as Second-line Therapy in Low-income Countries (Clin infect Dis 2018 Jul 28)

Jennifer Thompson et al

Background
Limited viral load (VL) testing in human immunodeficiency virus (HIV) treatment programs in low-income countries often delays detection of treatment failure. The impact of remaining on failing protease inhibitor (PI)–containing regimens is unclear.

Methods
We retrospectively tested VL in 2164 stored plasma samples from 386 patients randomized to receive lopinavirmonotherapy (after initial raltegravir induction) in the Europe–Africa Research Network for Evaluation of Second-line Therapy (EARNEST) trial. Protease genotypic resistance testing was performed when VL >1000 copies/mL. We assessed evolution of PI resistance mutations from virological failure (confirmed VL >1000 copies/mL) until PI monotherapy discontinuation and examined associations using mixed-effects models.

Results
Median post-failure follow-up (in 118 patients) was 68 (interquartile range, 48–88) weeks. At failure, 20% had intermediate/high-level resistance to lopinavir. At 40–48 weeks post-failure, 68% and 51% had intermediate/high-level resistance to lopinavir and atazanavir; 17% had intermediate-level resistance (none high) to darunavir. Common PI mutations were M46I, I54V, and V82A. On

average, 1.7 (95% confidence interval 1.5–2.0) PI mutations developed per year; increasing after the first mutation; decreasing with subsequent mutations (P < .0001). VL changes were modest, mainly driven by nonadherence (P = .006) and PI mutation development (P = .0002); I47A was associated with a larger increase in VL than other mutations (P = .05).

Conclusions

Most patients develop intermediate/high-level lopinavir resistance within 1 year of ongoing viral replication on monotherapy but retain susceptibility to darunavir. Viral load increased slowly after failure, driven by non-adherence and PI mutation development.

Comment:

This EARNEST trial substudy underscores importance of periodic viral load testing of patients on second-line antiretroviral therapy. If failure is identified early, accumulation of further Protease Inhibitor (PI) mutations and compromised third-line ART can be avoided.

(3) Emerging concepts in HIV- associated Cryptococcal Meningitis (CurrOpin Infect Dis 2019; 32 (1): 16-23)

Lawrence David et al

HIV-associated cryptococcal meningitis remains a significant contributor to AIDS-related mortality despite widened access to antiretroviral therapy. Even in clinical trial settings 10-week mortality is roughly 40%. A number of important clinical trials have either recently concluded or are actively recruiting. Recent findings: Global burden of disease estimates suggest cryptococcal meningitis causes 181 100 deaths annually. Screening blood for cryptococcal antigen in HIV-infected individuals with CD4 cell counts less than 100 cells/[mu]l and preemptive antifungal treatment for those with detectable cryptococcal antigen reduces the incidence of cryptococcal meningitis and is likely to reduce mortality. Cryptococcal meningitis treatment with conventional 14-day courses of amphotericin are associated with high toxicity and mortality and can be reduced to 7 days if given alongside flucytosine. Flucytosine is a significantly superior adjunct to amphotericin treatment compared with fluconazole. In settings without amphotericin B dual oral antifungal combinations of flucytosine and fluconazole offer an effective alternative treatment. A single, high-dose of liposomal amphotericin is effective at reducing fungal burden and is being tested in a phase III trial.
Summary: Recently completed and ongoing clinical trials are increasing our understanding of how to optimize induction therapy for cryptococcal meningitis. Advocacy efforts are needed to broaden access to amphotericin formulations and flucytosine.

Comment:

This article spells out important aspects In managing Cryptococcal meningitis In HIV.

Answers to the PICTORIAL CME...

Image A: "Reverse halo sign"

The reverse halo sign also known as the Atoll sign (because of its resemblance of a coral atoll) is a pulmonary nodule with a central ground-glass opacity surrounded by a denser consolidation in crescentic or ring form. In the photograph depicted above, this sign was demonstrated in a diabetic elderly lady with pulmonary mucormycosis. In contrast, the Halo sign found in invasive pulmonary aspergillosis has a central dense consolidation surrounded by a ground- glass opacification. Both the signs are indicative of angioinvasive nature of the underlying fungal infection.

Pathologically the central area of reverse halo sigh represents alvelolitis and cellular debris within alveolar spaces and the rim of consolidation represents granulomatous tissue.

While the reverse halo sign was initially thought to be classically associated with cryptogenic organizing pneumonia (COP), later on it was found that it neither sensitive nor specific for COP. Other conditions which may present with the reverse halo sign on CT (computed tomography) chest include pulmonary mucormycosis, invasive pulmonary aspergillosis, granulomatosis with polyangiitis, sarcoidosis, Pneumocystis jiroveci pneumonia (PCP), lipoid pneumonia, tuberculosis and pulmonary neoplasms.

When seen in a susceptible host, the reverse halo sign may be useful in narrowing down the differential diagnosis of a pulmonary nodule.

References: Brit J Radiol 2012 sept; 85 (1017): 1226-35, Clin infect Dis 2011 May; 52 (9):1144-1155

Image B: skin lesions:"Rash of meningococcemia"

Petechial lesions coalesce to form larger purpuric or ecchymotic lesions. This rash was seen in a Human immunodeficiency virus (HIV) infected individual on stable anti-retroviral therapy (ART) with a CD4 cell count of >600 cells per microliter. She presented with fever and hypotension of 2 days duration and this rash was found on admission in Intensive care unit (ICU). She was later diagnosed to have disseminated meningococcal infection based upon a positive peripheral blood PCR (polymerase chain reaction) for menigococci. The lesions of meningococcemia begin as macules but rapidly progress to increasing numbers of petechial or purpuric lesions which develop on distal extremities and trunk, usually sparing palms and soles. Lesions may later take a complicated form known as purpurafulminans

Differential diagnosis of purpuric skin lesions includes septic vasculitis caused by meningococcemia, gonococcemia, streptococcal shock syndrome, Rickettsioses, infective endocarditis; autoimmune vasculitis syndromes; thrombotic disorders such as disseminated intravascular coagulation (DIC), antiphsopholipid syndrome, thrombotic thrombocytopenia purpura (TTP) and warfarin induced skin necrosis.

References :ClinDermatol 2006;24 (5):414, CMAJ Jan 2010:182 (1)

Community Acquired Urinary Tract Infection by Pseudomonas oryzihabitans.

Dr. Sunita Bhatawadekar. Dr. Kunal Lahiri

Microbiology Department
BVDUMC & BHRC, Pune.

Introduction-

Pseudomonas oryzihabitans, previously known as Flavimonas has been placed in CDC group Ve-2. P. oryzihabitans has been recovered from various clinical samples, including wound swab, sputum, ear swab, conjunctival scrapings, Urine, peritoneal fluid and blood. P. (Flavimonas) oryzihabitans bacteremia was also reported in a neonate. [1, 2]

P. oryzihabitans appears to be an emerging pathogen. P. oryzihabitans is an uncommon pathogen associated with indwelling intravenous catheters infection. [3, 4] We report a case of urinary tract infection (UTI) caused by P. oryzihabitans in a patient with anterior stricture of urethra. Chrysomonasluteola belongs to CDC group Ve-1. Recently two cases of infection from Indian patients by chrysomonas have been reported, from Mumbai and Hyderabad. [5, 6] There is no documented report of P. oryzihabitans infection in any Indian patient. In May 2011, six cases of P. Oryzihabitans bacteremia in NICU were reported. [2]

Case report-

A 45 yr. old male patient was admitted in surgery ward with complaints of difficulty in passing urine since last two months. There was no history of burning micturition and hematuria. No history of chronic illness suggestive of immunocompromised status. Test for HIV and HBsAg was negative. Hemoglobin was 13.8g/dl and ESR was19 mm/hr. General and systemic examination was normal. Ultra sonography of abdomen and pelvis was normal. Retrourethrogram showed narrowing in anterior urethra. Case was provisionally diagnosed as stricture of urethra with UTI. Urine sample was received for culture and sensitivity, and processed by routine semi quantitative method. On blood agar rough wrinkled yellow pigmented colonies were grown and on Mac Conkeys agar non lactose fermenting colonies were grown. Gram negative, motile, oxidase negative, nonfermenter bacilli were isolated. Isolate was further identified as Pseudomonas oryzihabitans (% id98.3) by the API ID 32 GN automated identification system (bioMérieux, Marcy l' Étoile, France). Identification was based on following tests- negative nitrate reduction, esculin hydrolysis, lysine decarboxylase, arginine dehydrolase and orthonitrophenyl-beta-d galactopyranoside activity (ONPG), positive oxidation fermentation glucose, maltose, mannitol, xylose activity. Antibiotic sensitivity was done by disc diffusion method using Clinical and Laboratory Standards Institute (CLSI) guidelines. Isolate was sensitive to piperacillin, cephalosporins, imipenem, meropenem, cotrimoxazole, aminoglycosides, fluoroquinolones and resistant to nitrofurantoin. Patient was treated with oral norfloxacin 400 mg twice daily for ten days, and advised to come for follow up after fifteen days. Initial retrourethogram showed narrowing in anterior urethra, but as patient responded to antibiotic treatment and dysuria was relieved, repeat retrourethogram was not done.

Discussion-

In hospitals, P. oryzihabitans has been isolated from sink drains and respiratory therapy equipment. In nature, this organism has been isolated from rice paddies. P. oryzihabitans bacteremia was reported in 12 patients at National Taiwan University hospital.[4] Four cases of community acquired pneumonia infection by P. Oryzihabitans were reported, three in HIV positive patients and one in a patient with chronic myeloid leukemia.[7] Most of the reports of P. oryzihabitans infection were of nosocomial origin in

individuals with one of the predisposing factors like low birth weight neonate, premature neonate, biliary tract infection, peritonitis, subdural empyema, pneumonia, and were associated with presence of indwelling catheters. [1,2,4,8] There are very few reports of community acquired infection by P. oryzihabitans, like infection of Hickman catheter traced to a synthetic bath sponge, pneumonia, and soft tissue infection [3,7,9] Some case reports also have been documented in otherwise previously healthy individuals. [10]P. oryzihabitans isolated from blood sample of catheter associated infection in AIDS patient was sensitive to broad spectrum cephalosporins, aztreonam, imipenem, aminoglycosides, ciprofloxacin and trimethoprim- sulfamethoxazole, and resistant to ampicillin, amoxycillin-clavulinic acid and cephazolin.[3] In contrast to the previously reported cases, where Pseudomonas (Flavimonas) showed resistance to cephazolin, cefuroxime and trimethoprim, our isolate was found to be sensitive to these antibiotics.[8] This suggests that the strain could well have been a community isolate.

P. oryzihabitans although a saprophyte, could as well emerge as a potential pathogen. The clinical Microbiologists therefore should not ignore them as laboratory contaminants, because reports of infections are on rise both in immune-compromised and in immune-competent individuals. Although P. oryzihabitans has been isolated occasionally from the environment, the source of human infection has been well documented only in few cases, in two reports source of infection was traced to a bath sponge. [2, 3] In Pub –Med search with P. Oryzihabitans infection in Indian patients, no item was found. This may be the first case report of P. Oryzihabitans UTI infection in Indian patient. Thus proper identification of the nonfermentor is the need of the day. Clinicians and laboratory personnel also have to be made aware of the pathogenic role of P. oryzihabitans which may become increasingly prevalent in near future.

References:

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2) Prifti H, Oikonomidou D, Pappa O, Tryfinopoulou K ,Vatzeli K, Karaiskos K, et al.Outbreak of Pseudomonas (Flavimonas) oryzihabitans bacteraemia in a neonatal intensive care unit. May 08, 2011, 13:30 - 14:30.

Available from
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Legend for the image :
Figure1: Colonies of P. oryzihabitans on Mueller-Hinton agar. (100X)

• EQAS

Dr Bharati Dalal, Bharati Hospital, Pune

- This department participates in the EQAS programme conducted for Bacteriology & Serology at CMC Vellore since 2011 and Mycology at PGI Chandigarh since 2016

• Bacteriology and Serology

Sr. No.	Date	Marks scored	% marks	Remarks
1	June 2017	Bacteriology -51/55 Viral Serology- 6/6	93 % 100 %	Satisfactory Satisfactory
2	March 2018	Bacteriology -68.5/69 Viral Serology- 8/8	99 % 100 %	Satisfactory Satisfactory
3	June 2018	Bacteriology -67/69 Viral Serology- 8/8	97.10 % 100 %	Satisfactory Satisfactory

• Mycology

Sr. No.	Date	Marks scored	% marks	Remarks
1	June 2017	Bacteriology -51/55 Viral Serology- 6/6	93 % 100 %	Satisfactory Satisfactory
2	March 2018	Bacteriology -68.5/69 Viral Serology- 8/8	99 % 100 %	Satisfactory Satisfactory
3	June 2018	Bacteriology -67/69 Viral Serology- 8/8	97.10 % 100 %	Satisfactory Satisfactory

Antibiogram 2017

Bharati Hospital, Pune

Dr. Abhijeet Mane, Dr. Anuradha Tolpadi, Dr. KK Lahiri, Bharati Hospital, Pune

Table No. 1 Distribution of isolates from various samples			
Samples	Total	Growth	No Growth
Blood	2693	471 (17.48%)	2222
Urine	3467	1322 (38.13%)	2145
Pus	1452	1095 (75.41%)	357
Respiratory	1101	384 (34.87%)	717
Miscellaneous	690		

Table No. 2 MRSA & ESBL - PREVALENCE			
MRSA prevalence	59.15%	ESBL prevalence	57.10%

Table No. 3 Important isolates - ICU							
PUS (n=105)		URINE (n=173)		BLOOD (n=178)		RESPIRATORY (n=184)	
E.coli	30 (28.5%)	E.coli	73 (42.2%)	CONS	56 (31.4%)	Klebsiella	60 (32.6%)
Klebsiella	19 (18.0%)	Klebsiella	34 (19.7%)	E.coli	23 (13.0%)	Acinetobacter	45 (24.4%)
Pseudomonas	16 (15.2%)	Pseudomonas	12 (7.0%)	S.aureus	22 (12.3%)	Pseudomonas	36 (19.5%)

Table No. 4 Important isolates - NICU							
PUS (n=24)		URINE (n=40)		BLOOD (n=103)		RESPIRATORY	
Klebsiella	7 (29.1%)	E.coli	73 (42.2%)	Klebsiella	40 (39.0%)	unremarkable	
S.aureus	6 (25%)	Klebsiella	34 (19.7%)	CONS	18 (17.4%)		
E.coli	4 (16.7%)	Pseudomonas	12 (7.0%)	Acinetobacter	15 (14.5%)		

Table No. 5 Important isolates -PICU							
PUS (n=23)		URINE (n=6)		BLOOD (n=43)		RESPIRATORY (n=16)	
Klebsiella	5 (22%)	E.coli	3 (50%)	CONS	11 (25.6%)	Acinetobacter	10 (62.5%)
S.aureus	4 (17.4%)			S.aureus	3 (7.0%)		
CONS	3 (13.0%)			Acinetobacter	4 (9.3%)		

Table No. 6 Important isolates - PAEDIATRIC WARD							
PUS (n=75)		URINE (n=113)		BLOOD (n=67)		RESPIRATORY	
S.aureus	39 (52.0%)	E.coli	63 (56%)	CONS	27 (40.2%)	unremarkable	
E.coli	16 (21.3%)	Klebsiella	23 (20.3%)	Acinetobacter	8 (12.0%)		
		Enterococcus	10 (8.9%)	Pseudomonas	5 (7.4%)		

Table No. 7 Important isolates - ALL WARDS (ADULT)							
PUS (n=630)		URINE (n=643)		BLOOD (n=73)		RESPIRATORY (n=151)	
S.aureus	173 (27.4%)	E.coli	285 (44.3%)	CONS	18 (25%)	Klebsiella	56 (37.0%)
E.coli	104 (17%)	Klebsiella	111 (17.2%)	S.aureus	15 (20.5%)	Pseudomonas	32 (21.1%)
Pseudomonas	97 (15.3%)	Enterococcus	66 (10.2%)	Pseudomonas	10 (14.0%)	Acinetobacter	12 (7.9%)

Table No. 8 Important isolates - ALL OPDs (ADULT)							
PUS (n=238)		URINE (n=347)		BLOOD		RESPIRATORY	
S.aureus	86 (36.1%)	E.coli	169 (49.0%)	unremarkable		unremarkable	
E.coli	35 (14.7%)	Klebsiella	52 (15.0%)				
Pseudomonas	32 (13.4%)	Enterococcus	32 (9.2%)				

Table No. 9 SUSCEPTIBILITY(in percentage) of important Gram negative isolates 2017 - ICU										
Antibiotic	Pus (n=105)			Urine (n=173)			Blood (n=178)	Respiratory (n=184)		
	Esch.coli	Klebsiella	Pseudomonas	Esch.coli	Klebsiella	Pseudomonas	Esch.coli	Klebsiella	Acinetobacter	Pseudomonas
	n=30	n=19	n=16	n=73	n=34	n=12	n=23	n=60	n=45	n=36
Piperacillin	5	13	66	2	0	33	0	9	4	84
AmpiSulb	0	0	-	15	0	-	0	0	11	-
AmoxClav	30	36	-	24	4	-	23	19	11	-
Piptaz	41	41	55	41	22	33	50	55	14	74
Cefuroxime	14	6	-	7	0	-	9	10	0	-
Cefotax	10	12	-	7	0	-	0	12	-	-
Ceftaz	10	12	66	7	0	50	0	12	8	80
Ceftriaxone	14	12	-	7	0	-	9	12	-	
Cefepime	18	47	62	11	4	25	26	42	17	69
Imi/Mero	82	76	54	75	37	22	83	65	24	70
Cotrimoxazole	27	47	-	25	22	-	35	43	22	
Gentamicin	77	47	69	48	15	44	61	62	16	72
Amikacin	86	76	77	71	31	44	86	75	21	84
Ciproflox	19	41	62	21	16	20	27	51	14	71
Colistin	100	100	82	100	96	77	100	94	97	92
PolymixinB	100	100	82	100	96	77	100	94	97	92
CefoSulba	53	62	50	50	19	22	52	56	33	64
Tigecycline	100	81	-	-	-	-	100	100	53	-
Norflox	-	-	-	18	13	-	-	-	-	-
Nitrofurantoin	-	-	-	59	11	-	-	-	-	-
Fosfomycin	-	-	-	100	40	-	-	-	-	-

Table No. 10 SUSCEPTIBILITY (in percentage) of important Gram negative isolates 2017 – NICU			
Antibiotic	Urine (n=40)	Blood (n=103)	
	Klebsiella	Klebsiella	Acinetobacter
	n=13	n=40	n=15
Piperacillin	9	3	0
AmpiSulb	0	0	0
AmoxClav	11	26	0
Piptaz	18	26	11
Cefuroxime	9	0	0
Cefotax	9	3	0
Ceftaz	9	3	0
Ceftriaxone	9	3	-
Cefepime	9	22	10
Imi/Mero	18	42	20
Cotrimoxazole	0	42	22
Gentamicin	30	48	10
Amikacin	18	45	0
Ciproflox	40	39	10
Colistin	100	100	90
PolymixinB	100	100	90
CefoSulba	0	32	29
Tigecycline	-	-	-
Norflox	71	-	-
Nitrofurantoin	9	-	-
Fosfomycin	80	-	-

Table No. 11 SUSCEPTIBILITY (in percentage) of important Gram negative isolates 2017 – PAEDIATRIC WARD			
Antibiotic	Pus (n=75)	Urine (n=113)	
	Esch.coli	Esch.coli	Klebsiella
	n=16	n=63	n=23
Piperacillin	9	4	6
AmpiSulb	33	5	0
AmoxClav	30	36	40
Piptaz	50	64	65
Cefuroxime	18	12	13
Cefotax	20	10	14
Ceftaz	20	10	14
Ceftriaxone	20	10	14
Cefepime	27	28	31
Imi/Mero	75	90	88
Cotrimoxazole	45	37	19
Gentamicin	54	60	76
Amikacin	92	88	82
Ciproflox	25	41	25
Colistin	100	100	100
PolymixinB	100	100	100
CefoSulba	57	77	44
Tigecycline	100	-	-
Norflox	-	33	60
Nitrofurantoin	-	86	40
Fosfomycin	-	94	60

Table No. 12 SUSCEPTIBILITY (in percentage) of important Gram negative isolates 2017 – ALL WARDS							
PUS n=238	Pus (n=630)		Urine (n=643)		Respiratory (n=151)		
	Esch.coli	Pseudomonas	Esch.coli	Klebsiella	Klebsiella	Pseudomonas	Acinetobacter
	n=104	n=97	n=285	n=111	n=56	n=32	n=12
Piperacillin	5	53	6	6	16	80	14
AmpiSulb	30	-	29	0	0	-	16
AmoxClav	28	-	45	36	42	-	9
Piptaz	67	75	67	50	71	85	54
Cefuroxime	5	-	14	19	18	-	-
Cefotax	8	-	14	10	21	-	
Ceftaz	8	60	14	10	21	88	14
Ceftriaxone	8	-	14	10	21	-	-
Cefepime	29	67	29	37	54	87	55
Imi/Mero	81	86	85	70	81	87	55
Cotrimoxazole	38	-	44	43	66	-	50
Gentamicin	54	78	67	53	79	91	54
Amikacin	87	74	82	57	92	87	37
Ciproflox	29	67	30	34	64	86	63
Colistin	97	99	99	93	100	89	100
PolymixinB	97	99	99	93	100	89	100
CefoSulba	75	74	70	60	64	85	100
Tigecycline	90	-	-	-	90	-	100
Norflox	-	-	31	38	-	-	-
Nitrofurantoin	-	-	78	34	-	-	-
Fosfomycin	-	-	95	67	-	-	-

Table No. 13 SUSCEPTIBILITY (in percentage) of important Gram negative isolates 2017 - ALL OPDs				
Antibiotic	Pus (n=238)		Urine (n=347)	
	Esch.coli	Pseudomonas	Esch.coli	Klebsiella
	n=35	n=32	n=169	n=52
Piperacillin	7	63	12	18
AmpiSulb	0	-	44	0
AmoxClav	40	-	53	50
Piptaz	77	88	76	50
Cefuroxime	11	-	27	27
Cefotax	12	-	21	23
Ceftaz	12	60	21	23
Ceftriaxone	12	-	21	23
Cefepime	42	50	46	37
Imi/Mero	89	80	88	65
Cotrimoxazole	35	-	46	46
Gentamicin	75	71	68	59
Amikacin	92	80	86	70
Ciproflox	29	60	44	50
Colistin	100	100	100	97
PolymixinB	100	100	100	97
CefoSulba	75	70	70	58
Tigecycline	100	-	-	-
Norflox	-	-	40	94
Nitrofurantoin	-	-	81	30
Fosfomycin	-	-	90	75

Table No. 14 SUSCEPTIBILITY (in percentage) of important Gram positive isolates - 2017

	ICU	NICU	PAEDS	ALL WARDS		ALL OPDs	
	BLOOD (n=178)	URINE (n=40)	PUS (n=75)	PUS (n=630)	URINE (n= 643)	PUS (n=238)	URINE (n=347)
	S.aureus n= 22	Enterococcus n=10	S.aureus n=39	S.aureus n=173	Enterococcus n=66	S.aureus n=86	Enterococcus n=32
Penicillin	0	37	5	1	31	6	18
Oxacillin	47	0	22	33	0	44	0
Erythromycin	31	0	42	36	0	41	-
Vancomycin	93	85	97	100	89	92	95
Teicoplanin	100	100	100	99	95	91	91
Chloramphenicol	66	-	100	92	-	91	-
Clindamycin	60	-	62	62	-	75	-
Tetracycline	81	0	97	91	24	98	20
Rifampicin	93	0	97	93	57	98	0
Linezolid	100	100	100	99	100	100	95
Ciprofloxacin	17	0	2	19	42	26	14
Gentamycin	76	14	58	72	29	74	29
Cotrimoxazole	28	0	60	55	0	61	-
Norfloxacin	-	0	-	-	25	-	35
Nitrofurantoin	-	50	-	-	59	-	50
Tigecycline	-	100	100	100	100	100	100

UPGRADATION OF MICROBIOLOGY LABORATORY SERVICES

The Microbiology section of Bharati Hospital laboratory received certificate of accreditation by NABL one year back. Since then the department has been working tirelessly to maintain the quality standards of NABL and also to introduce new tests in our directory of services for the betterment of patients. The last one year has seen the following tests being introduced in our laboratory :

1) Dengue NS1, IgG and IgM by ELISA.

2) HAV and HEV IgG and IgM - Done by CMIA and ELISA respectively

3) Mycobacterium tuberculosis culture using MGIT

4) Line probe assay

5) ANA detection by IFA and ELISA

6) Calcofluor white stain

7) Cryptococcal antigen detection

8) Colistin microbroth dilution test

NABH Accreditation promises

- Top Standard quality care of patients by qualified doctors, nurses & paramedical & supported by auxiliary staff.
- Continuous up gradation of patient care facilities with "State of the Art" Equipments and Diagnostics
- Highest value for patient Safety & Rights.

All Super Speciality services under one roof

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- ✓ Paediatric Haemato – Oncology
- ✓ Neurology
- ✓ Neurosurgery
- ✓ Paediatric Neurology
- ✓ Paediatric Surgery
- ✓ Nephrology
- ✓ Cardiology
- ✓ Infertility
- ✓ Joint Replacement
- ✓ Endoscopy
- ✓ Laparoscopy
- ✓ Oragan Transplant (Upcoming)
- ✓ Open Heart Surgery (Upcoming)

NABH Accredited Blood Bank

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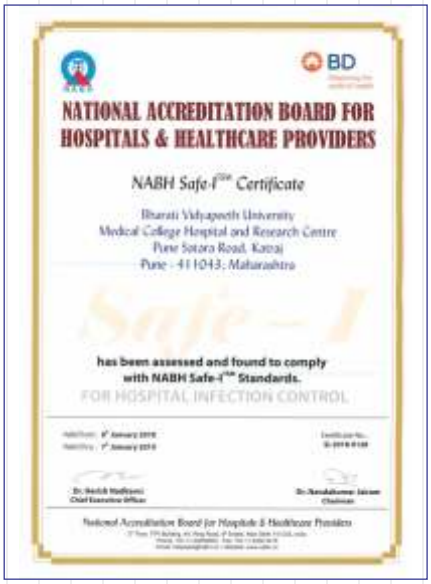
NABH Accredited Institutional Ethics Committee

NABL Accredited Laboratory

NABH Accredited Hospital



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