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Medtalks with Dr KK Aggarwal

CMAAO Coronavirus Facts and Myth Buster

Why Certain COVID Patients Die?

- Advancing age and underlying medical problems explain only part of the phenomenon.
- Some people, especially men, succumb because their immune systems are hit by friendly fire.
- A study in *Science* revealed that 10% of around 1,000 COVID patients who developed life-threatening pneumonia had antibodies that disable interferons. These autoantibodies were not found in 663 people with mild or asymptomatic COVID infections. Only four of 1,227 healthy individuals were found to have the autoantibodies.
- In a second study by the same team, it was noted that an additional 3.5% of critically ill patients had mutations in genes that control the interferons involved in fighting viruses. The body has 500-600 of these genes, therefore, it seems possible that researchers will find more mutations, said lead author of the second study.
- Interferons are the body's first-line of defense against infection.
- They are a fire alarm and a sprinkler system all in one.
- Laboratory studies suggest that interferons are suppressed in some people with COVID-19, probably by the virus itself.
- Interferons are vital for protecting the body against new viruses, such as the coronavirus, which the body has never encountered.
- When infected with the novel coronavirus, the body should have alarms ringing. If the alarm doesn't get out, you could have viruses everywhere in large numbers.
- Patients didn't make autoantibodies in response to the virus. It appeared that they had them before the pandemic began. These autoantibodies never caused a problem until patients were infected with COVID-19. Somehow, the novel coronavirus, or the immune response it triggered, seems to have set them in motion.
- It is not known whether autoantibodies against interferon also increase the risk from other viruses, such as influenza.
- Furthermore, 94% of patients in the study with these autoantibodies were men. About 12.5% of men with life-threatening COVID pneumonia had autoantibodies against interferon, compared to 2.6% of women.

(*Source: Medscape*)

Why Viruses Spread More Easily in Winter?

- In winters, people tend to spend more time indoors, where ventilation is poor and we're in closer proximity to other people.
- The Air Outside is Less Humid: Viruses stay stable and linger for longer when the air is less humid.
- Nasal Membranes Are Drier: Feels dry and cracked. These cracks make you more vulnerable to infection. Staying hydrated and using a saline nasal spray can help.
- Other Illnesses Cause Coughing and Sneezing: It's possible to have co-infections of COVID-19 and say, the seasonal flu. So even if a person has an asymptomatic case of COVID-19, another infection may cause the coughing and sneezing that is known to increase the spread of the coronavirus.
- The virus lipid membrane may get thicker.

Mask Fatigue and Air Travel

- It's getting harder for some people to comply with the safety rules but a mask is life-saving.
- Don't wear gloves, it gives you a false sense of security—just use your hands and wash them when you get home.
- If you do need to fly, wear eye protection, a mask and minimize the time the mask is down. For instance, eat a protein bar, then put up your mask immediately after you take a bite while you chew. There are studies on how air circulates on

airplanes; it goes in a downward direction which reduces the transmission between rows as well.

- When you arrive at your destination, change your clothes and take a shower to minimize the risk. COVID does live on the skin for up to 6-9 hours and can live on surfaces. It's been shown to live on plastic for up to 72 hours and on cardboard and paper for 24 hours.
- Don't eat indoors with people outside of your household. That is a major scientifically-proven mechanism of transmission. If you are sitting across the table from someone, you can still transmit it.

Mucocutaneous Manifestations of MIS-C

DG Alerts: Mucocutaneous manifestations of multisystem inflammatory syndrome in children: An array of mucocutaneous findings was identified in hospitalized children with multisystem inflammatory syndrome in children (MIS-C) or suspected MIS-C during the COVID-19 pandemic, reported a case series published in *JAMA Dermatology*.

Of the patients assessed, 83% developed mucocutaneous changes, with the most common being conjunctival injection, palmoplantar erythema, lip hyperemia, periorbital erythema and edema, strawberry tongue and malar erythema. Other cutaneous morphologic findings included scarlatiniform eruptions, morbilliform eruptions, urticarial eruptions and reticulated eruptions. Among those with mucocutaneous changes, 19 patients experienced fever a mean of 2.7 days (range, 1-7 days) prior to the recognition of the first mucocutaneous finding. The duration of mucocutaneous findings ranged from hours to days (median duration, 5 days [range, 0-11 days]).

Overall, 19 patients had cardiac involvement as noted by elevated troponin and/or brain natriuretic peptide levels, while 10 had abnormal echocardiogram findings; 5 patients with cardiac involvement needed inotropic support. Ten patients were admitted to the intensive care unit (ICU). There appeared to be no statistically significant associations between the presence of mucocutaneous findings and cardiac dysfunction, need for inotropic support or ICU admission, which suggests that mucocutaneous changes were not tied to disease severity in MIS-C.

Conjunctivitis, lip hyperemia or cracking, and palmoplantar erythema exhibited an even distribution across all ages; however, urticarial eruptions were noted in those below 2 years of age, and periorbital and palmoplantar edema were evident in those younger than 6 years. (*Source: JAMA Dermatology*)

Baricitinib *plus* Remdesivir

Baricitinib *plus* remdesivir shows promise in treatment of COVID-19: The combination of baricitinib, an anti-inflammatory drug and remdesivir, an antiviral, was found to reduce the time to recovery among hospitalized COVID-19 patients, suggested clinical trial results published in the *New England Journal of Medicine*. The study was supported by the National Institute of Allergy and Infectious Diseases (NIAID), part of the National Institutes of Health.

The ACTT-2 trial started on May 8, 2020 and recruited 1,033 volunteers at sites in 8 countries. **Participants were randomized to receive either oral baricitinib tablets and intravenous (IV) remdesivir or oral placebo tablets and IV remdesivir.**

Combination of baricitinib and remdesivir reduced median time to recovery among hospitalized COVID-19 patients from 8 days to 7 days. Patients requiring high-flow oxygen or noninvasive ventilation during hospitalization were shown to obtain the largest benefit. The median time to recovery in these patients was reduced from 18 days to 10 days. Furthermore, participants' condition at Day 15 (measured by **an eight-category ordinal scale** which ranked the severity of their condition) was significantly improved when they received the combination therapy. Those who were given the two treatments also had slightly fewer serious adverse effects. (*Source: NIH*)

IMA-CMAAO Webinar on "COVID Experience in Mumbai"

28th November, 2020 (4-5 pm)

Participants: Dr KK Aggarwal, President-CMAAO; Dr RK Datta; Dr Jayakrishnan Alapet; Dr Brijendra Prakash; Dr S Sharma

Faculty: Dr Rahul Pandit, Director Critical Care Medicine & ICU, Fortis Hospital, Mumbai COVID Task Force Member, Maharashtra

Key points from the discussion

- Cases in Mumbai have reduced since November and are now 400-500/day. In September, there were around 2,000 cases every day.
- Children are protected due to a reasonable amount of immunity from the thymus gland, which is present in an active child up to 8-10 years of age.

IJCP SUTRA: "Cut down on food or drinks rich in caffeine including coffee, tea, cola, energy drinks and chocolate. Caffeine is a mood-altering drug, and it may make symptoms of anxiety disorders worse."

- An exploratory trial was conducted with thymosin alfa in 15 patients to examine if it could activate CD4 and CD8 cells and increase lymphocyte count. Critically ill patients (as per ICMR criteria) were selected for the trial. Thymosin was started on Day 1 (3.2 mg TDS SC). Patients started showing improvement in CD4, CD8 and lymphocyte cell count by Day 4 and there was statistically significant improvement by Day 7. Cytokine markers also reduced (C-reactive protein [CRP], D-dimer); ferritin was not so robust. Lactate dehydrogenase (LDH) did not change much. These findings need to be tested in a large trial.
- The average lymphocyte counts in these patients were in single digits when they were in ICU. The average total leukocyte count (TLC) was 4,100; the lymphocyte count was 9.2% (average). So, if we act on moderate disease patients, perhaps we can stop progression to severe disease.
- Thymosin alfa has not been tried in human immunodeficiency virus (HIV) patients; no patient in the trial had co-existing HIV. It had no effect on monocytes and eosinophils. Neutrophil count reduced.
- Exploratory studies needed to study if these cases produced more antibody levels.
- Tocilizumab (single cytokine blocker) is losing its role in COVID-19. If supportive care and steroids are given in time in hypoxia, it is not needed. But in some patients where cytokine storm is just in place, it may have some role.
- Multiple cytokine inhibitors (Baricitinib) seem promising, but whether there is a need to use them is not clear.
- Oral baricitinib is available in India; it blocks inflammation in 72 hours, but not thrombosis. It is given in rheumatoid arthritis patients.
- Favipiravir and remdesivir should be given when the virus is replicating; they have no role if given after 7-9 days when replication is over; should be started on 3rd-4th day.
- There is less data on ivermectin and doxycycline in Mumbai; seem promising and a good randomized controlled trial (RCT) is needed.
- To prevent thrombosis, aspirin can be given; if the patient has developed a slightly higher microvascular state, low molecular weight heparin (LMWH), warfarin, acenocoumarol, dabigatran, rivaroxaban and apixaban. If these do not work, then thrombolysis can be done with streptokinase, tenecteplase.
- When we treat COVID-19, there are three options: Attack the virus within 48 hours; attack the inflammation (early diagnosis); attack the thrombosis and recover the damage (fibrosis) done.
- Up till now, mucosal vaccines have been only up to 60-70% effective. So, re-dosing (multiple doses) is required.
- All nucleic acid vaccines are highly inflammatory and may cause inflammatory reactions.

Minutes of Virtual Meeting of CMAAO NMAs on "Can I Refuse Vaccine"

12th December, 2020 (Saturday, 9.30 am-10.30 am)

Participants, Member NMAs: Dr KK Aggarwal, President-CMAAO; Dr Alvin Yee-Shing Chan, Hong Kong, Treasurer-CMAAO; Dr Marthanda Pillai, India, Member-World Medical Council; Dr Marie Uzawa Urabe, Japan Medical Association; Dr Angelique Coetzee, President-South African Medical Association; Dr Md Jamaluddin Chowdhury, Bangladesh Medical Association; Dr Ashraf Nizami, Pakistan Medical Association

Invitees: Dr Russell D'Souza, Australia UNESCO Chair in Bioethics; Dr S Sharma, Editor-IJCP Group

Key points from the discussion

- Analysis of graphs of the top 15 countries in the world shows that India, Argentina and Poland are still experiencing the first wave of the infection. The remaining 12 countries have seen more than one wave; of these, the second wave is shorter than the first wave only in Brazil and Colombia. In the rest, the second peak is significantly higher than the first wave.
- There is a law in the US where employers can fire employees who have not taken the flu vaccine. Relief has been granted three times on religious, medical grounds and personal liberty.
- Vaccine is now available in the UK, US, UAE and China.
- With the impending arrival of the COVID-19 vaccine in many more countries, many questions will come up, which need to be answered.
- Can a government/hospital mandate vaccination?
- Is informed consent required before vaccination?

- Is an employer exempt from paying workers' compensation to an employee who refuses to be vaccinated and then contracts the virus while on the job?
- Can a prospective employer require COVID-19 vaccination as a pre-condition of employment?
- Can a patient refuse treatment from a doctor who has not taken the vaccine?
- If a hospital allows employees to refuse vaccination and keep working and an outbreak occurs, and it is suggested through contact tracing that unvaccinated workers infected patients, will a court hold the hospital liable for patient's damages?
- It was suggested that CMAAO countries could develop an awareness program about the efficiency and side effects of the vaccine, including the need of the vaccine.
- It is important to maintain the recommended storage temperature and transport temperature. Storage temperature for the Pfizer COVID-19 vaccine is -70°C, which is not feasible in developing countries.
- How will the vaccine act if given to an asymptomatic positive individual?
- If a person already infected with the virus is given the vaccine, will there be a reaction?
- CDC says that people who have already had COVID "may still benefit from getting vaccinated". WHO has not defined any selection criteria. All must take it.
- Professional autonomy is not absolute. So, if the law says that vaccine has to be taken, we have to take it.
- Pfizer has been granted legal indemnity by the UK government and the company will not be liable for any adverse event due to COVID-19 vaccine.
- Patients with history of severe allergy/ anaphylactoid reaction should not take the m-RNA vaccine.
- Health officials have cautioned that those who take Sputnik V vaccine should give up alcohol for almost 2 months. This has already caused a backlash.
- No vaccine is mandatory but people still take it. But it cannot be given without informed consent.

Colchicine in Patients with Stable Coronary Artery Disease

For patients with chronic coronary disease receiving other secondary preventive strategies, colchicine 0.5 mg (or 0.6 mg) daily should be added to the medical regimen (Grade 2B).

Chronic inflammation is a risk factor for CAD events, such as myocardial infarction (MI), and colchicine is known to have anti-inflammatory effects.

In the LoDoCo2 trial, more than 5,500 patients with chronic coronary disease were randomized to receive 0.5 mg of colchicine once a day or placebo. After a median follow-up of around 2½ years, those receiving colchicine had a decreased risk of MI (3.0% vs. 4.2%) and ischemia-driven coronary artery revascularization (4.9% vs. 6.4%) in comparison with the control group. Treatment was well-tolerated except for a small increase in myalgias. Uptodate suggests adding colchicine 0.5 mg (or 0.6 mg) once daily to other secondary preventive strategies in patients with stable CAD.

Trial

The LoDoCo2 trial randomly assigned 5,522 patients, 85% men, with chronic coronary disease to 0.5 mg of colchicine once per day or placebo.

After a median follow-up of around 2½ years, the risk of MI was 30% lower in the colchicine group (3.0% vs. 4.2%), and there was a 25% lower risk of ischemia-driven coronary artery revascularization (4.9% vs. 6.4%).

The difference between the two groups in the risk of death from any cause (2.6% vs. 2.2%, respectively) was insignificant. Except for a somewhat greater rate of myalgia with colchicine (21.2% vs. 18.5%), no other significant adverse events were seen.

(*Source: UpToDate; N Engl J Med. 2020;383(19):1838.)*

Effect of Colchicine on hs-CRP

- Among patients with stable CAD, raised levels of biomarkers of inflammation, including high-sensitivity CRP (hs-CRP) ≥2.0 mg/L, predict future vascular events.
- Long-term low-dose colchicine is safe and effective for dampening inflammation.
- An open-label pilot study evaluated whether colchicine could significantly lower hs-CRP in patients with stable CAD having hs-CRP was ≥2.0 mg/L, despite taking both aspirin and high-dose atorvastatin therapy.
- Plasma hs-CRP level was measured in 200 patients

with clinically stable CAD taking aspirin and atorvastatin.

- In 64 patients, hs-CRP was ≥2.0 mg/L.
- In 20 of these patients, hs-CRP was measured again at 2 weeks (no treatment group), and in 44 patients, hs-CRP was measured again after 4 weeks of colchicine 0.5 mg twice daily (treatment group).
- In the no treatment group, mean baseline hs-CRP did not decrease significantly, (4.28 ± 2.03 mg/L at baseline and 3.70 ± 2.30 mg/L after repeated measurement; mean change 11.0%).
- hs-CRP appeared to decrease in all patients given colchicine treatment (mean baseline hs-CRP decreasing from 4.58 ± 2.05 to 1.78 ±1.38 mg/L ($p < 0.001$), absolute decrease of 2.80 mg/L and a relative decrease of 60%).
- In 28 patients (64%) in this group, the decrease in hs-CRP was >50% from baseline, and in 31 patients (70%), hs-CRP decreased to <2.0 mg/L.
- There were no significant side effects. Low-dose colchicine (0.5 mg twice daily) has the potential to effectively decrease hs-CRP in patients with clinically stable CAD and increased hs-CRP independent of aspirin and atorvastatin use.

(*Source: Am J Cardiol. 2007; 99(6):805-7.*)

Colchicine is an anti-inflammatory drug, indicated for the treatment of pericarditis or gout. In the COLCOT trial, 4745 patients with MI within 30 days who were receiving optimal medical therapy were randomized to colchicine 0.5 mg daily or placebo.

After a median follow-up of about 2 years, the risk of primary composite endpoint (death from cardiovascular causes, resuscitated cardiac arrest, MI, stoke or urgent hospitalization for angina leading to coronary revascularization) was found to be lower in the colchicine group.

This result was largely guided by lower risks of angina and stroke. Adverse events were generally similar in the two groups.

While the results of COLCOT appear promising, treatment with colchicine is not given on account of the absence of improvement in hard endpoints such as cardiovascular death or MI and a relatively high discontinuation rate (about 18.5% in both groups).

(*Source: N Engl J Med. 2019;381(26):2497.*)

In Non-CU Patients, Day 2 Blood Sugar of Over 250 or Less Than 70 Associated with Poor Outcomes in Patients with COVID-19

Both hyperglycemia and hypoglycemia were found to be associated with poor outcomes in patients with COVID-19, in a study published in *Diabetes Care*.

An analysis of 1,544 patients with COVID-19 from 91 hospitals in 12 states revealed that glucose level at admission was a robust predictor of death among the 360 patients directly admitted to ICU and severe hyperglycemia after admission strongly predicted death among the 1,184 patients admitted to a non-ICU setting. Of the patients, 279 (18.1%) died in the hospital. The mortality for ICU patients (31%) appeared to be nearly twice that in non-ICU patients (16%).

Among non-ICU patients, severe hyperglycemia (blood glucose >250 mg/dL) on **Days 2 to 3 had an independent association** with high mortality compared with patients with blood glucose <140 mg/dL. This relationship was not significant for admission glucose.

In patients who were admitted directly to the ICU, severe hyperglycemia on admission was tied to increased mortality. This relationship was not significant on Day 2. Hypoglycemia (blood glucose <70 mg/dL) was also found to be linked with increased mortality.

(*Reference: https://care.diabetesjournals.org/content/diacare/early/2020/12/08/dc20-1857.full.pdf; Source: Diabetes Care*)

Are Old Vaccines Helpful Against COVID-19?

Vaccines stimulate broad, innate immune response, which plays a vital role in fighting COVID-19. Can this approach bridge the time until entire populations are vaccinated, particularly against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)?

Three vaccines are dominating the discussion:

- Bacillus Calmette-Guérin (BCG) has been tied to milder courses of infection for respiratory syncytial virus, human papillomavirus, herpes simplex and influenza. Fifteen clinical trials are evaluating it for COVID-19; however, a drawback is the 1% rate of adverse events.
- Oral polio vaccine (OPV) has also been associated with milder infections for decades. It decreases the morbidity from influenza 3.8-fold.
- Measles-mumps-rubella (MMR) drew the focus in the spring when 955 sailors aboard the U.S.S. Roosevelt tested positive for COVID-19, while only 1 was hospitalized. As recruits routinely

receive MMR, re-vaccination of others might prevent the fatal inflammation of COVID-19.

A virus that enters the body comes across the complex cascade of defensive proteins that make up innate immunity. This includes cytokines, present in mucosal sites in the lungs, nose and genitals, and that recruit immune cells. Complement proteins also kill the viruses. Being non-specific responses, they're called innate. Cells of innate immunity include **macrophages, neutrophils and natural killer cells,** and the **epithelial and endothelial cells** that interface the circulation. If innate immunity is unable to contain an infection, **the adaptive immune system starts work: T cells, B cells and antibodies** bring specificity and memory. Adaptive immunity also hikes cytokine and complement secretion.

A cytokine storm is the **turn to the dark side of the innate response.**

An RNA virus delays interferon (a cytokine) production, blocks signals and escapes natural killer cells, and replicates explosively until it invades the bloodstream. A wave of inflammation goes through linings and lymphoid tissues as the adaptive response pours out more cytokines. **This scenario would not happen if the innate immune system is 'trained' to make interferon early** and get ahead of the virus. It will recruit cells to clear the damage so that the adaptive immunity isn't kicked into overdrive. The virus will be controlled, or at least slowed, and the patient will be better.

Children have a strong innate immune system because they don't have pre-existing antibodies and T cells, because they haven't seen the pathogens. Children have enough innate response to SARS-CoV-2 even if viral loads in the nose are high. But they don't get as sick with respect to the respiratory tract, developing pneumonia. **Innate immunity is even intact in MIS-C, which points to a delayed impairment of adaptive immunity.**

(*Source: Medpage Today*)

US FDA Grants EUA for Moderna's COVID-19 Vaccine

The US Food and Drug Administration (FDA) becomes the second vaccine to be authorized for emergency use in the United States, and will likely increase the number of doses available in the coming days. The EUA for the Moderna vaccine comes after a review by the independent Vaccines and Related Biological Products Advisory Committee (VRBPAC) members on December 17, which voted 20-0 with one abstention, recommending the EUA. The vaccine is authorized for use in individuals aged 18 years and above. (*Medscape*)

Johnson & Johnson may Apply for EUA in February

Johnson & Johnson has recruited 45,000 participants in phase III clinical trials of its coronavirus vaccine and hopes to apply for a EUA from the FDA in the month of February. Data from phase III is expected to be available by January end.

If the data suggest that the vaccine is safe and effective, the company is expected to submit an application for EUA to the US FDA in February.

This vaccine doesn't need to be frozen and only requires one dose. Johnson & Johnson had to halt its clinical trials in the fall as a participant developed an "unexplained illness". However, the trials were re-started 2 weeks later.

Johnson & Johnson said earlier this month that it would reduce the number of phase III participants from its original aim of 60,000. (*Medscape*)

Two Doses of Oxford/AstraZeneca Vaccine Provoked Good Immune Response: Early Data

Oxford's COVID-19 vaccine candidate induces a better immune response when a two full-dose regimen is used rather than a full-dose followed by a half-dose booster, as per the university. The vaccine candidate, licensed to AstraZeneca, had in previously published interim late stage trial results, shown higher efficacy when a half-dose was followed by a full-dose, compared to a two full-dose regimen. More work is needed; however, to confirm the result. The subgroup details from the Phase I/II clinical trials made no reference to the half-dose/full-dose regimen, which, according to Oxford, had been "unplanned" but approved by regulators. (*Reuters*)

Vitamin D Deficiency in COVID-19 Quadrupled Death Rate

- Vitamin D deficiency on hospital admission was found to be associated with 3.7-times increased likelihood of dying from COVID-19, suggests a new study.
- Around 60% of patients with COVID-19 were vitamin D deficient at the time of hospitalization, with men in the advanced stages of COVID-19 pneumonia having the greatest deficit.
- The results were independent of comorbidities known to be affected by vitamin D deficiency, stated the authors, led by Dieter De Smet, MD, from AZ Delta General Hospital, Roeselare, Belgium.

The findings emphasize the need for RCTs specifically focused at vitamin D-deficient patients at intake, and urge for general avoidance of vitamin D deficiency as a safe and inexpensive possible mitigation of the pandemic, suggest researchers in their article, published online in the *American Journal of Clinical Pathology*.

A 3.7-fold hike in death rate if someone's vitamin D level was below 20 [ng/mL] is staggering. It is among the most important risk factors to consider.

It is not clear if vitamin D levels act as an acute-phase reactant, declining due to the infection, with larger fall indicating more severe disease, or whether vitamin D deficiency is leading to worse outcomes.

This is possibly due to the loss in the protective action of vitamin D on the immune system and against the SARS-CoV-2-induced cytokine storm.

The study had reported more prevalent vitamin D deficiency among men compared to women, most likely because women are more often treated with vitamin D for osteoporosis.

The study should prompt all clinicians and health authorities to consider vitamin D supplementation as an additional tool in the fight against COVID-19, more so for the prevention of infection in individuals at high risk of both COVID-19 and hypovitaminosis D, such as the elderly. (*Medscape*)

Can RAS Dysfunction Explain COVID Effects?

An article published in the *New England Journal of Medicine* proposed endothelialitis as the unifying mechanism for the extensive pathology of COVID-19. An alternative view indicates that virus-induced upregulation of the renin-angiotensin system (RAS) is accountable for the diverse systemic effects of COVID-19. (*Medscape*)

Results of ACTIV-3 Trial Published

Preliminary results of a Phase 3, randomized, placebo-controlled clinical trial evaluating the investigative monoclonal antibody LY-CoV555 in hospitalized COVID-19 patients have been published in *The New England Journal of Medicine*. The antibody was found not to yield clinical benefit compared to placebo. (*NIH*)

REGN-COV2 in Outpatients with COVID-19

REGN-COV2, a neutralizing antibody cocktail, was shown to reduce viral load in nonhospitalized patients with COVID-19, with a greater effect seen in patients whose immune response had not yet been initiated or who had a high viral load at baseline, in an interim analysis of an ongoing phase 1-3 trial published in *The New England Journal of Medicine*. (*DG Alerts*)

Pulse Oximeters

- Pulse oximeters have 3-fold higher odds of missing oxygen starvation in Blacks as compared to whites.
- Among 1,609 patients treated earlier this year at the University of Michigan Hospital, in Ann Arbor, 11.7% of Blacks were found to have an alarming arterial oxygen saturation of <88%, measured directly in the blood, despite their pulse oximetry levels showing the normal range of 92-96%. The devices, originally designed for people with light skin, missed low oxygen levels in only 3.6% of whites, which represented a statistically significant difference.
- When investigators assessed data from 8,392 other patients treated at 178 ICUs during 2014 and 2015, pulse oximeters appeared to miss low blood oxygen levels in 17.0% of Black patients compared to 6.2% of whites; again a significant difference.
- Dr Michael Sjoding of the University of Michigan Medical School, and colleagues noted that reliance on pulse oximetry to triage patients and adjust supplemental oxygen levels may place Black patients at an escalated risk for hypoxemia. The analysis is published in the *New England Journal of Medicine*.
- The devices make use of red and infrared light to gauge the color of hemoglobin, which darkens to purple-red as the oxygen levels decline. Since pulse oximeters were mostly tested on whites when they were developed, they are calibrated for light skinned individuals.
- The racial discrepancy was evident even after the investigators excluded people with diabetes and elevated carboxyhemoglobin levels. (*Source: Medscape*)

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