UPDATE III

CORONA VIRUS DISEASE (COVID-19) AND HOMEOPATHIC PERSPECTIVE

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Abstract: The author discusses the role of Homoeopathy in asymptomatic corona positive cases. The progression of pneumonia and other pathologies is a challenging concern and the author formulates many clinical rubrics such as Cytokine storm, Ferritin high, Hypoxia, Fibrosis etc. for the aid of homoeopathic warriors. The author integrates the pathophysiological aspects with clinical stages and homeopathic remedies in order to help homeopathic intervention in OPD and IPD cases. The role of remedies viz. Antim ars, Ferrum phos, Veratrum viride, Tuberculinum etc. has been underscored along with their relations with a host of remedies. The readers are requested to read earlier three documents – original, update I and update II in connection with Covid-19 and also the author's two papers – 'Acute prescribing : Challenges and solutions' and 'Therapeutics of Acute Respiratory Tract Infections'.

QUESTIONS

1. What is your approach towards asymptomatic patients?
2. How to treat and prevent lung and other organs pathologies during progressive stages of Covid-19? How can we increase the function of lungs after the patient is out of Covid-19 disease?
3. As a homeopath, how do you interpret the role of micro RNA in Covid-19?

QUESTION

What is your approach towards asymptomatic patients?

ANSWER

This one is a pertinent question. Many homeopaths think that if there are no symptoms, no remedy is needed at both preventive and treatment level. But there are many issues that need to be looked into. If we accept that 'no' homeopathy is needed, we are outrightly rejecting the contribution of Hahnemann of GE. This is not only discredit to Hahnemann but also to fellow homeopaths who used the concept of GE in several infectious diseases such as Scarlet fever, Dengue, Swine flu, Japanese encephalitis, Chikungunya**,** Viral hepatitis, Spanish flu, etc. in successful manner. Should this golden treasure of homeopathy's success stories be buried because corona virus doesn't produce symptoms in some cases? Further, who will guarantee that today's asymptomatic will remain symptom-free throughout life? The cases are reported that today's asymptomatic stage is replaced by symptoms and even rapid progression within couple of days. Are we risking the lives of the asymptomatic patients by

refusing them the HP based on the Law of Similars or Isopathy? The vaccination in conventional medicine is given to healthy people. How many homeopaths raised the question to allopaths, 'why do you give vaccination to healthy people'?

It is interesting to see that there is a clear demarcation line between healthy individual and asymptomatic patient.

I reiterate through three sentences.

- Prophylaxis is an employment before getting illness.
- If you ask about asymptomatic patient with positive test, it means that virus has entered in the body and it is not prophylaxis. But he can be helped by GE which can arrest the development of replication of virus in the body.
- Subclinical stage cannot be ascertained unless it is known to be positive.

Asymptomatic patient

A Covid-19 patient is considered asymptomatic if he/she has already contracted the virus (as he is tested positive) but doesn't show any symptoms such as fever, dry cough, pain in throat, loss of taste or smell, shortness of breath etc. Such an infected person continues his daily life without having any discomfort but continues to be a 'silent spreader' of the disease. As a result, they end up transmitting the virus to many uninfected individuals.

Pre-symptomatic

These are the infected individuals who do not develop any Covid-19 symptoms almost a week after contracting the novel coronavirus. But they later display symptoms such as coughing, fever, loss of sense of taste and smell etc.
In the above two types, the patient has corona but not covid-19 as a disease.

Mildly symptomatic

Individuals who display mild symptoms, such as common cold/cough, fever and do not feel exceptionally sick as Covid-19 has been stereotyped to be.

These are three types of Covid-19 carriers which can induce silent spread of the infection.

I put below the recent research.

Observations on outbreak of Covid on Cruise trip` `Diamond Princess' (New England Journal of Medicine)

- Of 712 patients being infected 410 (58%) were asymptomatic. The symptoms developed only in 10% asymptomatic Covid positive patients. Majority of asymptomatic positive cases remained positive throughout the observation period.
- The median time(from test positivity) to develop symptoms was 4 days (Range 3-7 days) . That means they were transmitting infection without symptoms.

- The Risk of duration of Pre-symptomatic was higher with advancing age, hypertension and diabetes.
- The median duration of RTPCR Positivity was 9 days (Duration between positivise and negative test), range 3-21 days.

This study clarifies many facts

- A Large proportion of Covid positive patients (58%) are asymptomatic throughout the period (range 3-21 days , Median 9 days)
- Time duration from Test positivity to development of symptom is 4 days (3-7 days)
- Asymptomatic patients transmit infection throughout the duration (median 9 days)

If a asymptomatic or pre-symptomatic person happens to live in a red zone, a homeopath has every reason to be particularly wary of the silent COVID spread and he must institute HP for arresting the further progress of Covid-19. Mildly symptomatic patients must be treated as per presenting totality based on the principle of individualization.

If someone in the family is over sixty five years of age and already has other health conditions, such as hypertension, diabetes, asthma, heart disease, cancer etc., he is in need of HP.

Demographic data as obtained across various countries about Case Fatality Rate (CFR) of Covid-19 by age shows very large differences. It is not that only old people are dying; the children and your people have also succumbed to the virus.

Assume that there is not a single Covid-19 patient in Andaman Island. It is obvious that no GE is required. However, there is one family of 10 people in Andaman and amongst them one is recently tested covid positive. GE is required for the family and also for those who have come in contact.

QUESTION

How to treat and prevent lung and other organs pathologies during progressive stages of Covid-19? How can we increase the function of lungs after the patient is out of Covid-19 disease?

ANSWER

The issue is not only of prevention of lung pathologies but also of cascade of progressive pathologies in the whole body.

This is a big question and unless one studies the patho-physiological and clinical aspects, one can't dive deep in homeopathic management.

PATHO-PHYSIOLOGICAL ASPECTS OF COVID-19 IN THE LIGHT OF NEW FINDINGS

Corona virus has an affinity not only for the ACE2-alveolar receptors, but also for ACE2 receptors on the vascular endothelium. This leads to vasoconstriction and thrombotic intravascular activity. Bleeding occurs through mucosal macrophages activity after endothelial damage. The uncontrolled activation of the coagulative cascade may be explained on this line.

The sequential pathway is not that there is pneumonia followed by disseminated vascular coagulation and multi-organ failure. Rather, endothelial damage, immune cascade and systemic vasculitis are followed by pneumonia, ARDS, renal failure, disseminated vascular coagulation and multi-organ failure. This knowledge will help to perceive the clinical stage of the patient and if a right homeopathic remedy is given at right stage, the further progress of the disease could be averted(95).

CYTOKINE STORM (27, 92)

The 'cytokine storm' is the most dangerous and potentially life-threatening event related to COVID-19.

The cytokine storm, and the consequent ARDS, results from the effects of a combination of many immune-active molecules. Interferons, interleukins, chemokines, Colony-stimulating factors and TNF-alpha represent the main components involved in the development of Cytokine storm.

LABORATORY ABNORMALITIES

- Decreased number of immune cells
- Elevations in markers of kidney or liver damage
- Elevations in inflammatory markers (like CRP)
- Abnormalities in markers of blood clotting
- Elevated ferritin (involved in infection response)

Symptoms

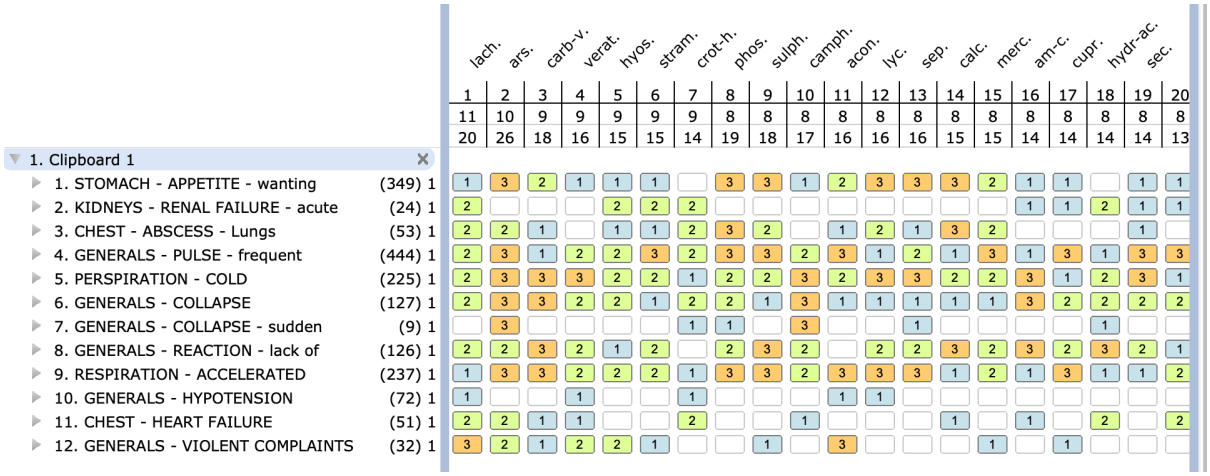
Symptoms include fever and chills, fatigue, loss of appetite, muscle and joint pain, swelling of extremities, nausea, vomiting, diarrhoea, rashes, cough, shortness of breath, rapid breathing, rapid heartbeat, low blood pressure, cardio-vascular collapse or shock, acute renal failure, sepsis syndrome, seizures, headache, confusion, delirium, hallucinations, tremor, and loss of coordination.

Out of the above symptoms, we should focus on those symptoms which are intense and indicative of a cytokine storm.

RUBRICS RELATED TO CYTOKINE STORM

- Stomach, appetite, wanting
- Kidneys, renal failure, acute
- Chest, abscess, lungs

- Generals; pulse; frequent
- Blood, oxygen, diminished
- Perspiration, cold
- Generals; collapse
- Generals; collapse; sudden
- Generals; reaction, lack of
- Generals; violent complaints
- Generals; vitality decreasing
- Respiration; accelerated, quick
- Clinical; hypotension



Cytokine storm (a rubric prepared by Dr Ajit Kulkarni)

- ACET-AC ACETAN ACON *adren* AM-C ANT-M ANT-A ANT-T ANTHR ANTIPYRIN APIS arn ARS ARS-H ARS-I *bapt* bell calc-ar CAMPH CARB-AC CARB-AN CARB-V CARBN-S *carc* CHIN crat CROT-H CUPR CUPR-ACET CUPR-AR DIG ELAPS ferr *ferr-p* glon graph HELL HYOS *hippoz* HYDR-AC iod *ion-rad* KALI-BR kali-n KALI-P kreos LACH lat-m LAUR lob lyc MED MERC merc-c merc-cy mur-ac NAJA op ox-ac ph-ac PHOS plb PSOR PYROG SEC SENEG SEP stann stram STRONT-C STRY SUL-AC SULPH TAB thymu TUB VERAT VERAT-V vip ZINC zinc-m

MIASMATIC ASSESSMENT

Cytokine storm represents the tubercular miasm. If the pathological changes become irreversible, the patient lands into syphilitic miasm.

Apart from many remedies which can be used to tide over the acute crisis as developed after cytokine storm, one of the nosodes is **Tuberculinum**.

CLINICAL IMPORTANT POINTS

It is observed in Covid-19 cases that the fever gets resolved within a span of 3-5 days even without any treatment. Then oxygen saturation has to be monitored. Usually it starts falling from 5-7 days onwards. In severe cases, it may fall early too.

If fever is intense (39.4°C (103°F) to 40°C (104°F), right from the onset, usually it is not Covid-19 infection. Maximum patients demonstrate the pattern of response of gradual onset followed by rapid development.

Usually a right homeopathic remedy should bring fever to normal within 48 hours. If fever is the same, or increased and generals are not better, reconsider the remedy. If you are damn sure that the remedy is the same, give the same remedy in high potency every 3-4 hours.

It is necessary to explore biomarkers to determine the extent of lung lesions and disease severity.

Patients with positive RT-PCR had significantly higher neutrophil count and C-reactive protein (CRP), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and Urea levels in serum. In addition, patients with positive RT-PCR had lower white blood cell (WBC) count and serum albumin level compared to others. ALT, CRP, NEU, LDH and Urea had very good accuracy in predicting cases with positive RT-PCR for COVID-19, respectively. Interleukin 6 (IL6) and Ferritin levels also have a big share in prognosis. D-dimer and decreased blood platelet are helpful to identify early-stage patients with poor prognosis.

D-dimer is a degradation product of crosslinked fibrin resulting from plasmin cleavage. Elevated D-dimer in COVID-19 patients is associated with higher mortality and is considered a sensitive biomarker to rule out venous thromboembolism.

CRP is an indicator of inflammation. There is correlation between CRP levels, lung lesions and disease severity and this can provide reference for clinical treatment. CRP levels can be used in the early diagnosis of pneumonia and patients presenting with severe pneumonia are found to have high CRP levels. Hence, CRP levels should be used as a key indicator for disease monitoring.

Lactate dehydrogenase (LDH) is also a key indicator. LDH is an intracellular enzyme found in cells in almost all organ systems, Severe infections may cause cytokine-mediated tissue damage and LDH release. Since LDH is present in lung tissue (isozyme 3), patients with severe COVID-19 infections can be expected to release greater amounts of LDH in the circulation, as a severe form of interstitial pneumonia, often evolving into acute respiratory distress syndrome, is the hallmark of the disease.

Interleukin-6 (IL-6) is a multi-functional cytokine that regulates immune responses, acute phase reactions, haematopoiesis and may play a central role in host defence mechanisms. IL-6 is usually not produced constitutively by normal cells, but its expression is readily induced by a variety of cytokines, lipopolysaccharide or viral infections.

When COVID-19 infects the upper and lower respiratory tract it can cause mild or highly acute respiratory syndrome with consequent release of pro-inflammatory cytokines like IL-6. Increased IL-6 in serum is expected to predict the severity of pneumonia and the prognosis of patients.

Ferritin is the cellular storage protein for iron. Elevated levels of ferritin, or hyperferritinemia, may indicate severe COVID-19. Ferritin is able to activate macrophages which play a critical role in innate immunity. This is evidenced by hyperferritinemia in patients with septic shock. When activated, macrophages begin to secrete cytokines. At high levels, the so called "cytokine storm" develops. Thus, hyperferritinemia has been associated with severity and adverse outcome.

Normal ferritin levels range from 12 to 300 nanograms per milliliter of blood (ng/mL) for males and 12 to 150 ng/mL for females. In Covid-19, if the ferritin level crosses 500, you must expect active lung pathology and if it crosses 1000 ng/ml, the prognosis is not good.

High Ferritin level (a rubric prepared by Dr Ajit Kulkarni)

Clinical; Ferritin, high: adren ant-c antoa ant-t arn ARS ars-h ars-i bell bry cadm calc-a calc calc-p CHIN CHIN-AR CHIN-S cob-n cupr FERR-AR FERR-I FERR-M FERR-P HEP iod ip LACH lyc merc mur-ac nat-m Phos, plb PULS sec, SUL thea TUB vanad verat ZINC

It is interesting to note that excess of iron in the body causes skin colour change which becomes bronze and Adren, Sec and Tub do cover this rubric (Skin; bronze).

In this rubric, there is Ant-c also. If one Antimony is available, we can include other two grand antimony remedies – Antim-ars and Antim-tart and this explains why they are useful in averting the lung pathologies and cytokine storm.

ROLE OF TUBERCULINUM

Incidentally, I mention the recent finding that COVID-19 could accelerate activation of dormant tuberculosis. If the novel coronavirus activates a sizable proportion of these dormant infections, it could severely upset the global health. Tuberculinum (and Bacillinum too) could help avert a global TB pandemic.

The researchers say that CoV infections could be causing lung inflammation that leads to reactivation of dormant TB in the lung. Others say that both lung lesions and liver infection by the mycobacteria, are enhanced by the presence of influenza A which also show a type I interferon signaling pathway that increases mycobacterial growth.(93)

It is clinically verified that patients of TB are highly susceptible to influenza.

In Tuberculinum, we have given the following indications in Absolute MM, "Rapid and complete physical break-down without any apparent cause, but generally after acute infections as pneumonia, influenza, no signs of vital reaction, declining, a running down without finding the right relief, or at best but temporary, in spite of well-selected remedies and best attention which fail to impress; they simply do a lip-service, without touching the deep-rooted dyscrasia and chronicity". (43)

Tuberculinum should be a standby, an immediate deployment remedy, not only during Covid treatment but also after the patient has recovered from Covid.

In view of neurological affection of corona virus, resulting in necrosis and thrombosis of brain, the patient is likely to develop organic psychosis and cognitive and behavioural problems and Tuberculinum is one of the prominent remedies.

For the prevention of progressive pathologies, I recommend two grand remedies Ferrum phos and Ver-viride. Early intervention of them in Covid-19 cases will help avert the complications.

FERRUM PHOS

Essence

- First stage of all inflammatory affections.
- Can be use in early stage of all febrile conditions before exudation set in.
- Susceptible to chest problems.
- Hyperaemia. Hemorrhagic diathesis. Hemoptysis of pure blood in pneumonia.
- Covers thrombosis and disseminated intravascular coagulation.
- Marked Prostration.
- Act on hemoglobin, can improve oxygen carrying capacity of haemoglobin.

Larynx

- Laryngitis with fauces inflamed and red. Croup. Hoarseness. Much mucus in throat and rattling in chest.

Cough

- Short, painful, tickling. Hard, dry and sore chest. < night. Hacking; < morning and evening; bending head forward or touching larynx; night or during day when asleep. Incessant, tormenting cough → epistaxis; < cold air.

Sputum

- Hemoptysis in pneumonia.

Chest

- Congestion of lungs.
- Very little thirst; first stage.
- Pleurisy; stitching pain < coughing and deep breathing.

Comments

Ferr-p. is a remedy par excellence for inflammation. It is indicated when there is consolidation. The concomitant is bright red flushing.

Ferr-p. usually runs a high temperature and has a rapid, bounding pulse.

Viral proteins rip off Ferritin from Hemoglobin which causes reduction in oxygen carrying capacity of RBCs leading to happy hypoxia. Abnormal Hemoglobinopathies is caused by corona virus and hemoglobin fails to carry sufficient O_2.

Ferrum remedies such as Ferrum ars, Ferrum met and Ferrum phos are indicated at this stage. Ferum phos avert Disseminated Intravascular Coagulation (DIC) and reduce the risk of thromboembolic episode (cerebrovascular accident like stroke, infarction and myocardial Infarction.

Relations

- Excitement and terror of Acon. is not observed in Ferr-p.
- Midway between sthenic activity of Acon. and Bell. and the asthenic sluggishness and torpidity of Gels.
- Earlier stage of Phos and Ver-vir.

VERATRUM VIRIDE (43)

- Suddenness and violence. No haemorrhage.
- Rapid violent congestive conditions.
- Inflammations any and everywhere.
- Shock: Peripheral circulatory failure and low blood pressure leads to: (Pictures) a vasovagal attack / syncope: pallor and cyanosis, cold, clammy sweat, slow respiration, dilatation of pupils, nausea and vomiting, loss of consciousness, slight convulsion of face, trunk, arms. Causes: emotions, sudden fright, sight of blood, violent pains, severe fatigue. Subacute shocks (*Nux-v.*). Nervous shock, not due to depletion (unlike *Carb-v*. or *Verat.).*
- Tendency to cyanosis.
- Collapse: Vomiting, very slow pulse, cold sweat.
- Coma: Spasms, blue face.

Nose:

- Sub-acute allergic conditions (Bellokossy).
- Nose looks pinched, blue, cold.
- Sneezing rapid and persistent, with warm biting feeling in mouth.

Respiratory:

- Dyspnea with cold sweat on face. Bronchial asthma. Stertor (*Op., Visc*.). Violent cough from the very start. Cough < entering a warm room from cold (*Phos*.). Spasmodic cough from spinal congestion or cerebral irritation, with spasms. Croup, membranous.

<u>*Pneumonia*</u>:

- Intense pulmonary congestion, great arterial excitement, high fever. Broncho-pneumonia patient not anxious (unlike *Acon.*). Old pneumonic congestions with superadded acute pleurisy (*Lyc.*). Pneumonia with full, fast pulse, faint at stomach, violent (cerebral) congestion, arterial excitement, oppression and heaviness on chest, nausea, vomiting, expectoration of pus and florid blood, high fever.
- Congestive and hepatization stages; flushed face like *Bell*., cold surface, constriction. Chest oppressed as from a heavy cold; with eruptive fever. Constrictive zigzag temperature. *Phos.* follows well later, dizziness, faintness on attempting to sit up, orthopnea, cold sweat and the ubiquitous nausea (symptoms of heart failure). (Burning) pain under one nipple. Croup with high fever, *Phos.* controls cough since but not fever, later convulsions.
- Respiratory congestions of hypertensives. Chronic chest affection. Pleurisy, early stage, stitching pain, cannot breathe, must hold sides (*Bry.*). Pleurodynia more on left side, pain spasmodic, > walking, with nausea and gush of sweat.
- Inflammatory affections of the heart and its membranes, especially those caused by infections.

Thermic:

- First stage of inflammatory fevers in robust, plethoric persons (*Ferr-p.* in anemic). Farrington says, "Rather pictures asthenic fever of a low type."
- Fevers beginning with chilliness, nausea, vomiting and cold limbs. Intense critical excitement during febrile state with cerebral congestion (due to vascular irritation) or irritation of spinal centres.

Relations

- A wonderful remedy in cytokine storm and application of this remedy will prevent damage to the lungs. It has a big sphere after Ferrum phos in tiding over the acute crisis.
- Represents like *Bell*. or *Ferr-p*., the acute invasive stage of inflammation and is useful until the inflammation is fully established, but not later unlike *Hep., Phos*. or *Sulph*. Has (like *Op.*) no anxiety or losing about as in *Acon.*, but has the sthenic violence of *Bell*. and also is the paleness of *Ferr- p.* wanting. According to some writers *Verat-v.* will not reach beyond the sthenic stage like *Bell*. etc., hence not useful in conditions like typhoid, septic fever etc.
- Brain group*: Bapt., Gels., Op., Phos., Verat-v., Zinc.*
- There are 3 trios to be considered.

    - Bry-Ferr-p-Ver-v
    - Ars-Ferr-p-Phos
    - Bry-Ferr-p-Phos

One of the important remedies for prevention of pathologies (after Ferr-p. and Ver-v.) is Ant-ars. A patient of Covid-19 where Ars-alb has helped only partially and the patient is developing severe breathlessness, think of Ant-ars.

ANTIM ARSENICOSUM (43)

- General sick feeling. Sense of weakness. Great prostration, strength rapidly ebbing.

Nose:

- Flapping of nostrils; with quietness and respiratory symptoms like *Bry.*

Respiratory:

- Excessive dyspnea; hard wheezing, rattling breathing, cannot lie, worse a.m.; emphysema.
- Cough: Loose, rattling, much mucous expectoration, < lying down, eating. Strangling cough.
- Chest: Colds, bronchitis, marked dyspnea, rattling, wheezing, cyanosis from hard breathing.
- Bronchiolitis; hyperventilation (rapid abdominal breathing), temp. 100, pulse 90, tongue clean, very restless, rapid heart- beats, CCF.

- Pneumonia: Catarrhal; with influenza. Hypostatic pneumonia. Old (right or 1eft sided) pneumonias. Pulmonary sclerosis from (or not from) myocardial disease; severer than that disease would warrant, with emphysema.
- Pleurisy: Serous or sero-fibrinous exudation on either side, but of right side it takes longer time for absorption. Pleurisy of left side. Old pleuritic exudations, with sense of weakness.
- Pleuro-pneumonia, esp. of (upper) left lung, with recent or old exudations; chiefly in desperate cases, threatening asphyxia, strength rapidly ebbing, pulse weak and rapid, cyanosis.
- Fibroid phthisis.

Heart:

- Pericarditis, with effusion. Myocarditis. Endocarditis; after influenza, systolic murmurs, loose coarse rales over entire right lung, weak feeble pulse, temperature subnormal, profuse cold sweat over face and upper part of body (Dr. Royal).
- CCF; left heart failure with nocturnal attacks of breathlessness, rattling, High B.P; after influenza, nephritis or pleurisy.

Relations

- Warm-blooded (unlike *Ars.,* but like *Ars-i.*). Restless (like *Ars.*) and rattling; expectoration (like *Ant-t.*).
- Similar: *Am-c., Ant-t., Ars., Aur., Hep., Lach., Merc., Phos., Puls., Stry-p., Sulph.*

HYPOXIA

Clinical; hypoxia (a rubric prepared by Dr Ajit Kulkarni)

- ACET-AC ACETAN ACON AM-C am-caust ANT-AR ant-c ANT-T APIS ARN ARS ARS-I atro AUR bapt BELL bov brom bry butho-t CAMPH carb-an CARB-V CARBN-S CHIN CHLOL crat CROT-H CUPR cupr-ar cur DIG FERR ferr-i FERR-P GRAPH HELL HEP HYDR-AC just kali-c *kali-cy* kali-i kali-n kreos LACH lat-m LAUR lyc merc merc-c mur-ac naja nat-s NICOT OP ph-ac PHOS PSOR *pyr*, *samb sec* spong stann *stry* SULPH TAB TUB VERAT VERAT-V vip zinc

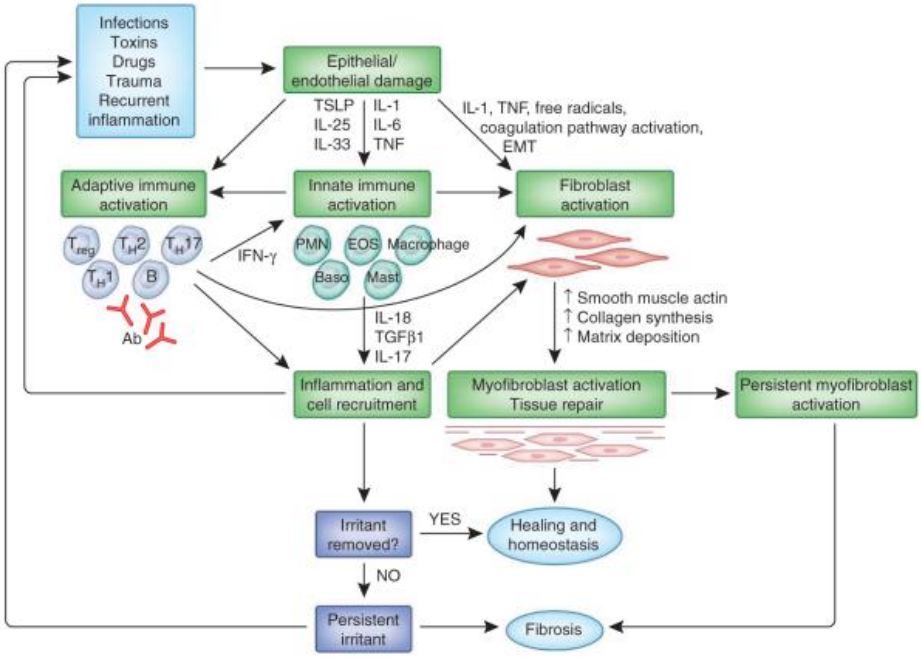
THE MATRIX OF FIBROSIS (88)

Mechanism of fibrosis is complex and an individual's immune forces are involved in repair.

Fibrosis is defined by the excessive accumulation of fibrous connective tissue (components of the extracellular matrix (ECM) such as collagen and fibronectin) in and around inflamed or damaged tissue, which can lead to permanent scarring, organ malfunction and ultimately death.

Fibrosis is the final and common pathological outcome of many inflammatory diseases. Although collagen deposition is an indispensable and typically reversible part of wound

healing, normal tissue repair can evolve into a progressively irreversible fibrotic response if the tissue injury is severe or repetitive or if the wound-healing response itself becomes dysregulated.



Overview of wound repair and fibrosis

Intrinsic, autocrine and epigenetic mechanisms regulate fibrosis.

As tissues become more fibrotic, the increased tissue stiffness and decreased elasticity result in mechanical stress, which exacerbates the tissue injury and perpetuate the activation of local fibroblasts.

Endoplasmic reticulum (ER) stress which leads to apoptosis of key structural cells (epithelial cells, endothelial cells and hepatocytes) is an important driver of fibrosis.

Also, telomere shortening in mesenchymal cells promotes injury leading to fibrosis.

Any remedy you select must attempt to target activation, proliferation and/or recruitment of fibroblasts.

Numerous studies have suggested that macrophages have stage-specific roles in fibrosis. Macrophages show profibrotic activity in the early phases of the wound-healing response by producing inflammatory mediators that can exacerbate tissue injury, such as IL-1β, TNF-α and reactive oxygen and nitrogen species. They also produce profibrotic mediators such as TGF-β1. But in the later stages of the wound-healing response, a subset of macrophages converts into a suppressive phenotype that expresses a variety of anti-inflammatory mediators, such as IL-10, Arg1, pr grammed death ligand-2 and Relm-α, which promote wound healing and direct the resolution of the inflammatory response.

There are many distinct immunological and molecular mechanisms that can contribute to the progression of fibrotic disease. Dysregulated innate and adaptive immune responses are major contributors to fibrosis. However, cell-intrinsic modifications in fibroblasts and other structural cells can also contribute to fibrosis.

MIASMATIC CORELATION

Fibrosis is a defense mechanism. Small fibrosis which don't hamper the functional capacity of the affected organ is Psoric. Hypertrophy of scar tissue (keloid formation) is Sycotic. Fibrosis the tissues of vital organs like lungs, liver, kidneys, brain etc. which affect the functionality of the affected organ is Tubercular in nature. A large area of fibrosis which has replaced the original tissue of the vital organ and such a structural damage causing functional incapacity of grave type denotes Syphilis miasm

CLINICAL; FIBROSIS (A rubric prepared by Dr Ajit Kulkarni)

- ANT-A ANT-T ARG-M ARG-N ARN ARS ARS-BR ARS-I AUR AUR-AR aur-m aur-m-n bar-c bar-i bar-m bar-s bell-p brom BAC BERYL bry calc CALC-AR CALC-F CALC-I CARB-AN CARB-V CARC chr-met caust CON crot-h CUPR cur dig dros ferr ferr-p, FL-AC FLOR-P germ graph GRIN HEP hip-ac HYDR IOD ion-rad kali-ar KALI-BI kali-c KALI-I kali-p kali-s LACH LAP-A LYC mag-c mag-m mag-s MED MERC MERC-D MUR-AC nat-ar nat-f NAT-M NIT-AC penic PHOS PLB PSOR ran-b sab sep SIL stann streptoc STRONT-C SULPH SYPH THIOSIN THUJ thyr TUB V-A-B Ver-alb, Ver-vir, vip X-RAY

Out of the above 71 remedies, taking into account the severe pathology of lung damage in Covid-19, I suggest following remedies as prominent.

- ANT-A ANT-T ARN ARS ARS-BR ARS-I brom BAC BERYL BRY CALC-AR CALC-F CARB-AN CARB-V CARC chr-met CAUST CON CROT-H CUPR cur dig dros ferr Ferr-p FL-AC GRAPH GRIN HEP hip-ac HYDR IOD ion-rad KALI-BI kali-c KALI-I LACH LAP-A LYC MED MERC MERC-D MUR-AC NAT-M NIT-AC penic PHOS PLB PSOR SIL stann STRONT-C SULPH SYPH THIOSIN THUJ TUB Ver-v Ver-alb

Arnica mont: Damage to the lungs is nothing but wounds and Arnica is always indicated for repair of the wounds. Two monogram words typify Arnica – Overworked, Stressed and Aged. Breathlessness and Covid-19 put a great load over the system. ARDS makes the lungs to work heavily, inflammation of heart makes the heart to work under stress plus if a patient has consumed Hydroxichloroquine, it is an additional load to keep the rhythm of heart harmonious. Thrombosis, bleeding are also the indications of Arnica. Arnica promotes fibrosis and saves the organ from further damage. Once fibrosis has set in, Arnica should not be used.

Ars-brom is when you get a strong halogen being attached to a strong Arsenic. Hence the remedy becomes syphilitic (like Ars-iod). The co-morbidity of diabetes, hypertension and obstinate intermittents are major pointers to Ars-brom.

Drosera is an interesting idea. It has action of absorption. Drosera heals the wounds as caused by tubercular bacilli or viruses by aiding fibrosis.

Kali remedies have fibrosis in their pathogenesis. Kali iodum ranks high and Kali-bich as well.

Crotalus hor and **Lachesis** are major anti-inflammatory remedies when there is cytokine storm, fulminating infection and affected tissues are rapidly damaged and fibrosed.

Remember, fibrosis is better than "ground glass opacities" in the lungs.

For improving the lung function, I suggest the indicated remedy (including the constitutional one) in LM scale to be repeated frequently.

Diaphragmatic and Pursed-lips breathing are important breathing exercises for improving the lung function.

QUESTION

As a homeopath, how do you interpret the role of micro RNA in Covid-19?

MicroRNAs (miRNAs) are a class of non-coding RNAs. They play an important role in regulation of gene expression.

miRNAs work by interacting with the 3' untranslated region, also known as 3' UTR of the target miRNA and induce its degradation and translational repression.

It has been reported that miRNAs bind to Toll-like receptors, activate downstream signaling events, and eventually lead to biological responses, such as tumor growth and metastasis and neurodegeneration. Thus, miRNAs act as an important chemical messenger (89).

MiRNAs play a vital role in the pathogenesis of various diseases, including viral infections, disease progression and inhibition (90). A recent study published in the journal Aging and disease has correlated MiRNA with aging.

The in-silico studies and pathway analysis have shown that that some of the human miRNAs targeting COVID-19 decreased with aging and underlying conditions. However further in vivo and in vitro studies can validate this (91).

We have already formulated 'Senile' as a word of Monogram for Covid-19. It means that the remedies that cover the Covid-19 pathogenesis and aging act on miRNAs. It is presumed that a proper indicted remedy will prevent the degradation and translational repression of miRNAs.

Let us take the example of **Conium maculatum**. Typical symptoms in covid-19 patients include fibrosis of lungs after the patient has recovered from corona virus infection. Conium is of particular importance in old age. It has action of rejuvenating the damaged tissues and thus it can be employed for fibrosis.

One of the indications of Conium is emaciation and it is a point of entry when one comes across it in Covid-19 patients.

Relations of Conium

Conium is a bridge between Gelsemium and Baryta carb. Conium is an intensified Bryonia. Note that all three – Gelsesium, Bar-carb and Conium have senility.

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