

# *COVID-19 & Allergy*

*Soheila Alyasin*

*Winter -1399*







# COVID-19 & Allergy

- Covid-19 (coronavirus disease 2019); severe acute respiratory syndrome
- China, December 2019 , pandemic, rapid increase in number of patients
- clinical progress & epidemiology : better prognosis and prevention
- underlying diseases : immunity : sever COVID-19:
  - age > 65y-o
  - chronic kidney disease
  - Obesity; BMI > 30
  - CVS – DM
  - Chronic pul disease
  - hypertension
  - cancer - smoking
  - immuno-compromised (ID- anti-inflammation – biologic - transplant – HIV)
  - chronic allergic diseases



**TABLE I.** Change in volume of pediatric asthma emergency department visits during the COVID-19 pandemic

<b>Timeframe</b>	<b>2019</b>	<b>2020</b>	<b>% Difference</b>
January	77	82	6.5
February	87	80	−8.0
March	98	60	−38.8
April	92	14	−84.8

# COVID-19 & Allergy

- ***Chronic allergic diseases : persist inflammation and tissue remodeling: susceptibility to infection***
- ***Asthma and COVID-19 : UK biobank & Seattle : sever outcome but no in Wuhan reports***
- ***Asthma, AR & Atopic dermatitis: exacerbation : common respiratory viruses***
- ***Impaired innate immunity (IFN type 1 ) : facilitate spread of virus***

• ***Yang JM. Allergic disorder ...COVID19. JA CLIN IMMUNOL.NOV 2020***

# COVID-19 & Allergy

- *But Kimura et al : A) type 2 inflammatory cytokines: interleukin IL:13 modulate expression of molecules that mediate SARS-COV2 host cell entry in atopic airway cell:*
- *B) Induce reduction Angi. converting enzyme 2*
- *C) Increase TMPRSS2 expression (complex pathway):  
rate and severity in asthma need to be determined*

# **COVID-19 & Allergy**

*Hypothesis: Allergy increase risk and outcome (death- ICU-hospital days)*

*in Korea 219,959 tested for SARS-COV2 , investigated allergic diseases among 7340 COVID patients and potential association of allergic disorder with likelihood of COVID-19*

- *mean age 49 +-19.9 y-o, male 47%, 14.9% asthma, 63% allergic rhinitis, 3.9% atopic dermatitis*
- *among 7340 COVID-19s :mean age 47+- 19 y-o, male 40%, 9.9% asthma, 57 % allergic rhinitis, 1.9 % atopic dermatitis*
- *Non allergic asthma: risk of COVID-19 ; OR 1.34*
- *Worse & more serious outcome in allergic asthma*

# COVID-19 & Allergy

- *Discussion :association of Asthma and COVID is controversial*
- *ACE2 is decreased, another entry molecules: TMPRSS2 increases in nasal & EC.*
- *Possible pathophysiology mechanism in allergy enhance susceptibility to COVID*
- *Virus: epithelial cell in airway : (up, lower) :inflammatory cascade :neutrophil recruitment- T cell- active monocyte/ IL25 ,IL33 in Ecell :Th2 :E, pro inflammatory cytokines (IL4- IL5 -IL13)- increase mucin production: symptoms of asthma*
- *In allergy :impair IFN in epithelial cell & M : respiratory viral infection*
- *so SARS-COV2 itself: exacerbate allergy, in turn facilitate viral infection*



# COVID-19 & Allergy

- *Non-allergic asthma : activation of **neutrophils** and mast cell , drive toward **Th1**,*
- *Because the immunologic profile of patient with **COVID: Th1 response**/ patient with non-allergic asthma: **aggravated Th1 response** , so sever **COVID***
- *Pediatric 611 but no ICU- death*



# COVID-19 & Allergy

- *COVID-19 has less impact on children and adolescence*
- *Children with wheeze constitute a significant proportion and the most chronic condition managed by pediatrician*
- *It has been suggested that asthma particularly when uncontrolled may be a risk factor for severe COVID*
- *COVID-19 in children: dry cough - fever ;mild*
- *COVID-19 in children with asthma: the most frequent symptom ; nasal discharge & blockage; & cough-fever is less often : 50% myalgia,*
- *73% mild, 20% moderate; no ICU*



# COVID-19 & Allergy

- *COVID-19 is not associated with severe asthma exacerbation like other viruses*
- *It's possible that; covid-19 does not induce bronchial hyperreactivity and asthma- like pathophysiology , although , in severe uncontrolled asthma : develop more severe COVID like influenza*
- *New patient restricted*
- *Use of spirometry restricted*
- *Nebulized treatment restricted*
- *- Social distance ,shelter, reduced school day( rhinovirus – triggers- allergen-pollution-exercise)*
  - Increase adherence to treatment ; ICS or po CS*
- *Specialist clinic messages on the need for treatment continuation ; no fear from ICS*
- *10% of children deteriorated : maybe; indoor allergen, psychosocial factors.*

# COVID-19 & Allergy

- *Coronavirus, URT& LRT infection before*
- *Prevalence of asthma in COVID-19 ?( real prevalence of asthma : 10% ; in USA : 7-17%, Chicago: 8 %)*
- *The symptoms of COVID-19 :cough , shortness of breath ,chest tightness are difficult to distinguish from severe asthma : for patients and doctors*
- *In this study in Chicago ,among 1542 COVID-19 ; 220 had asthma 14%  
40 -69 y-o, 53 % female*
- *But rate of obesity , hypertension , GER... was high compared to non asthma COVID*
- *D-dimer, CRP, LDH, ferritin lower but decrease ALC in ICU*

• *ChhibaKD. prevalence& chacterization of asthmain COVID19.J ALLERG CLINIC IMMUNOL. Aug 2020*

# COVID-19 & Allergy

- *No difference between hospitalization of asthma and non-asthma COVID (Bias)*
- *Rhinosinusitis and A. rhinitis : significant lower risk of hospitalization with COVID-19*
- *Relation between ICS and COVID-19 : proportion of not using ICS/LABA in outpatient COVID-19 : 57% & in ICU group 31 %: in general among patients with COVID-19 with asthma, the risk of hospitalization was not significantly different between using or not ICS/LABA*
- *Systemic CS using during last two weeks : no difference between outpatient and admitted patient*
- *One patient Omalizumab : ICU : discharged*
- *No difference between mortality rate*



- *The use of ICS did not increase the risk of COVID-19*
- *Jackson et al: allergic asthma have low ACE2( nose-bronchus)*
- *Peeter et al :reduction of ACE2& TMPRSS2 (transmembrane protease serine 2)*

# *COVID-19 & Allergy*

- *ChhibakD. prevalence& chacterization of asthmain COVID19.J ALLERG CLINIC IMMUNOL. Aug 2020*

*Ciclesonide: viral suppression of SARS-COV-2 : ICS : clinical protection*

*AR and rhinosinusitis :type-2 inflammation: reduced risk of hospitalization*



# COVID-19 & Allergy

- *Change in environment- medical practice -medication management -spend time - daily habit -travel pattern*
- *Changes by COVID-19 :Risk factor -Air quality- indoor environment- exercise – weight- medication*
- *Environment -medical practice - medication management*



# **COVID-19 & Allergy**

## **Environment**

- *Stay-at-home to control COVID-19 : impact many factor relate asthma control- morbidity & mortality*
- *School cancel :mean: no exposure to virus that exacerbate asthma*
- *Stay at home : limit physical activity,*
- *No yard, no Park( outdoor) ; or walking or bicycle*
- *Less car , less pollution (So2 - ozone - CO2 – PM)*
- *In 1996: Olympic Atlanta : lower traffic : decreased asthma to ER*
- *Remain indoor (smoke- indoor allergen ; mite ,cockroach, mold, mice)*

# COVID-19 & Allergy

## *Medical care*

- *largely moved to Telehealth - limited office visit*
- *Boston: sharp decrease asthma related emergency department*
- *Massachusetts : first week of May 1000, CRP positive COVID-19 , but decline pediatric asthma*

## *Medication Management*

- *Data : CS delay viral clearance, increase mortality of COVID-19??, doctors limit CS using( inhaler and systemic) in attacks, may decreased asthma control*
- *Children lower burden of COVID-19 , but risk of severe complication; multisystem inflammatory syndrome : is asthma a risk factor ?*







# COVID-19 & Allergy

- *The most frequent trigger of asthma in airway infection :weakly virulent virus such as rhinov. and RSV*
- *Epithelial cell and WBC of asthmatic : impaired production I of IFN primary or secondary to allergy :*
- *Such impairment of antiviral response suggests: asthma is high risk for COVID-19 ? but 8 studies : 17000 patients, comorbidity of asthma and COVID-19 were significantly lower than reported prevalence of asthma*
- *Regarding severity : DM –COPD were severe but asthma did not*

# COVID-19 & Allergy

- *2 Recp ACE2 & TMPRSS2*
- *In vitro treatment of epithelial cell of asthma with IFN : increase ACE2,*
- *IL-13 : reduces ACE2 , TMPRSS2*
- *Hypothesis: asthmatic patient are protected because decrease ACE2*





- *Lymphopenia in COVID-19*
- *Reduced E correlate positively with lymphocyte*
- *Eosinopenia in sever COVID : improved at discharge: maybe an indicator improving clinical status :*
- *Etiology ?BM?- apoptosis by IFN*
- *Eosinophil is protective or exacerbating?*



# *Allergy & COVID-19*

*1- ACE2 receptor inversely related to allergic sensitization*

*2- Eosinophil: 88% of patient had low E at admission and then returned to normal in follow-up: increase in E; may be an indicator of COVID-19 improvement*

*During the period of lower E count, PCR remained positive and after E nl : -ve during 5 days & CXR ay became normal*

*3- ICS : in vitro, CS inhibit human coronavirus replication*



# Allergy & COVID-19

- *106 patients hospitalized SARS pneumonia ; 23 asthma ( prevalence in the population 21.7%): chest tightness in asthma (OR 4.8),*
- *no difference: lymphopenia ,, lung parenchymal involvement*
- *63% controlled asthma, one patient allergic asthma, one patient CS and biologic,*
- *20% ICU (no difference between asthma and non asthma), no mortality in asthma*
- *in follow-up : 3 exacerbation*
- *Conclusion: no increase in exacerbation in asthma with COVID19*
- *85% of attack :viral (RSV- rhinoV- influenza -parainfluenza -coronavirus hmpv- bocavirus)*

*Grandbastien M.Sars-cov2 pneumonia in hospitalize asthmatic .J ALLERG CLIN IMMUNOL Vol8 2020*

# *Autoimmunity*



# *Autoimmunity & COVID-19*

## *1- Strong association between SARS-COV2 infection and autoimmunity*

- Subgroup of COVID: auto antibody*
- Hyper stimulation of immune system ;; fever, ferritin, cytokine storm*
- Lung macrophage has critical role in ARDS*
- Genetic predisposed patient :over activation of macrophages :*



# *Autoimmunity & COVID-19*

## *2- Autoantibody reported in COVID-19:*

- *Gazaruso et al: ANA 35.6% -*
- *lupus anticoagulant 11% of 45 patients*
- *Ro Ab*
- *ACLA 52 % + factor VIII elevation : coagulopathy*
- *Ab to IFN ( in vitro IFN block COVID-19 infection)*
- *Anti MDAS ( melatonin differentiated associated protein 5) is a recep for different RNA molecules & in amyopathic dermatomyositis*
- *Anti ACE 2 : ACE 2 is a recep for spike of corona , soluble ACE2 in blood , act as inactivator , complex of SARS-COV2 with serum ACE , lead to form Ab against ACE2*



# *Autoimmunity & COVID-19*

- *Different HLA are associated with Autol.*
- *Recently researches: genetic affect the SARS-COV-2: COVID-19 & genetic polymorphism*

# *Autoimmunity & COVID-19*

- *Common pathogenic viruses & Autoimmunity*

*: Such as : parvovirus B 19- EBV- CMV- herpes- HTLV- HA & B- Rubella & now : SARS-COV2*

- *Loss of smell like other autoimmune dis. Like : MS- MG- SLE*





# *Genetic & COVID-19*

- *Gene explain why some young pts die from COVID*
- *Science: 659 life threatening young pts: from 30 centers; they found:*
  - 1- inborn error of immunity in production of IFN (TLR3- IRF7)*
  - 2- Neutralizing auto antibody to type IFN , underlies life threatening COVID*



# *Genetic & COVID-19*

- *2244 critical ill COVID-19 - 208 UK ICU- young*
- *Novel genome; significant variation including antiviral restriction enzyme activator or OAS1, OAS2, OAS3 on chr 19p13.2 , near gene*

*IFNR2 ... loss of function; sever COVID-19 & other viruses*

*This suggest that administration of IFN : reduce critical COVID-19 ,When ? : early phase of disease , when viral load*

# Genetic & COVID-19

1- TYK2

2- DPP9 (dipeptidyl peptidase 9) | **GAIN OF FUNCTION**

3- CCR2( monocyte – macrophages chemotactic Rec)

- *TYK: inflammation of lung (Jack – STAT pathway)  
(baricitinib : inhibitor of Jack : reduce infla. of lung)(Europe)*
- *CCR2 : infilt. of monocyte / macrophage to lung : inflammation  
( monoclonal Ab of CCR2 in RA)*

***COVID-19  
&  
Inborn Error of  
Immunity***



# *Immune Deficiency & COVID-19*

- *COVID; case fatality 1-20%*
- *Each decade of life beyond age of 50 y-o :increase lethality ,*
- *male>female*



# *Immune Deficiency & COVID-19*

- *94 ID pts with COVID-19 +ve ; (25 -34 ) y-o median age*
- *56% P.ab deficiency*
- *9.6% immune dysregulation-*
- *6.4% phagocytic defect-*
- *25 pt asymptomatic-*
- *28 pts : admission-*
- *13 pts: needO2 –*
- *18 pts : ICU-*
- *7.4% AutInflam. disorder-*
- *15% CID-*
- *3% innate I-*
- *2% BM failure*
- *12 invasive ventilation-*
- *3 ext corporeal mem oxygenation-*
- *Death 9 (7 adult- 2 children)*

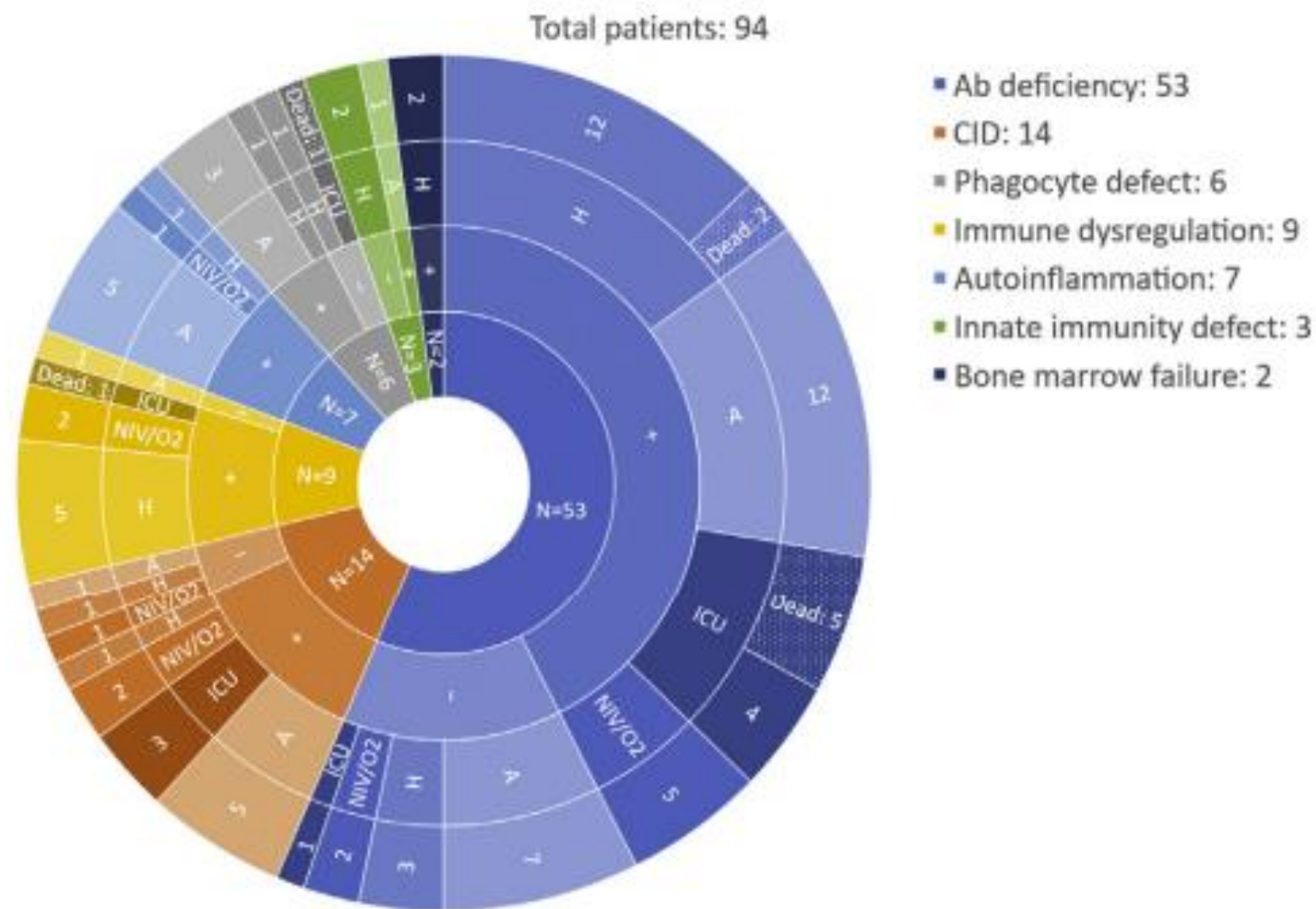


FIG. 1. Distribution of patients based on ICI status, comorbidity and

# *Immune Deficiency & COVID-19*

- *In this study: male /female 1.8;*
- *32 pts < 18 y-o. median age 25-34 y-o*
- *24 pts , mild, outpat.: age 3 to >75 y-o (Ab def.- SCID- CID- HIgE)*
- *Old age: Ab deficiency- CGD- CVID*
- *Invasive ventilation : preexisting comorbidity (heart- Down syn- lung...)*
- *Complication: bacterial pneumonia 6 - HLH 6 – MISC 1 - Kidney failure 3 – sepsis & Htn 2 - anemia- neutropenia- myocarditis*
- *Death ... 4 male*
  - CGD Borkheld. – GVHD, HSCT- sepsis &Htn - pneumothorax- pul Htn*
- *Adult death : comorbidity ( kidney- lung – malignancy- transplant)*



# *Immune Deficiency & COVID-19*

## *Treatment*

- *Ig- HQ- CS- mAb- anakinra 1 – favipiravir 1- enoxaparin 12- remdesivir 9- lopinavir & ritonavir 13*
- *Plasma 5 pts: 4 survive -*
- *Tocilizumab 6 pts, 1 death*
- *Remdesivir 9 pts: all survive, 5 ICU –*
- *HQ 31, 5 death*

# *Immune Deficiency & COVID-19*

- *All respiratory viruses can spread from person to person*
- *PID patients should protect themselves*
- *IVIG protect against wide range of infections, but does not guarantee against COVID-19*
- *PID with higher risk of sever course, continue working from home*
- *no school*
- *If not well : test*

# *Immune Deficiency & COVID-19*

- *Hygiene and social distance: key protect PID*
- *...>30% of patients with inborn errors of immunity: mild COVID-19 and may be a risk factor for severe disease in more younger pts*
- *PID with organ transplant , HSCT, cancer , immuno-modulatory drugs (autoInfla) CS may mask sign of infection (.. Fever)*



# *Immune Deficiency & COVID-19*

- *IFN impairment May be with sever COVID-19*
  - *neutralized antibody to IFN*
  - *genetic variation in IFN*
- *APECED: auto antibody to type one IFN; more severe form COVID-19; ICU*
- *Influenza vaccine : most PID and family members*

# *Immune Deficiency & COVID-19*

- *China report : corona positive in blood donor*

*; but IVIG : no risk of transmission of COVID-19 ( removal step)*

- *PID conditions does **not require** IVIG, no need to start IVIG: no antibody to COVID-19 in existing preparation*

# *Immune Deficiency & COVID-19*

- *Prior considered: ID : at risk population*
- *Case fatality ( nl Population 1-20%), in ID : 10% ; even is lower, because some death was due to ID no COVID*
- *Younger male : more sever course of COVID-19 (X –linked Disorder)*



# *Immune Deficiency & COVID-19*

- *Some adaptive ID : B cell – T cell (zap 70): asymptomatic or mild disease:*
  - 1- Certain compound of I system : not essential for controlling COVID-19*
  - 2- Four death of CVID ; 45% of death : were old & comorbidity*
  - 3- CGD s: mild disease : modest function of N in COVID immunity*
  - 4- STAT disorder : mild disease: STAT induces cytokine storm : STAT & IL6 induce inflammatory response & effect on Px*
  - 5- All autoinflammatory D. : asymptomatic or mild : ( colchicine - IL1 blocker)*

# *Immune Deficiency & COVID-19*

- *In a study on 987 pts. : 10% Auto Ab to IFN : 94% male  
650 pts : whole genome sequencing (like influenza)*
- *milder disease in some IFN pathy : sufficient residual to protect COVID-  
IFN produce by NFkB pathway too*
- *CVID may have autoAb to IFN: less frequent & less sever than nl population(  
social prevention)*

*Conclusion: subgroup of pts : milder course, risk factor like nl population*

*Younger age: more sever course, ICU*

# *Immune Deficiency & COVID-19*



# *Immune Deficiency & COVID-19*

- *ACE2 is receptor + TMPRSS2 (transmembrane serine protease) for host CELL entry : after entry endosome: lysosome*
- *Poor cellular I- CID – Innate I system defect (IFN )*
- *Primary ID: 1/85000 to 1/100000 ; 400 heterogeneous defects*
- *Genetic predisposition; IFN receptor defect*
- *IUIS(international union of immunologic society)*
- *9 category*
  - syndromic CID - predominant antibody deficiency --immune dysregulation –*
  - phagocytic disorder - auto inflammatory- innate immune defect- phenocopy of inborn error of immunity*
- *4518 registered patient; 2554 alive ;age mean 108 mo-o*
- *The patient visit every month ; COVID: if cough, fever, dyspnea*
- *PCR +ve in 19 patient : median age 109 month : 1/144 incidents, gp 1/178 (1.23 fold more)*



# *Immune Deficiency & COVID-19*

- *Underestimated( tight isolation)*
- *source unknown : 84% ----family member 16%*
- *CID 47%- humoral ID 4%- phagocytic 2% -- immune dysregulation 2% --*
- *---autoinflammation 1%*
- *no in innate (117 )and in complement(85) patients*
- *Hx of LRTI in 89%*
- *Sever distress requiring resp support: 10 patients , 7 lymphoproliferation(LAP-HSM)*
- *Bronchiectasis 21% - CVS (10.5%)- liver failure 10%*
- *CRP –ve in 8 patients*
- *Imaging: mild broncho vascular marking- peribronchial thickening- patchy opacities- collapse consolidation- mosaic ground glass*
- *8 pts deceased(42%): 10 fold higher*
- *2 pts were sever( SCID - familial H. Lymphohistiocytosis)*

*Delavari S.impact of SARS cov-2pandemic patient with ID. J CLIN IMMUNOL 2020*

# *Immune Deficiency & COVID-19*

- *Previous hypothesis : COVID19 is more life threatening in T cell & immune dysregulation: 150 fold higher risk*
- *In comparison report : Italy : Ab deficiency reported*
- *In a report Europe 582 PCR +ve in children : 0.5% : ID (COVID- neutropenia)*
- *Parrie et al: ER COVID :7.5-12.5% ID*
- *Report" decrease T cell (CD8 cytotoxic T cell): sever COVID*
- *Lymphopenia in COVID is like PID pts*
- *Another multinational cohort 94 pt ,37% mild symptom or no symptom – 9.5% mortality, 14 pts : CID & older : all recovered(optimal Tx :HSCT)*







# *Immune Deficiency & COVID-19*

- *a case was reported for Mashhad : CVID and bronchiectasis and cough (wet) crackle , mild tachypnea, retraction : antibiotic 7-days; meropenem-clindamycin –hydroxychloroquine; 6-month follow-up: no any problem*
- *3000 immune deficiency patients in Iran*
- *multicenter international report : 94 patient primary immune deficiency; 56% antibody deficiency (CVID) : 4 death (45-54 ) y-o*
- *combined immune deficiency are at higher risk for morbidity and mortality in viral infection*
- *the most death ; due to COVID-19 : immature activation and cytokine storm*
- *the report immune deficiency patient, > 50% antibiotic -33% hydroxychloroquine -10% IVIG*



A







# *Immune Deficiency & COVID-19*

- *Reports of primary ID : Italy ; 7 cases of Ab deficiency; one boy with specific Ab deficiency, two cases of XLA*
- *PID ,negatively correlate with severity of acute respiratory syndrome*

# *Immune Deficiency & COVID-19*

- *Agammaglobulinemia : lack of B cell: milder course of disease ; no need ICU*
- *T-cell response is more important in immunity against virus*
- *Little is known about exact pathogenesis of the virus ,but hyper inflammation and cytokine storm aggravate the clinical course*
- *PIDs are surprisingly less likely severe phase( immuno response defect)*



# *Immune Deficiency & COVID-19*

- *IVIg ; monthly to ID is a treatment in COVID-19 too.*
- *Possibility of pool of Ig, antibodies with ability of Cross Rx with COVID protein and immunomodulation on monocytes and macrophages that have control in role in cytokine Storm*
- *Unclear; PID is protective or disposing*



- *7 patient with primary immune deficiency :*
  - 5 CVID-*
  - 2 Agammaglobulinemia (no B cell- benign course)*
- *CVID dysfunctional B-cell; need multidrug ; IL-6 ; antiviral ; more aggressive course*
- *no difference in IgG of both groups on the time of diagnosis*
- *HRCT of CVID: ground-glass and alveolar infiltration*
- *HRCT in XLA was similar to 1-year before*

- *Primary immune deficiency who no Ab producing : no serology*
- *But others even with IVIG: serology could be done, especially IgM could be done because IVIG: no COVID-19 antibody and no IgM as a whole*
- *Recommend: second screen should be done from the patient, clinically recovered( combined immune deficiency) - no clear infection and remain positive longer (source of infection)*
- *On the other hand, no sufficient memory response: so recurrent infection*

*IUIS . Joint statement on the current coronavirus pandemic update. Nov 2020*



# *shedding of viable SARS COV-2 after immunosuppressive treatment for cancer*

- *20 patient : 18 HSCT & 2 lymphoma*
- *Nasal swap ;viral culture c*
- *COVID-19 by PCR ..11 sever*
- *78 samples viral RNA was detected up to 78 days (24 or 64 days )*
- *Viral culture in 5 patient till 61 days*
- *Viral shedding 2 months*

- *88 patients with immunodeficiency diseases were asked about symptoms of covid-19.*
- *Symptom of 9 patients was fever (3 Ataxia telangiectasia, 3 SCID, 1 Disseminated wart(CID) ,1 mucocutaneous candidiasis,1 B cell - Ab<sup>+</sup>),*
- *3 of them was cough (2 Ataxia telangiectasia, 1 Bruton) a*
- *4 of them was GI symptoms (1 Ataxia telangiectasia, 1 Bruton, 2 Disseminated wart).*
- *Two : Ataxia telangiectasia had chill,*
- *one : Bruton had anosmia and PCR+ve*
- *Another Bruton fever PCR –ve, admission ,CT +ve*
- *One :disseminated wart had sore throat.*
- *Expired: one AT, female, 16 y-o :Ca & chemotherapy, one SCID, new case, 2 mo after admission PCR*

- *28-year- old male subject, known case of Bruton's disease since his infancy*
- *In the summer, developed generalized muscle and body pain, sore eyes, fatigue, new loss of smell*
- *PCR of COVID19 was positive and the symptoms subsided a few days later without any hospital admissions or IV medical giving. The patient was relatively well until 21 days after the first time, he became symptomatic with high grade fever, generalized muscle and body pain much worse than the first time and non-productive cough .*
- *Spiral chest CT-scan showed lower lobe involvement of both sides: Azitromycine 500mg tablets, Naproxen 500mg tablets, Amp of Dexamethasone for IM*
- *Was suspected to have re-activation or re-infection of the COVID19*
- *Another positive result of PCR*

Hematology		Sample No : 4525			
	Result	Unit	Normal	Test	Result
CBC Diff					
W.B.C	H13.3	10^3/ul	4.5-11	Neutrophils	74
R.B.C	5.81	10^6 /ul	4.5-6.5	Lymphocyte	24
Hb	14.5	g/dL	13.5-17.5	Monocyte	1
HCT	46.4	%	40-54	Eosinophil	1
M.C.V	L79.9	fL	81-98		
M.C.H	L25.0	pg	27-32		
M.C.H.C	L31.3	g/dL	32-36		
R.D.W	12.3	%	11.5-14.5		
Platelets	156	10^3/ul	150-450		
E.S.R	2	m.m/h	0-15		
H : HIGH. L : LOW.					
Reported by : احمدي سعدي قاسم			Digitally Signed By : Dehghanian.MD Amirreza		
Immunology		Sample No : 1783			
	Result	Unit	Normal	Test	Result
» IgG	8.61	g/l	7-16		
»* IgM	L0.12	g/lit	0.4-2.3		
* : Rechecked. L : LOW.					
Reported by : طالبي مريم			Digitally Signed By :		
Serology		Sample No : 1697			
	Result	Unit	Normal	Test	Result
CRP	9	mg/l	Up to 6		
Reported by : باقرزاده پروين			Digitally Signed By : Dehghanian.MD Amirreza		



- *Another case of Bruton , 23y-o, with Poliomyelitis after OPV ,one mo ago*
- *Was on IVIG , fever one week, CT of lung : ground glass*
- *PCR –ve*
- *Discharged ( remdesivir- IVIG- prednisolone-antibiotic)*

*Good luck  
,Thank you*

