



COVID-19

&

Coeur

Science ou fiction?

Joëlle Morin, MD, FRCPC

Cardiologue-Intensiviste | Secteur insuffisance et greffe - IUCPQ

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Je n'ai pas
de
conflit d'intérêt

01 02 03

3



Comprendre les interactions entre le COVID-19 et le système Rénine-Angiotensine-Aldostérone



Nommer les facteurs de risques CV augmentant le risque d'atteinte grave lors d'une infection à COVID-19.




Connaître les complications cardiaques possibles du COVID-19 et les particularités de leur prise en charge

Introduction



4

- Une **pandémie mondiale de COVID-19** fait rage depuis plus d'un an: plus de 2.4 millions de décès y sont attribuables à travers le monde dans la dernière année.
- Les maladies cardiovasculaires et leurs facteurs de risque confèrent aux patients un risque **accru de développer une forme sévère de COVID-19**.
- **Plusieurs atteintes du système cardiovasculaire** ont été décrites en lien avec le COVID-19.

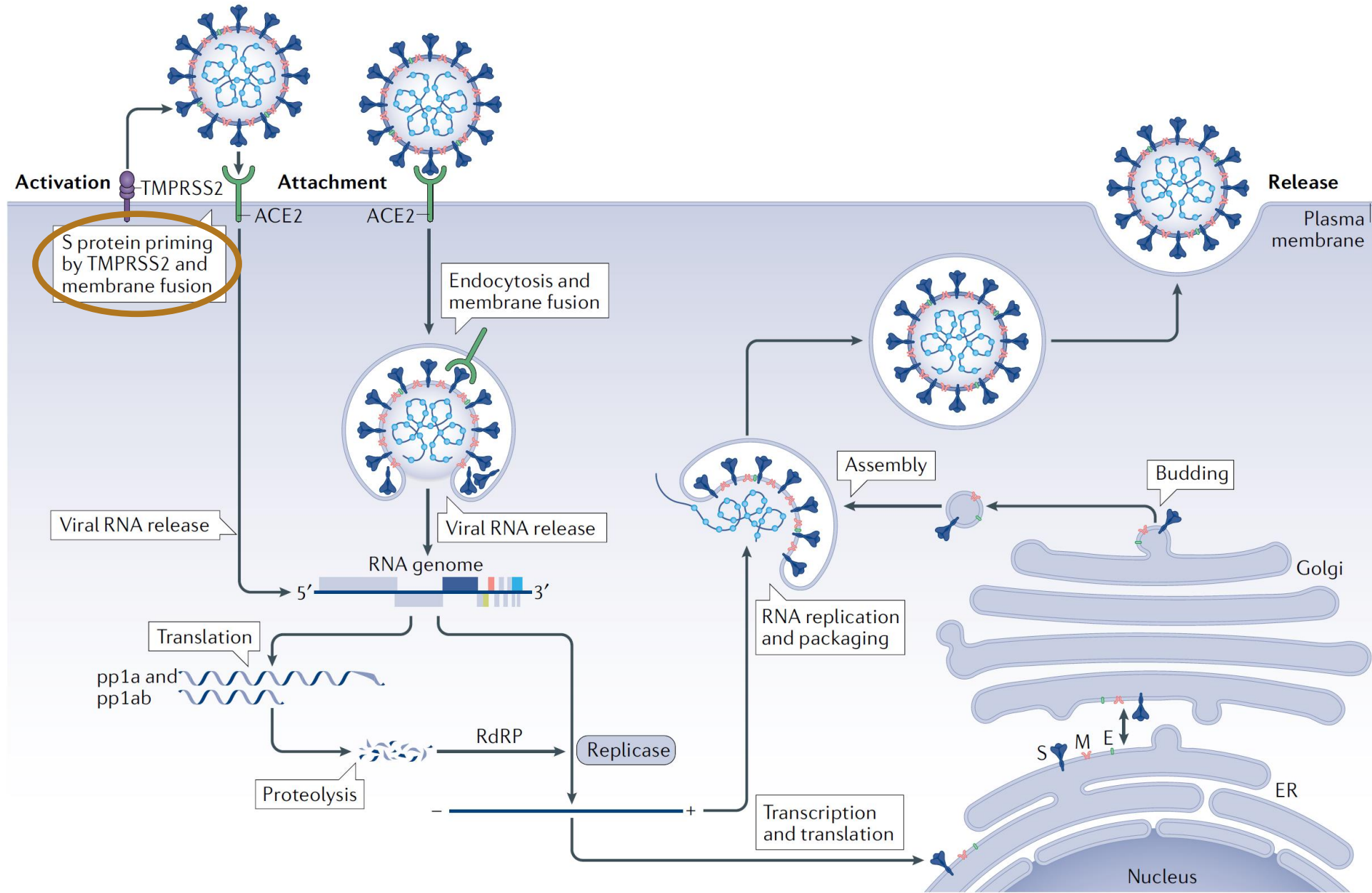
The background features a stylized illustration of a human heart in shades of purple and pink, positioned centrally. Surrounding the heart are several yellow, spherical virus particles with prominent spikes, characteristic of the COVID-19 virus. The overall color palette is warm, with various shades of pink, purple, and yellow.

Interaction avec le Système

Rénine-Angiotensine-Aldostérone
(SRAA)

COVID-19

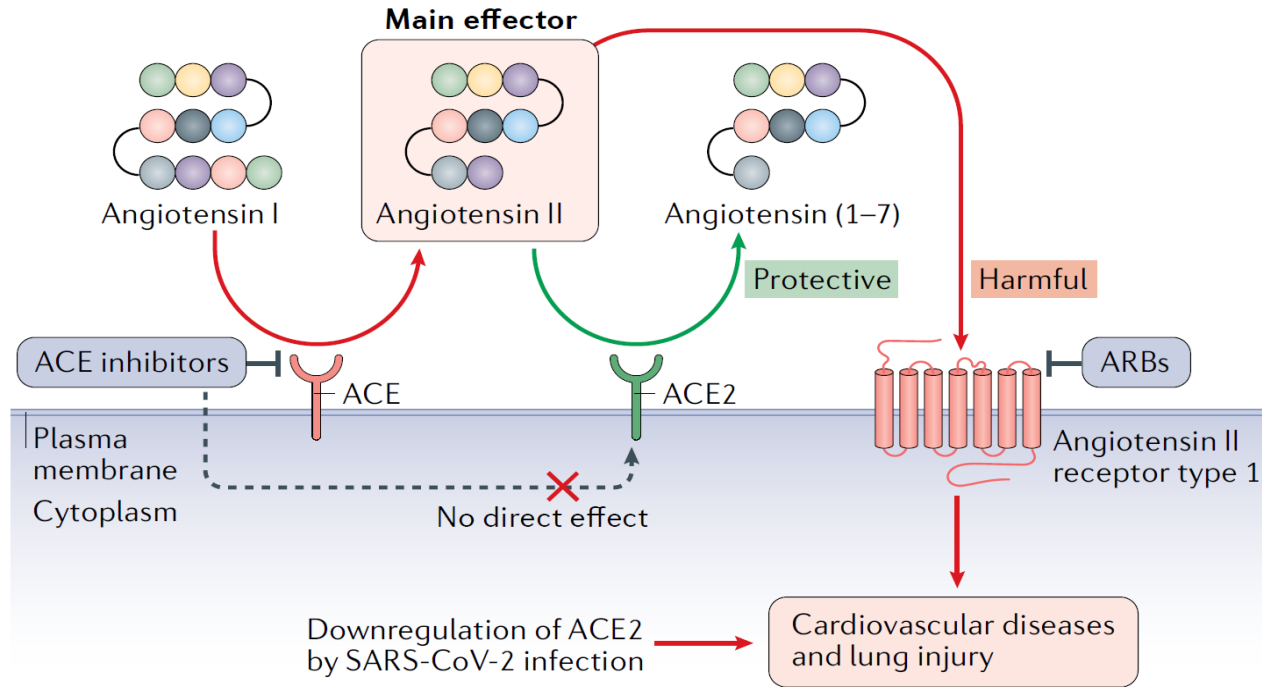
Cycle de répliation



Épithélium
respiratoire:
haute expression de
ACE2 et TMPRSS2

Enzyme de Conversion de l'Angiotensine 2 (ACE2)

COVID19 & Cœur | Dre Joëlle Morin



Angiotensine 2

- o Principale molécule effectrice du SRAA
- o Implication dans le développement de l'HTA, du DB, de l'insuffisance cardiaque, ...
- o Inactivée (transformée en angiotensine) par l'ACE2.

Enzyme de Conversion de l'Angiotensine 2 (ACE2)

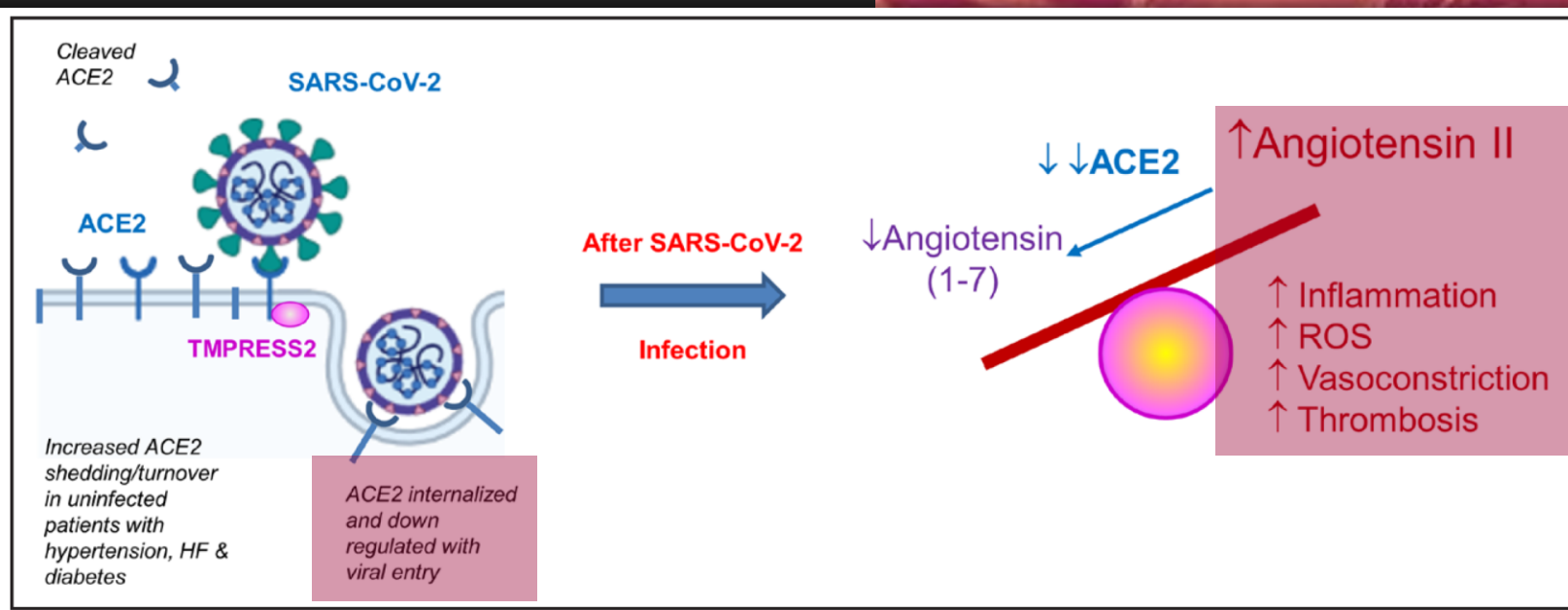


Figure 2. SARS-CoV-2 uses the ACE2 internalization receptor, facilitated by TMPRSS2 protease.

Enzyme de Conversion de l'Angiotensine 2 (ACE2)

Organotropisme:

- Séries autopsiques démontrant virus ou ARN viral a/n cœur, foie, reins, poumons, cerveau, intestin
- Symptômes variés du COVID-19:
2aire infection directe ou à la réponse inflammatoire

Table 2. Distribution of ACE2 and TMPRSS2 in Organs and Symptoms of COVID-19

ACE2/TMPRSS2 Distribution	Symptoms/Laboratory Findings
Lymphocytes/dendritic cells	Fever (>99%), fatigue (70%), myalgia, lymphopenia
Lung (type 2 pneumocytes, bronchial epithelium)	Dyspnea (31%), dry cough (60%), respiratory failure
Gastrointestinal smooth muscle	Nausea (30%), diarrhea
Myocardium	Myocarditis, heart failure, arrhythmias
Vasculature (smooth muscle)	Vasculitis, thrombosis, microangiopathy
Neurons	Anosmia, hypogeusia, encephalopathy, seizures, myopathy
Liver	Abnormal liver function
Kidney	Renal dysfunction

Antagonistes du SRAA & COVID-19

Les évidences cliniques

COVID19 & Cœur | Dre Joëlle Morin

De grandes séries de cas et méta-analyses

ont démontré que les IECA et BRA n'étaient pas associés avec une susceptibilité accrue de contracter le COVID-19, ou de souffrir d'une forme sévère/fatale.

- Italie (n = 6772 patients)
- Espagne (n = 1139 patients)
- USA (n = 5894 patients)
- (n = 1735 patients)

A

All-cause mortality

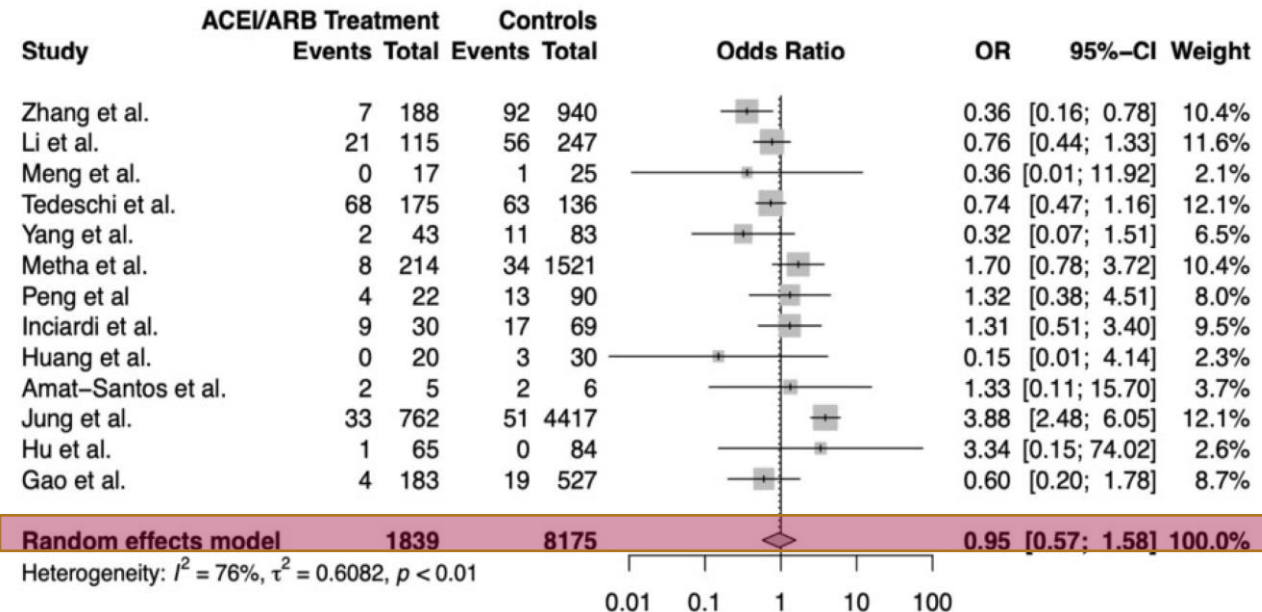
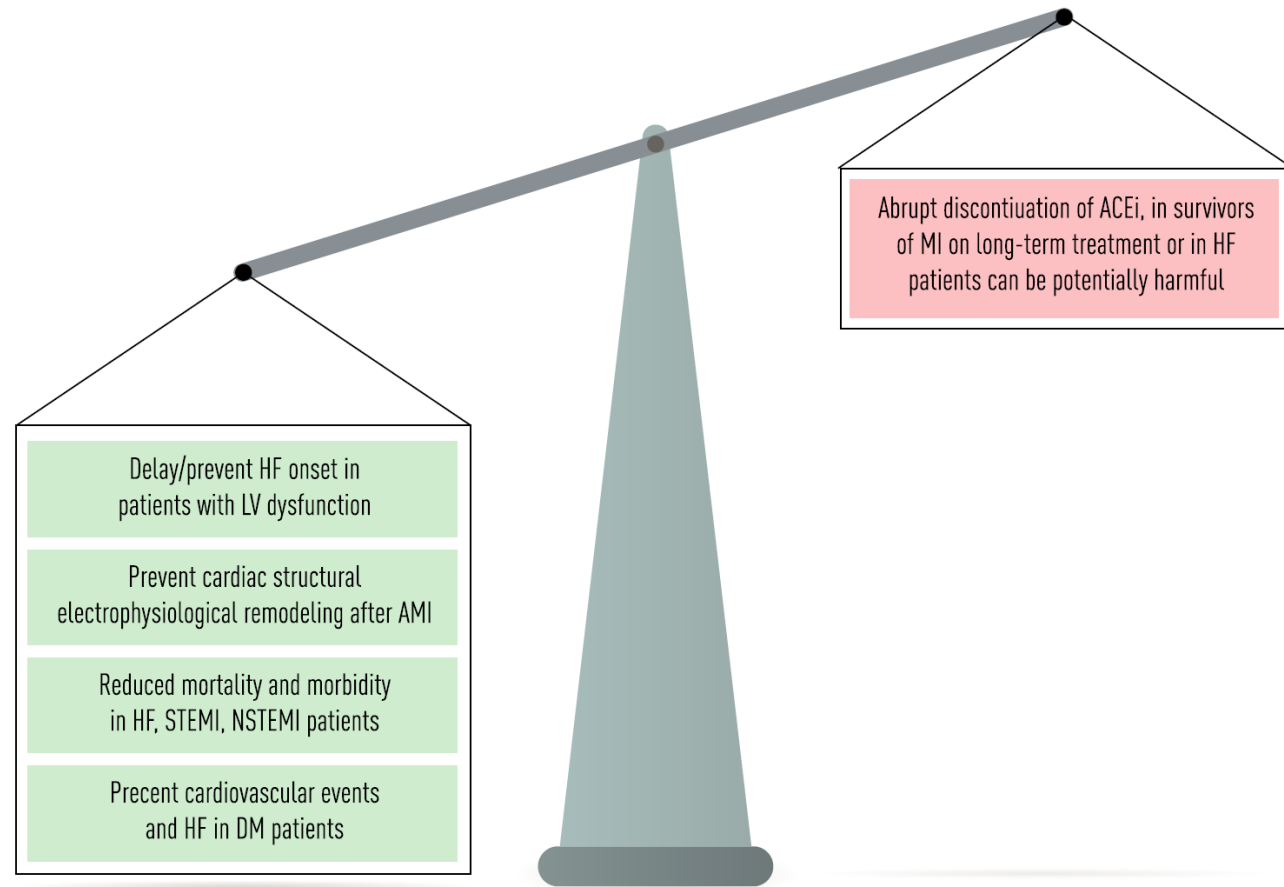
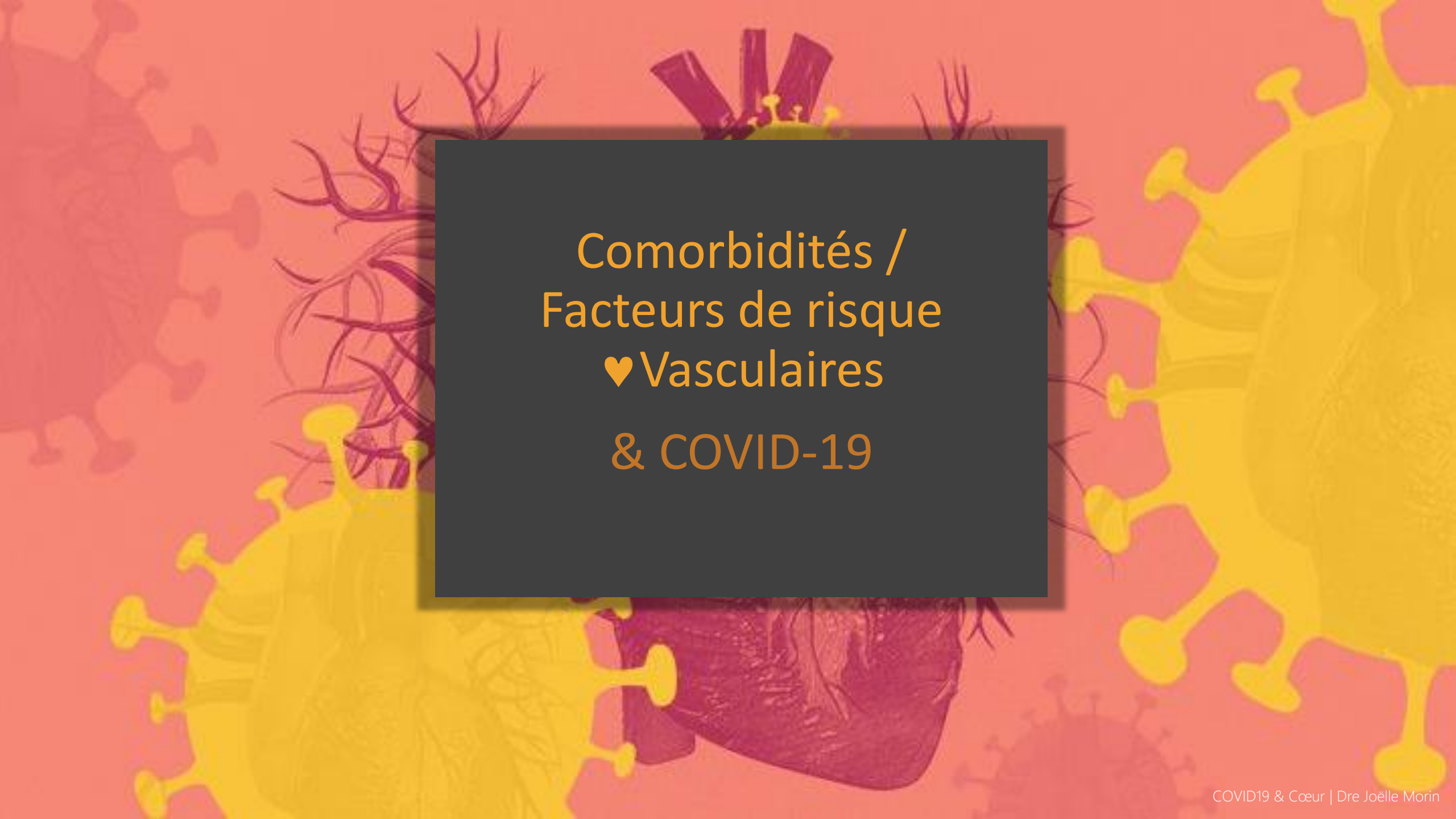


Figure 2 ACEIs/ARBs effects in cardiovascular disease. Abbreviations: heart failure (HF); left ventricular systolic dysfunction (LVSD); acute myocardial infarction (AMI); ST-segment elevation myocardial infarction (STEMI); non-ST-segment elevation myocardial infarction (NSTEMI); diabetes mellitus (DM); ACE inhibitors (ACEIs), myocardial infarction (MI).



Antagonistes du Système RAA

L'arrêt indiscriminé des IECA ou BRA
chez un patient avec indication pour ces molécules
a un potentiel néfaste



Comorbidités /
Facteurs de risque
♥ Vasculaires
& COVID-19

Table 1 | Prevalence of cardiovascular comorbidities in patients with COVID-19

Country	Number of patients	Prevalence of comorbidity among all patients (among patients who were ventilated or in ICU)				Ref.
		Cardiovascular disease (%)	Hypertension (%)	Diabetes (%)	Obesity (%)	
China	41	15 (23)	15 (15)	20 (8)	NR	17
China	138	14.5 (25.0)	31.2 (58.3)	10.1 (22.2)	NR	18
China	191	8 ^a (24) ^{a,b}	30 (48) ^b	19 (31) ^b	NR	19
China	150	8.7 (19.1) ^b	34.7 (42.6) ^b	16.7 (17.6) ^b	NR	22
China	1,099	2.5 ^a (5.8) ^a	15.0 (23.7)	7.4 (16.2)	NR	20
China	44,672	4.2 (22.7)	12.8 (39.7)	5.3 (19.7)	NR	21
Italy	1,591	NR (21)	NR (49)	NR (17)	NR	46
USA	393	13.7 ^a (19.2) ^a	50.1 (53.8)	25.2 (27.7)	35.8 (43.4)	47
USA	5,700	11.1 ^a (NR)	56.6 (NR)	33.8 (NR)	41.7 (NR)	51

Comorbidités cardiovasculaires et COVID-19: Prévalence

Définition variable selon les études.

Comorbidités cardiovasculaires et COVID-19 SÉVÈRE

14

Âge avancé

Obésité

Mx
Cérébrovasculaire

HTA

Mx
Cardiovasculaire

Sexe masculin

Diabète

Revue systématique narrative
de 28 études

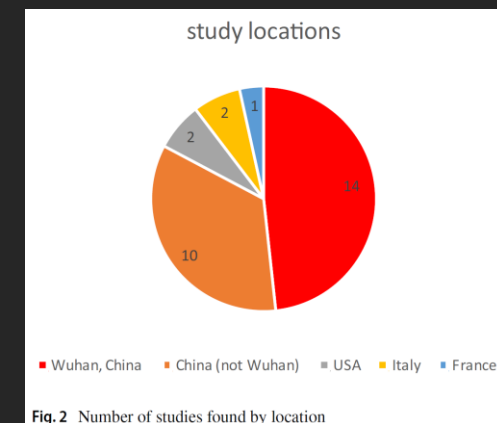


Table 1. Death Rate to Date of Patients With COVID-19 Infection and Specific Preexisting Conditions (World Health Organization Data)

Preexisting Condition	Death Rate, %
Cardiovascular disease	10.5
Diabetes mellitus	7.3
Chronic respiratory disease	6.3
Hypertension	6.0
Cancer	5.6
No preexisting conditions	0.9

COVID-19 indicates coronavirus disease 2019.

Table 3. Mortality in Confirmed Cases, or in All Cases, by Sex Distribution in Patients With COVID-19

Sex	Death Rate, %	
	Confirmed Cases	All Cases
Male	4.7	2.8
Female	2.8	1.7

COVID-19 indicates coronavirus disease 2019.

Comorbidités
cardiovasculaires
et COVID-19 mortel

Diabète et COVID-19

Étude de cohorte rétrospective (Chine)

500 patients non-diabétiques

84 patients diabétiques

57 patients « malade critiquement »

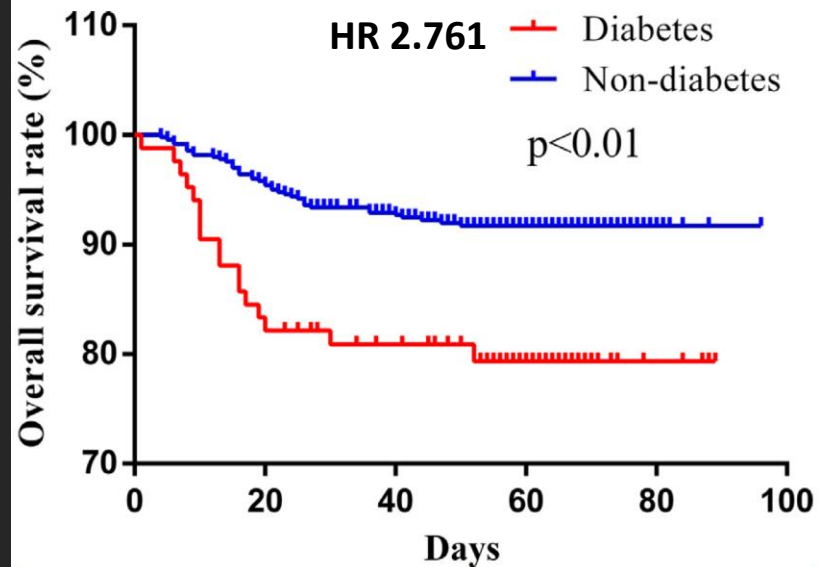


Figure 2 Kaplan-Meier survival curve of the patients with and without diabetes.

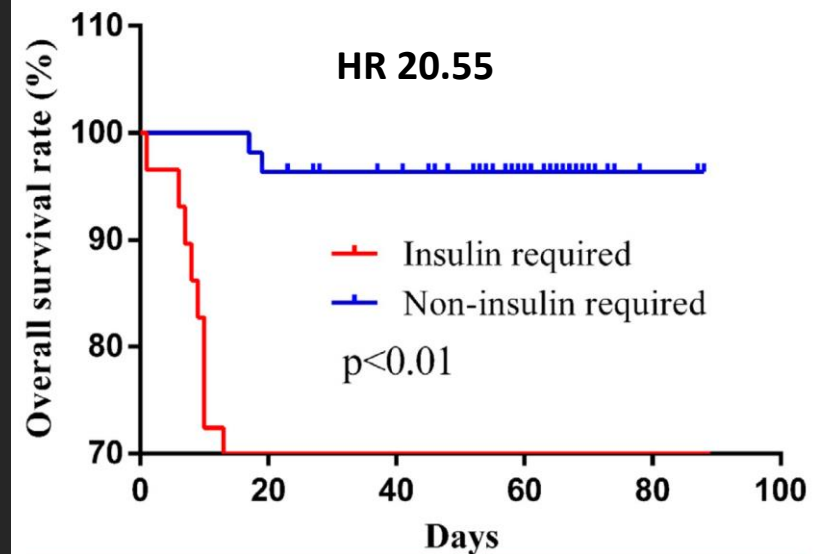



Figure 3 Kaplan-Meier survival curve of diabetic patients who needed and did not need insulin.



Complications Cardiaques du COVID-19

DOMMAGES
MYOCARDIQUES

INSUFFISANCE
CARDIAQUE

MYOCARDITE

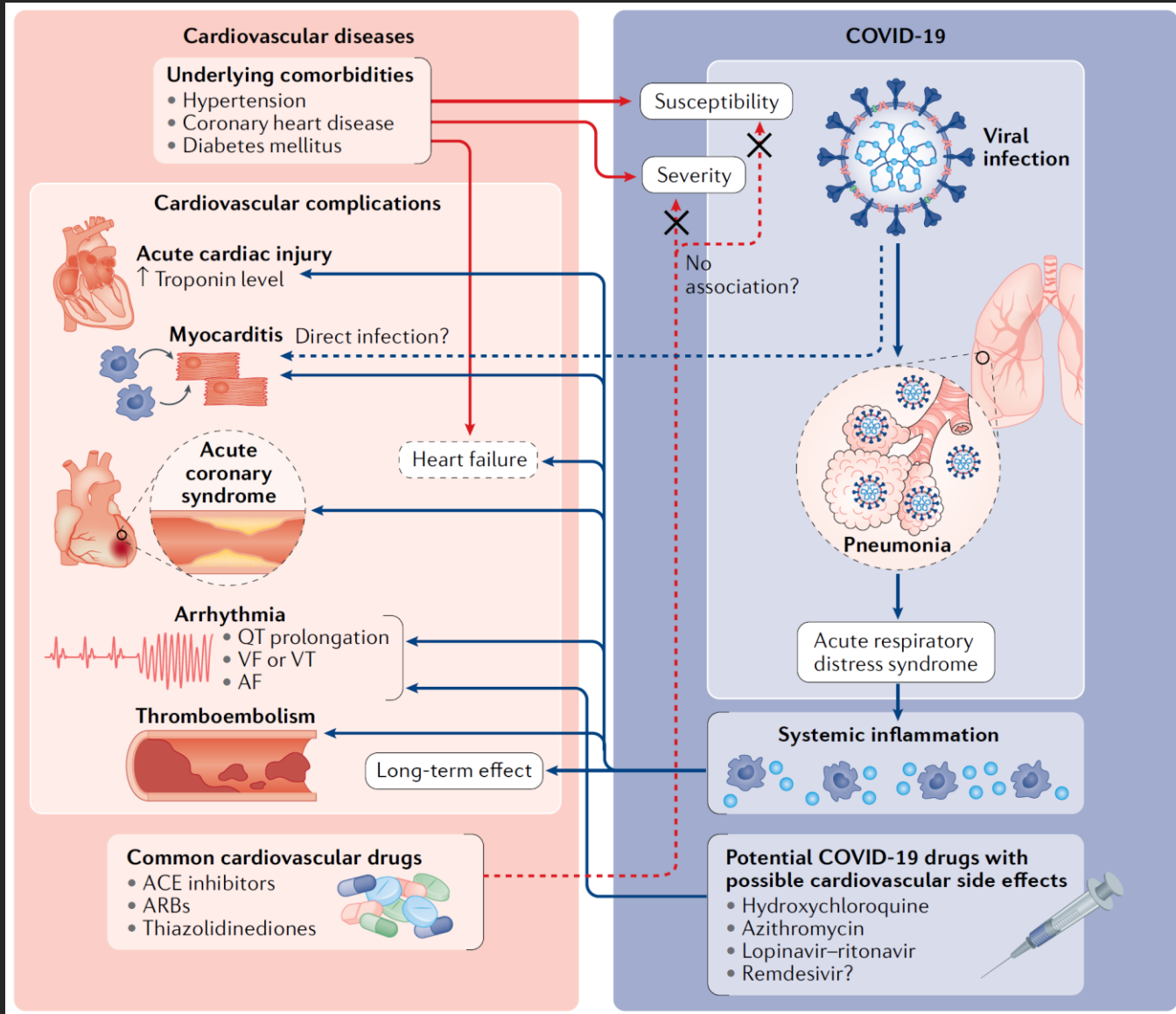
ARYTHMIES

SYNDROME
CORONARIEN
AIGU

SYNDROME
INFLAMMATOIRE
MULTISYSTÈME
DE L'ADULTE

Complications cardiovasculaires

Complications Cardiaques Pathophysiologie



Interactions bi-directionnelles

Mécanismes proposés:

- Infection cardiaque directe (?)
- Réponse inflammatoire systémique disproportionnée
- Altération balance apport O₂/demande métabolique
- Rupture de plaque
- Thrombose artérielle ou veineuse
- Désordres électrolytiques
- Effets 2aires de Rx potentiels
- Dysfonction endothéliale

Complications Cardiaques Timing

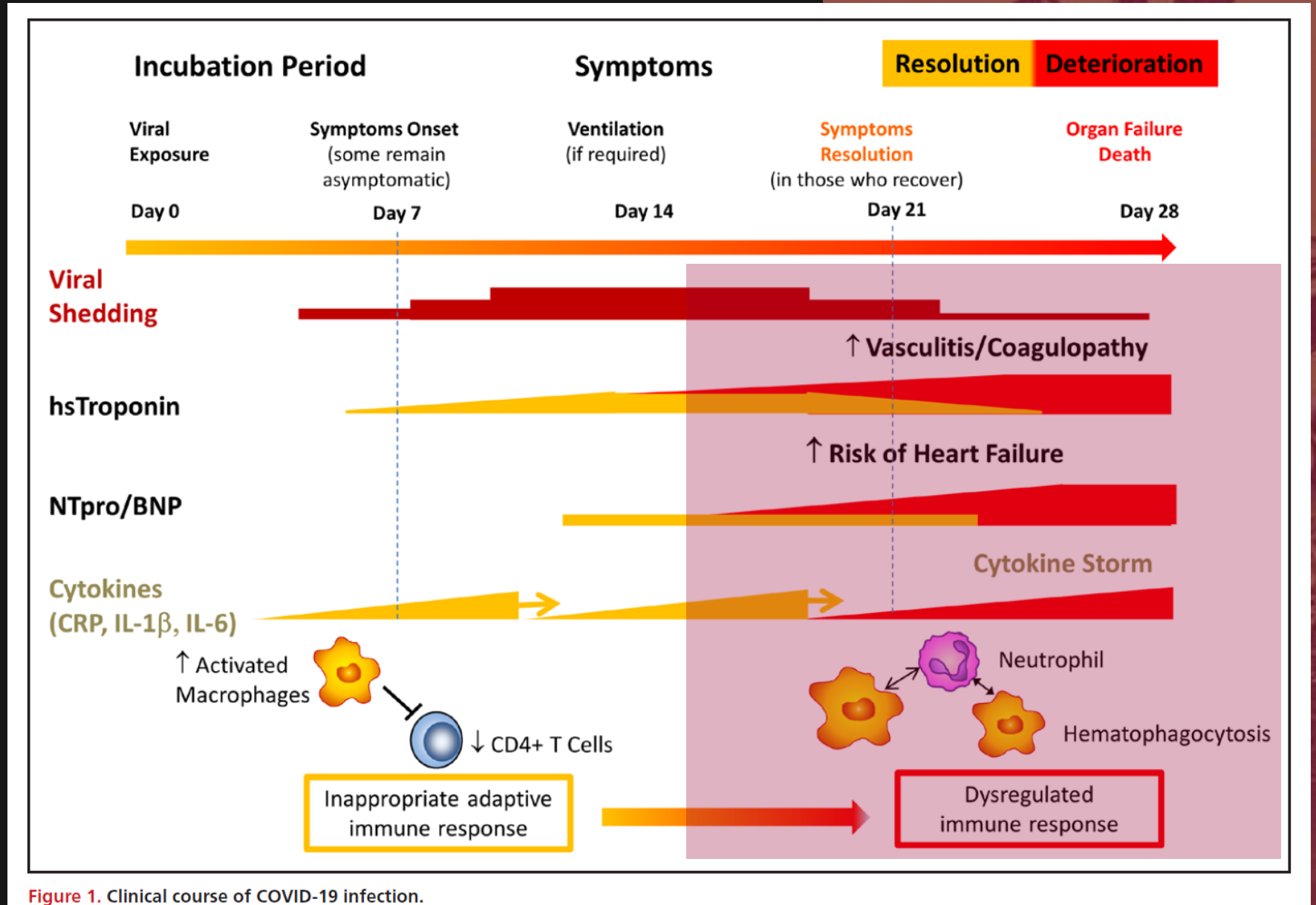
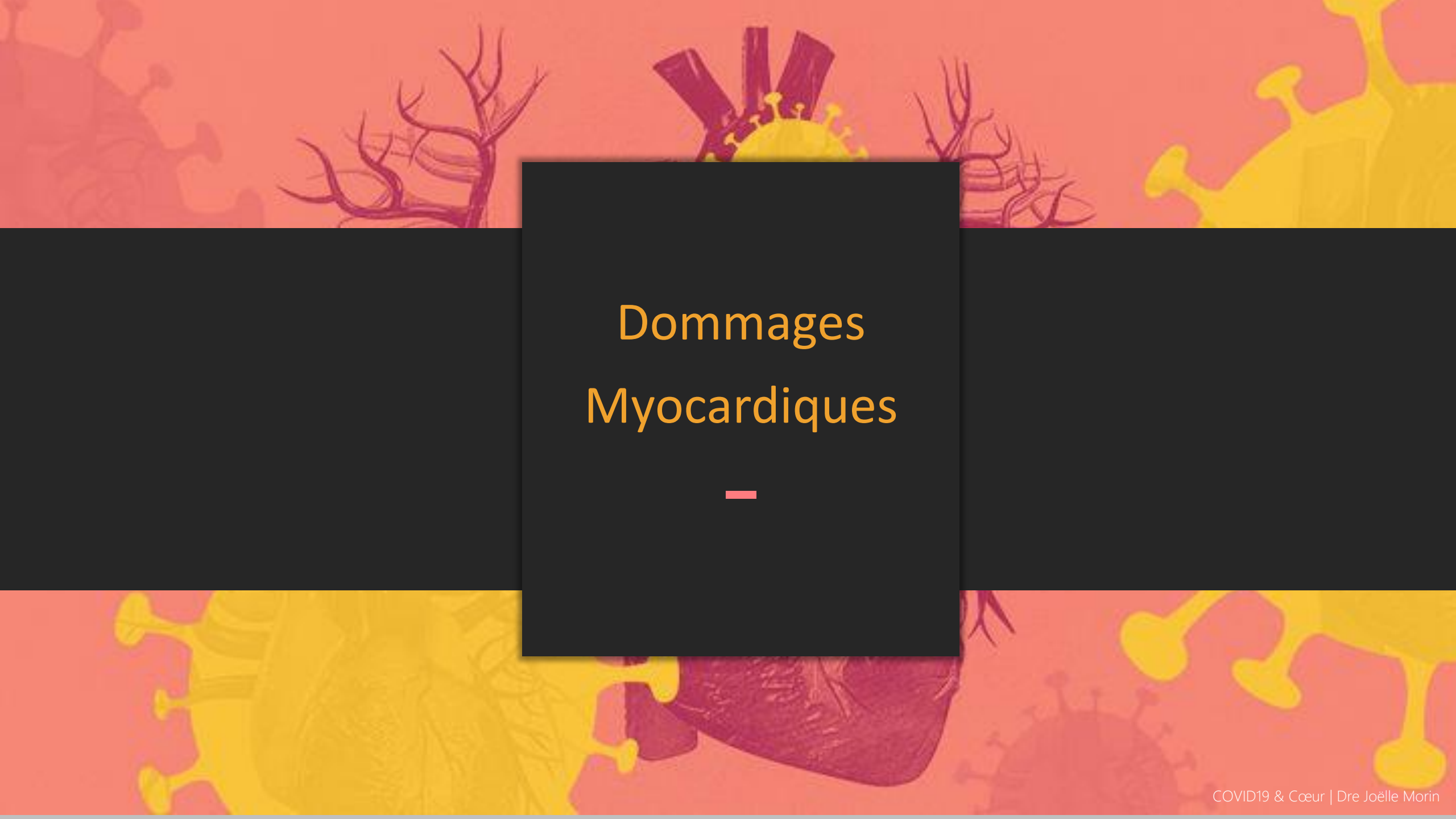


Figure 1. Clinical course of COVID-19 infection.



Dommmages Myocardiques

—

Dommmages Myocardiques



DÉFINITION

Variable selon les études



LE PLUS SOUVENT UTILISÉ

Troponine > 99e percentile de la limite supérieure de la normale



MARQUEUR DE MAUVAIS PRONOSTIC

Consistent à travers les études



Fourth Universal Definition of Myocardial Infarction (2018)

2. UNIVERSAL DEFINITIONS OF MYOCARDIAL INJURY AND MYOCARDIAL INFARCTION: SUMMARY

Universal definitions of myocardial injury and myocardial infarction

Criteria for myocardial injury

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values.

Criteria for acute myocardial infarction (types 1, 2 and 3 MI)

The term acute myocardial infarction should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischaemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and at least one of the following:

- Symptoms of myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Identification of a coronary thrombus by angiography or autopsy (not for type 2 or 3 MIs).

Post-mortem demonstration of acute athero-thrombosis in the artery supplying the infarcted myocardium meets criteria for *type 1 MI*.

Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute athero-thrombosis meets criteria for *type 2 MI*.

Cardiac death in patients with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes before cTn values become available or abnormal meets criteria for *type 3 MI*.

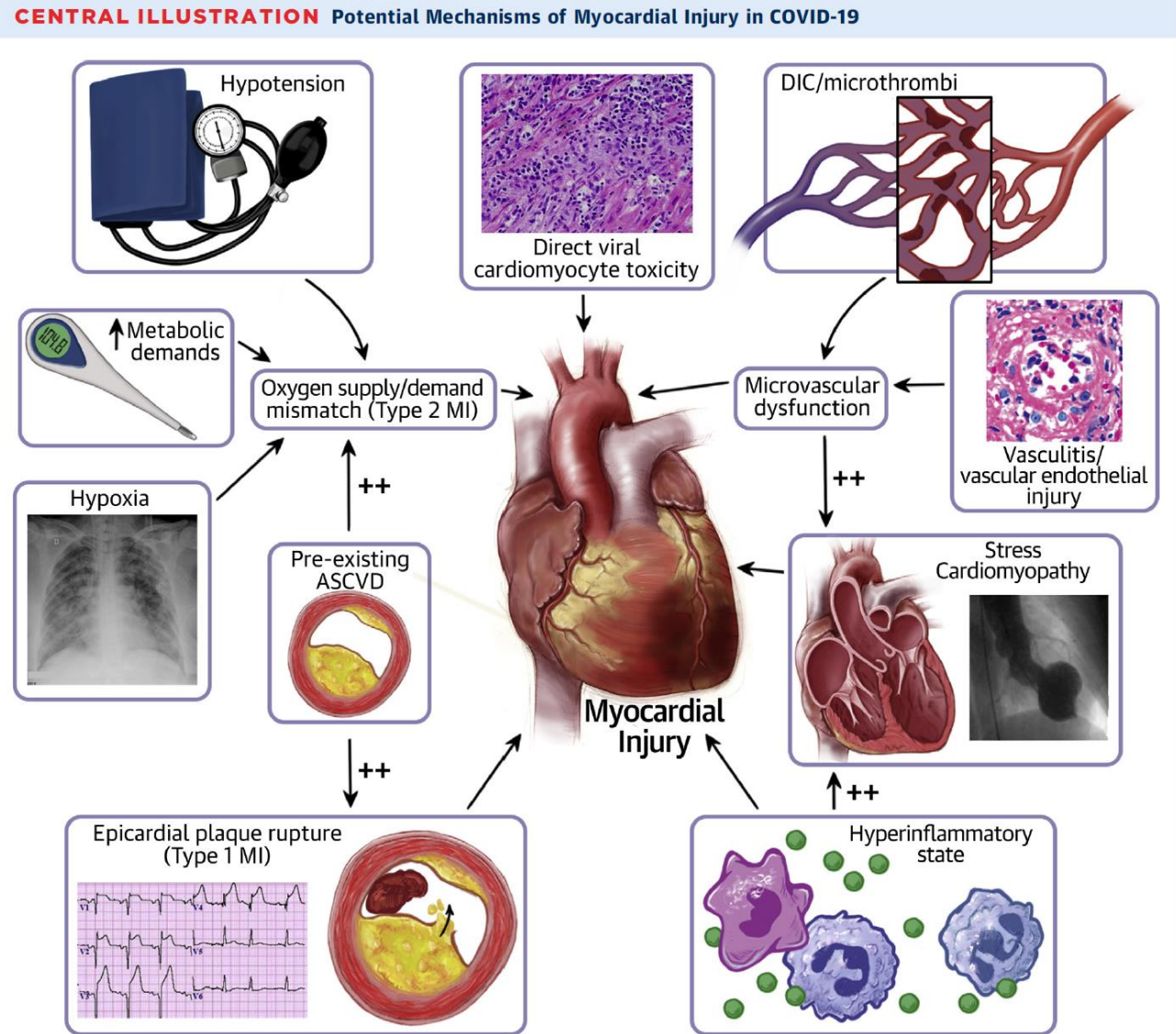
Dommages Myocardiques



LE PLUS SOUVENT
UTILISÉ

Troponine > 99e percentile de la
limite supérieure de la normale

Dommages Myocardiques Mécanismes Multifactoriels



Atri, D. et al. J Am Coll Cardiol Basic Trans Science. 2020;5(5):518-36.

ASCVD = atherosclerotic cardiovascular disease; COVID-19 = coronavirus disease-2019; DIC = disseminated intravascular coagulation; MI = myocardial infarction.

Dommmages Myocardiques

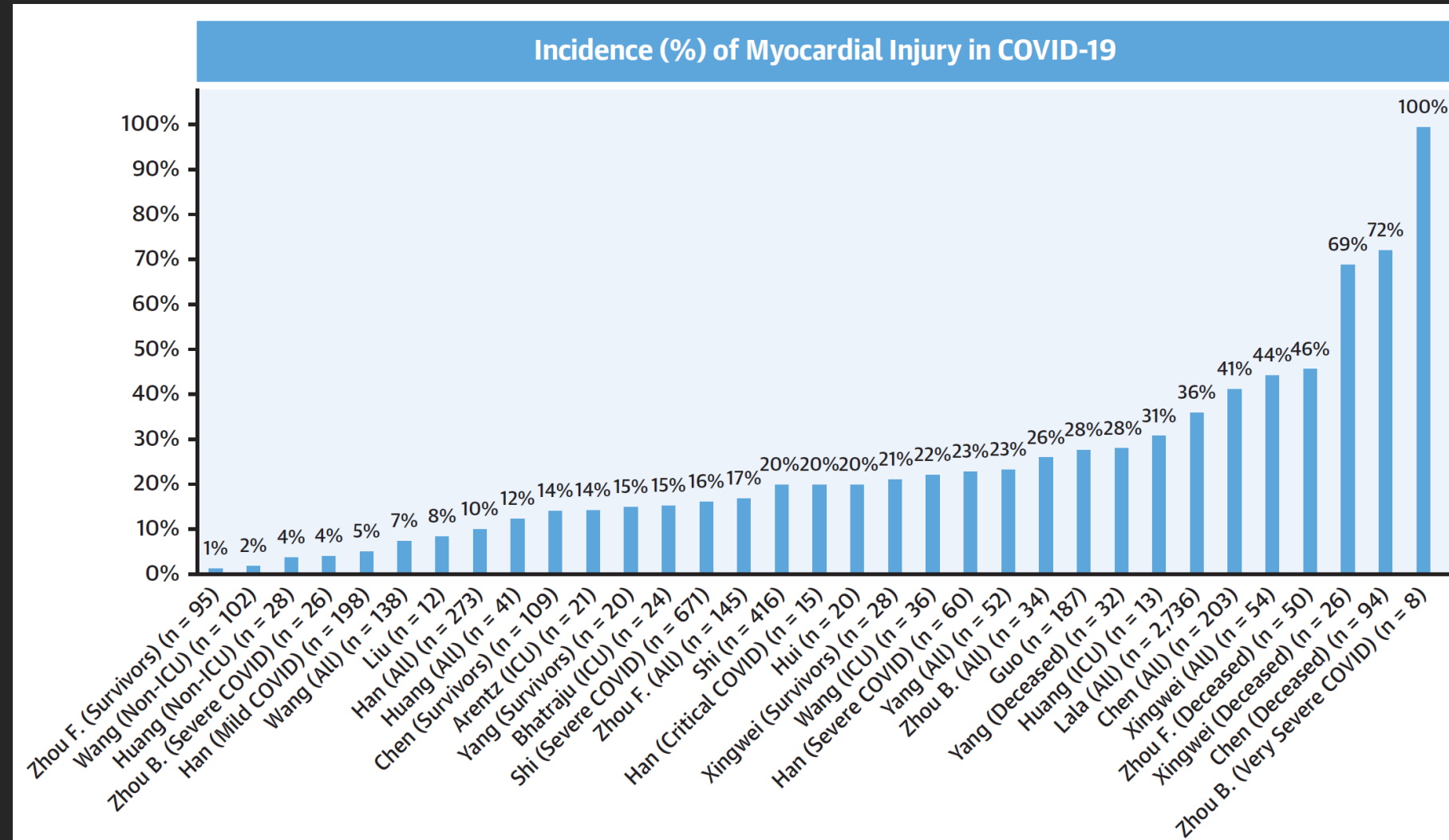
Prévalence

Prévalence

20 % en moyenne (méta-analyse)

Très variable selon le contexte:

- Externe VS hospitalisé
VS USI VS décès
- Augmente en fonction
de la sévérité de l'atteinte



Dommmages myocardiennes

Comparaison avec ARDS

26

Étude de cohorte

5 centres américains

243 patients intubés avec COVID-19

Comparaison avec une cohorte
historique d'ARDS 2aire pneumonie

Table 3. Characteristics of Intubated Patients With COVID-19 Compared With Patients With ARDS Secondary to Pneumonia

Characteristics	ARDS	COVID-19	P value
No. of patients	506	243	—
Troponin-positive	49.6	51.0	0.72
Troponin category			0.37
<ULN	50.4	49.0	
1 to 5 times ULN	24.1	22.6	
5 to 10 times ULN	8.3	12.4	
>10 times ULN	17.2	16.1	
Death	26.5	36.2	0.007
Ventilator-free days, d	13.1 (9.9)	13.0 (10.4)	0.85

Dommages Myocardiques

Prédicteur de Mortalité

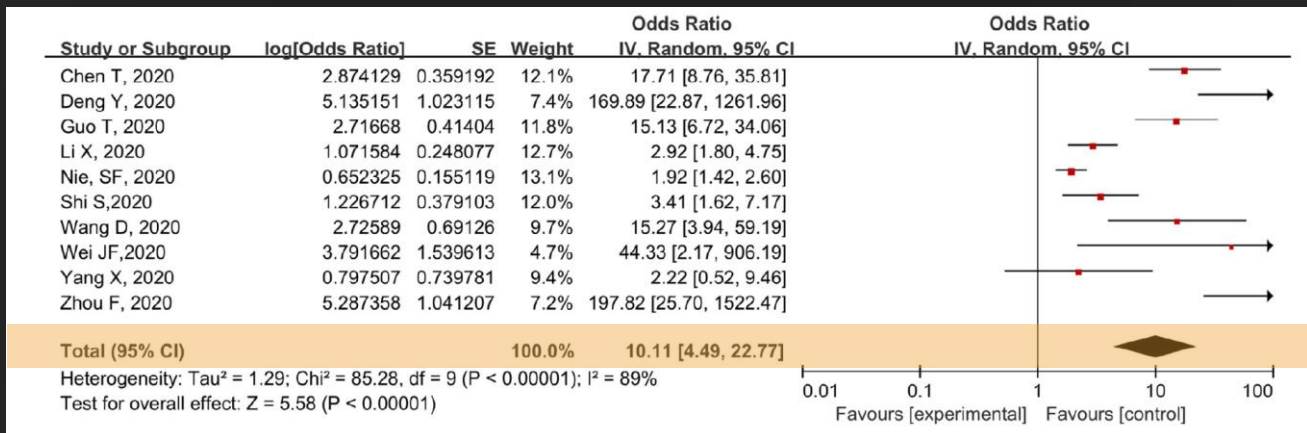


FIGURE 3 Meta-analysis for association between cardiac injury and all-cause death in patients hospitalized with COVID-19

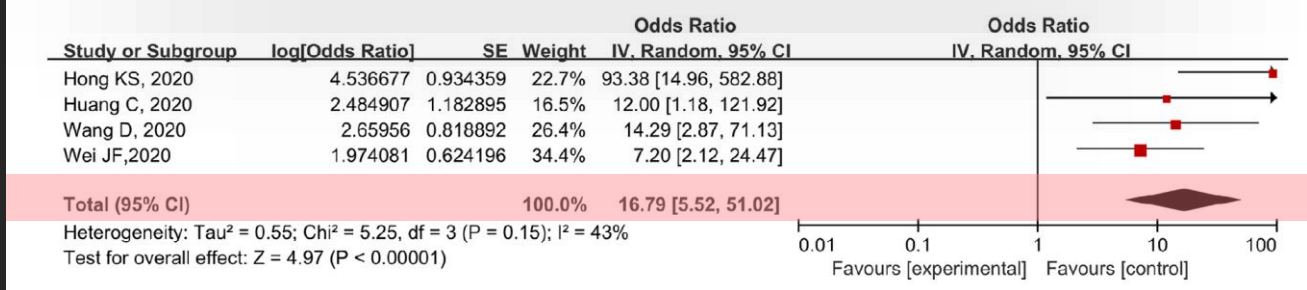


FIGURE 4 Meta-analysis for association between cardiac injury and all-cause death in patients hospitalized with severe COVID-19 cases



MÉTA-ANALYSE DE 21 ÉTUDES (2021)
 Chine principalement,
 mais aussi USA et Corée du Sud



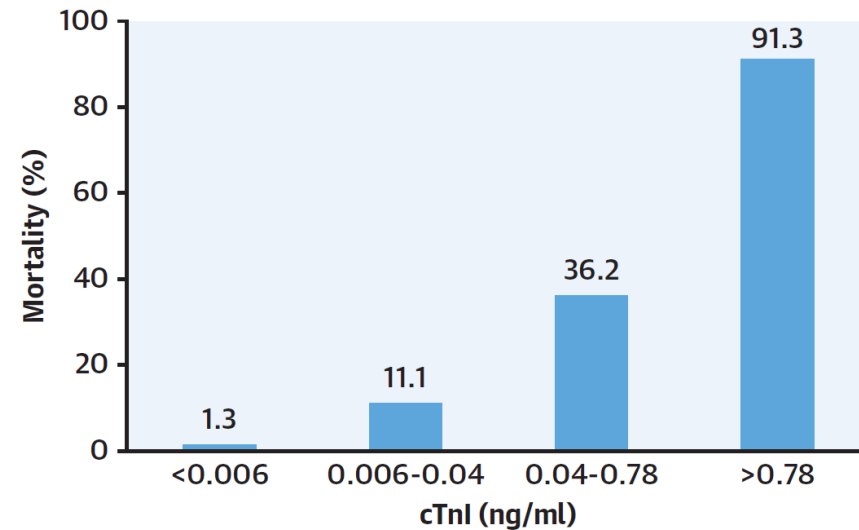
↑ RISQUE DE MORTALITÉ
 X 10 chez les cas hospitalisés
 X 17 chez les cas hospitalisés sévères

Dommmages Myocardiques

Prédicteur de Mortalité

Plus les troponines sont élevées,
plus le risque de mortalité est grand.

FIGURE 4 Relationship Between cTn and Mortality in COVID-19



Events (No.)	3	12	21	21
Patients (No.)	227	108	58	23

Data from Shi et al. (6) showing that the higher the cardiac troponin (cTn), the worse the outcome, and vice versa. Abbreviations as in Figure 2.

Dommmages Myocardiques

Prédicteur de Dysfonction VG/VD

Association entre le taux de troponines et la dysfonction ventriculaire droite, gauche ou biventriculaire

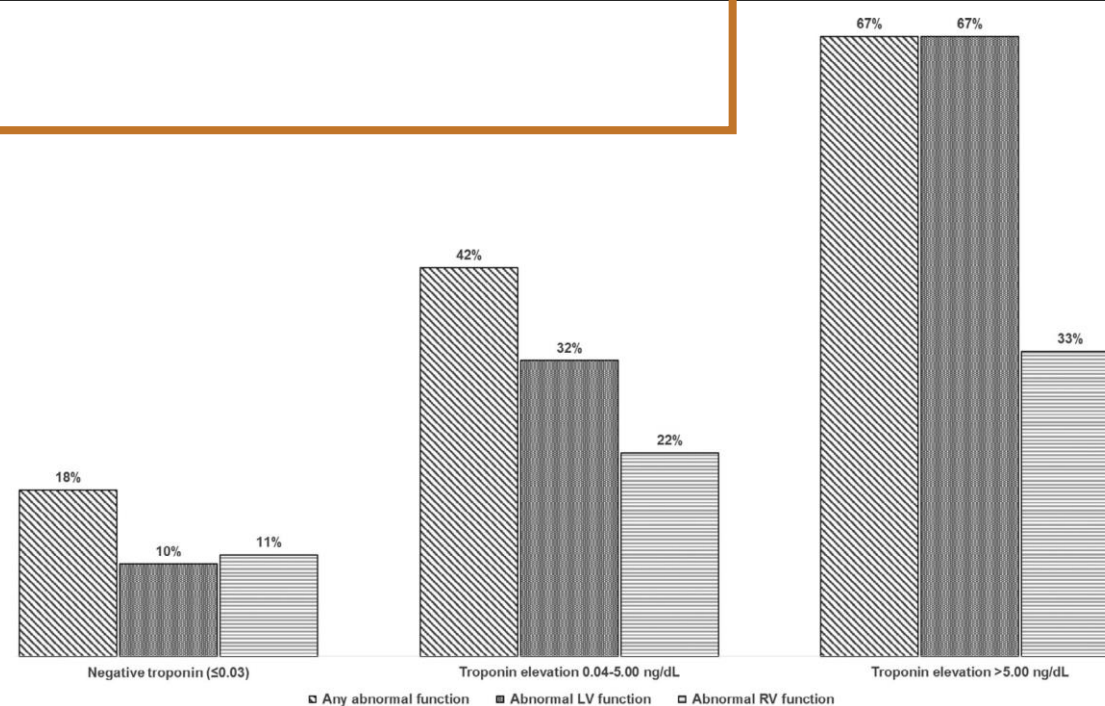


FIGURE 2 Troponin severity and abnormal cardiac function. A graded increase of prevalence of reduced cardiac function with increase of troponin-I levels was found for patients with no troponin elevation, mild troponin increase (0.04-5.00 ng/dL) and significant troponin increase (>5 ng/dL)

Étude rétrospective monocentrique (NYC)

143 patients consécutifs avec COVID-19 et ETT

(27 % IET)

(28 % mortalité)

Devrait-on mesurer les Troponines ?

30

IMPORTANCE DU
CONTEXTE CLINIQUE

PROBABILITÉ PRÉ-TEST
DU DIAGNOSTIC

RATIO RISQUES-BÉNÉFICES:
Éviter les investigations
découlantes qui ne changeront
pas la conduite

CORRÉLATION AVEC AUTRES
MARQUEURS DE SÉVÉRITÉ

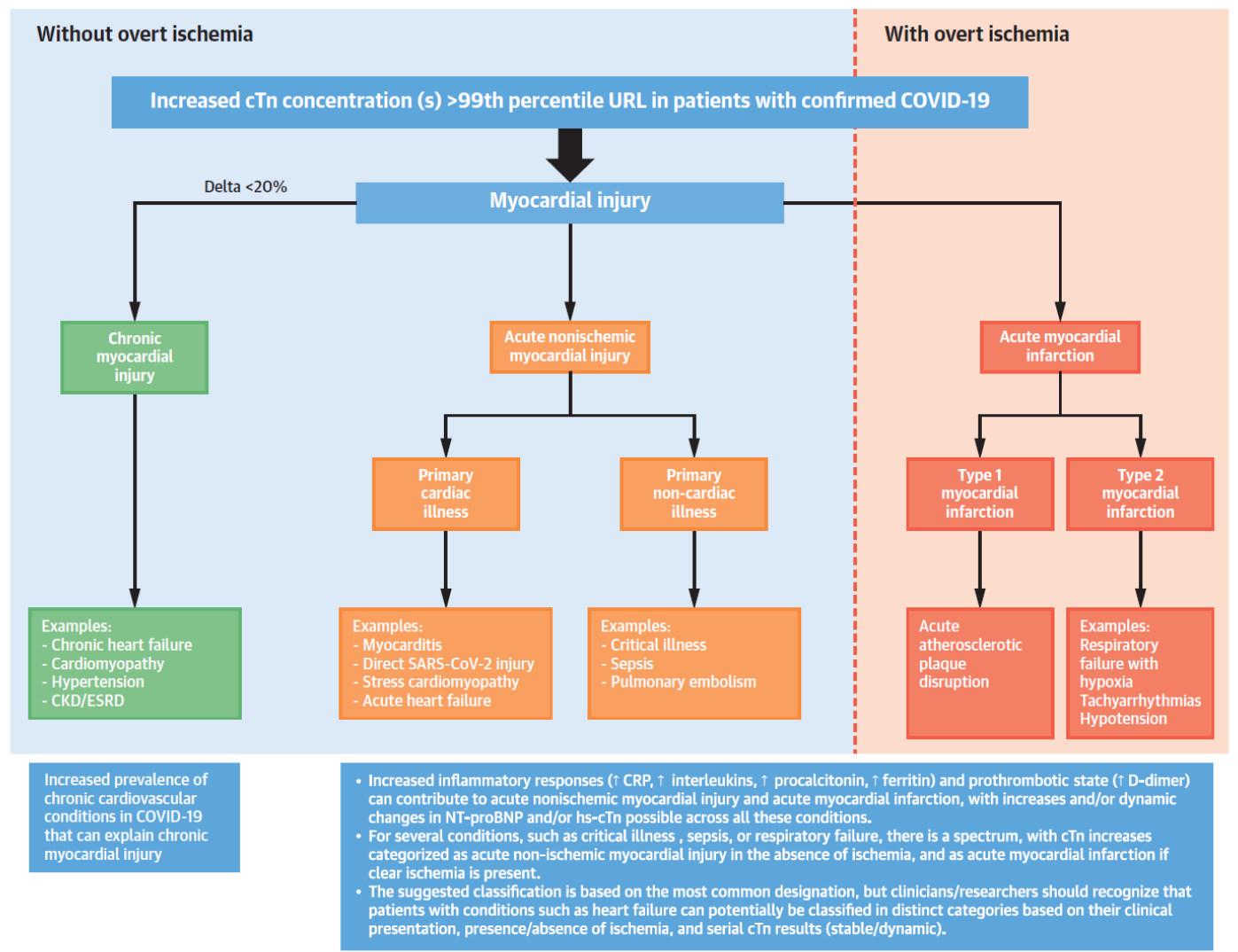
UN DOSAGE SÉRIÉ OFFRE
DAVANTAGE D'INFORMATION
QU'UNE SEULE VALEUR

Table 2

Recommendations on testing of cardiac biomarkers in patients hospitalized with COVID-19 infection.

	Recommendation
Chinese Clinical Guidance for COVID-19 Pneumonia Diagnosis and Treatment (7th edition)	In admitted patients
World Health Organization document - Clinical management of severe acute respiratory infection when COVID-19 disease is suspected (version 1.2, March 13, 2020)	At admission and as clinically indicated
American College of Cardiology	Only if clinically indicated
Handbook of COVID-19 Prevention and Treatment, The First Affiliated Hospital, Zhejiang University School of Medicine	In admitted patients
Asian Critical Care Clinical Trials Group	In admitted patients
BMJ Best Practice	In patients with severe illness


FIGURE 2 Classification of Myocardial Injury in COVID-19



Increases in cTn should be categorized as chronic myocardial injury, acute nonischemic myocardial injury, or acute myocardial infarction. For several conditions, there is a spectrum and the most common category is indicated, but can present in other ways. CKD = chronic kidney disease; COVID-19 = coronavirus disease-2019; CRP = C-reactive protein; cTn = cardiac troponin; ESRD = end-stage renal disease; NT-proBNP = N-terminal pro-B-type natriuretic peptide; URL = upper reference limit.

Troponines

Interprétation



Syndrome Coronarien Aigu

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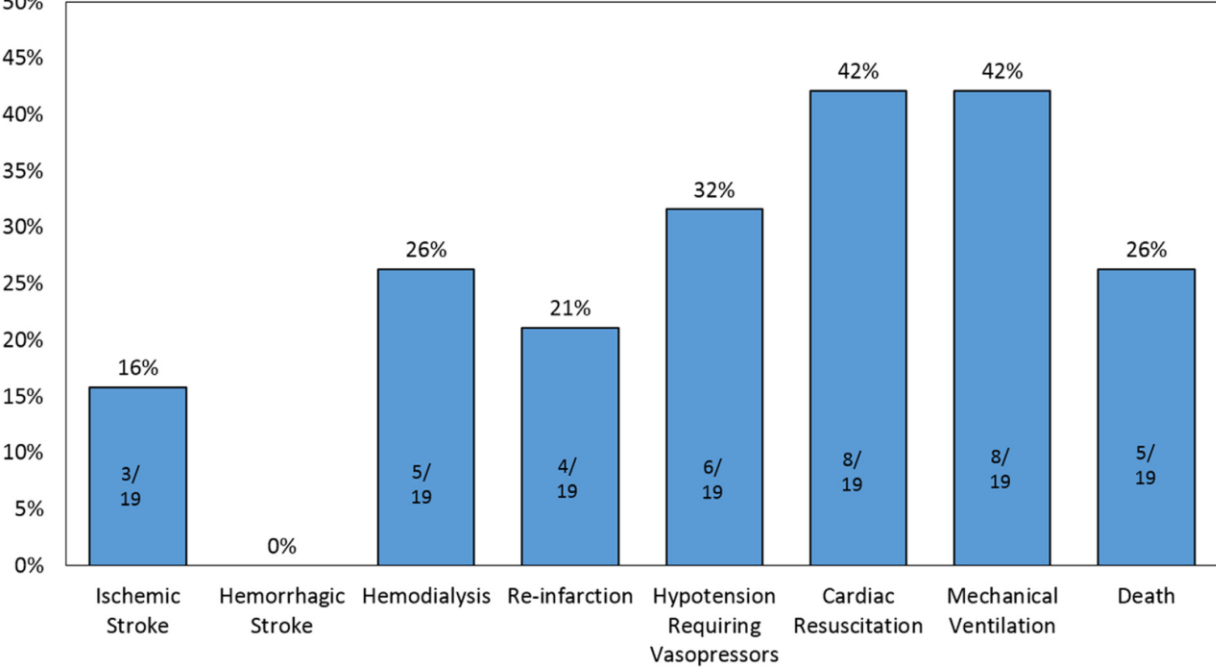


Figure 1. Clinical course and outcomes of COVID-19 patients presenting with STEMI who underwent primary PCI.

Étude retrospective multicentrique

4 centres

78 patients avec STEMI et COVID-19 +

- Pneumonie légère : 44 %
- Pneumonie sévère/ARDS : 13 %

Comorbidités:

- Âge moyen : 65 ans
- DLPD: 92 %
- HTA: 73 %
- DB2: 27 %
- MCAS: 78 %

Syndrome coronarien aigu

Mortalité cohorte totale = 12 %

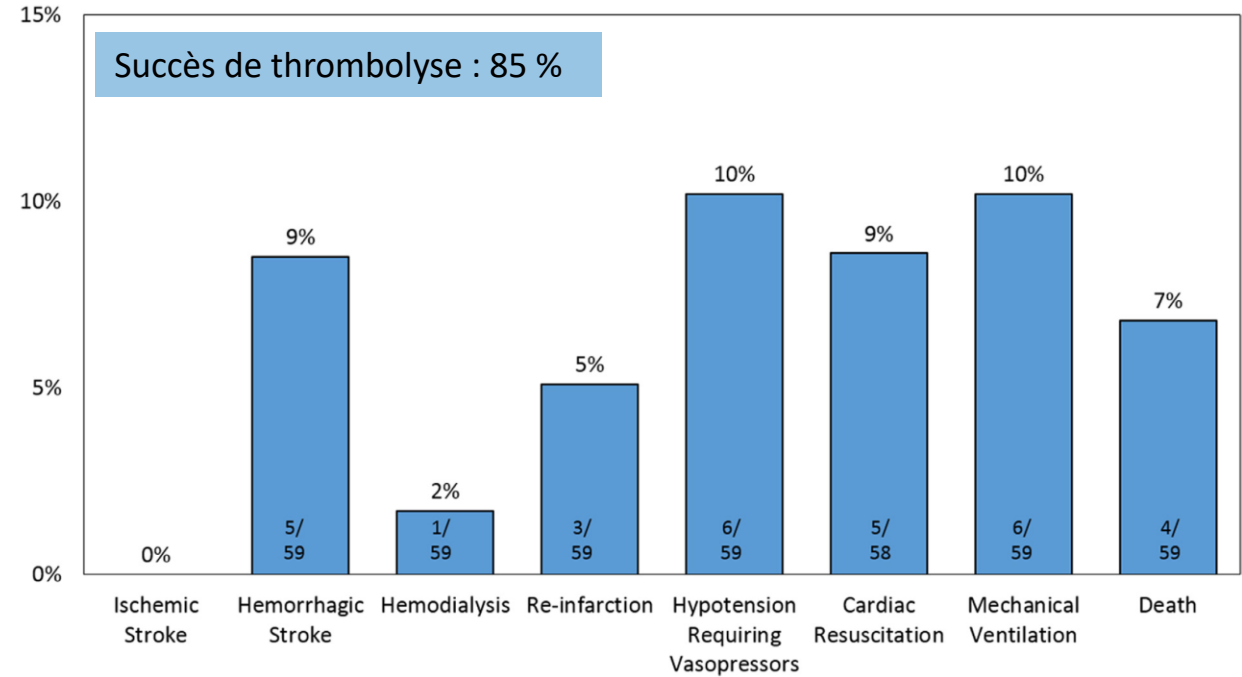


Figure 2. Clinical course and outcomes of COVID-19 patients presenting with STEMI who were treated with fibrinolytic therapy.

Table 1
Studies with COVID-19 patients presenting with ST-segment elevation on electrocardiogram and undergoing invasive coronary angiography.

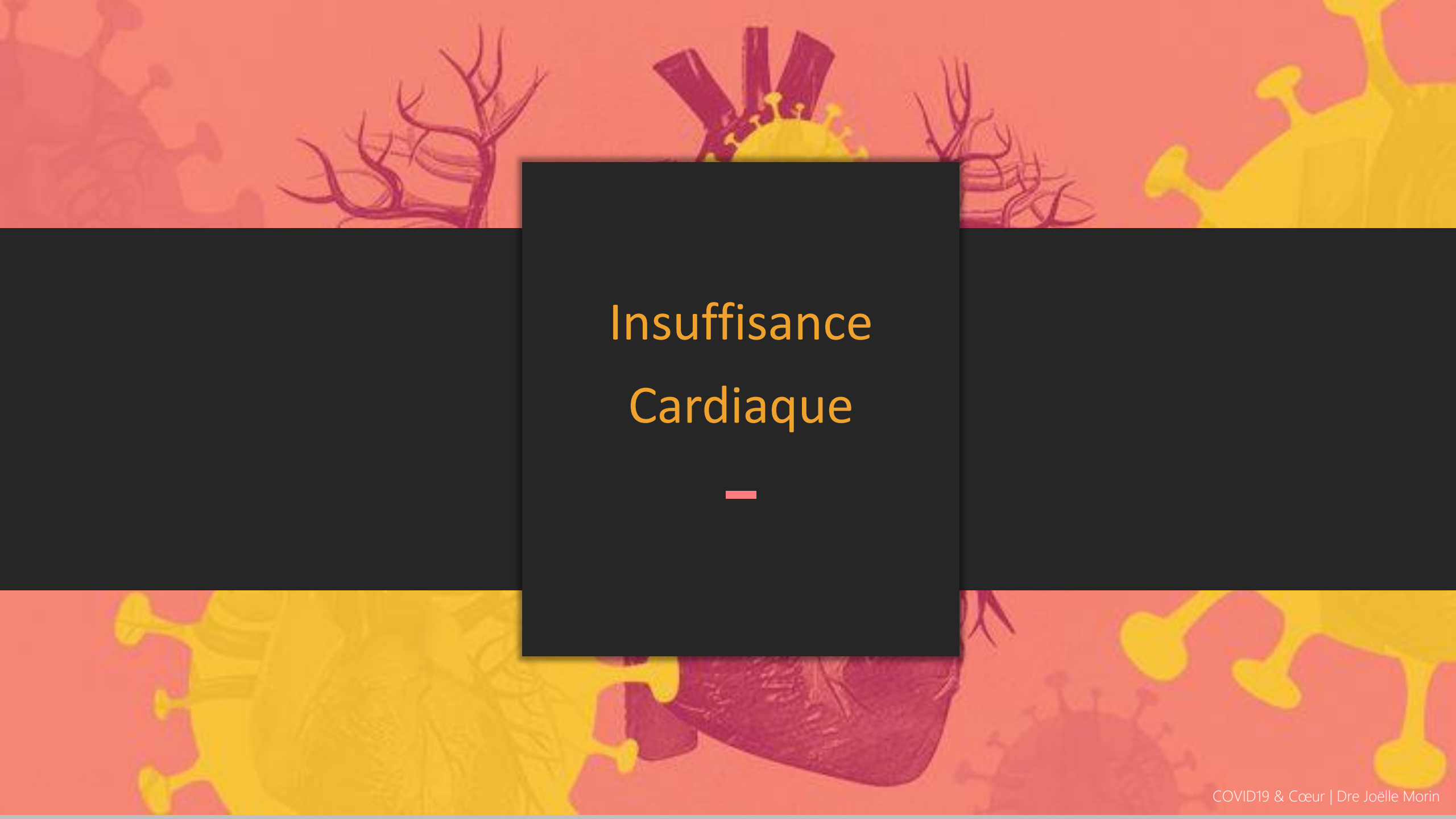
Author	Country	Sample size	Women (%)	Mean Age (years)	Hypertension (%)	Diabetes Mellitus (%)	Previous MI/PCI/CABG (%)	Culprit lesion identified during invasive angiography n/N (%)
Alaarag et al. [44]	Egypt	26	30.8	57.7	42.3	38.5	15.3	18/26 (69)
Bangalore et al. [24]	United States	18	17	63	65	35	–	6/9 (67)
Choudry et al. [22]	United Kingdom	39	15.4	61.7	71.8	46.2	15.4	38/38 (100)
Hamadeh et al. [45]	Lithuania, Italy	19	53	65	79	11	5	18/19 (95)
NACMI [46]	Spain, Iraq							
	United States	171	30	–	73	44	48	115/138 (83)
Siudak et al. [47]	Poland	145	28.7	63	46.2	14.5	25.5	123/143 (86)
Secco et al. [48]	Italy	31	22.6	72.3	71	38.7	35.4	8/10 (80)
Stefanini et al. [28]	Italy	28	28.6	68	71.4	32.1	10.7	17/28 (61)

CABG, coronary artery bypass grafting, MI, myocardial infarction, NACMI, North American COVID-19 STEMI Registry, PCI, Percutaneous coronary intervention.

Syndrome coronarien aigu

Particularités

- Plus de thrombose de tuteurs
- Charge thrombotique plus importante
- Plus de MCAS non obstructive



Insuffisance Cardiaque



Insuffisance Cardiaque

Insuffisance cardiaque de novo

- Rapporté chez 25 – 33 % des patients hospitalisés
- Présence de conditions médicales prédisposantes

Exacerbation aiguë d'une Insuffisance cardiaque pré-existante

- Immunité réduite
- Fragilité accrue
- Capacité d'adaptation hémodynamique moindre en situation d'infection

Insuffisance Cardiaque Mécanismes

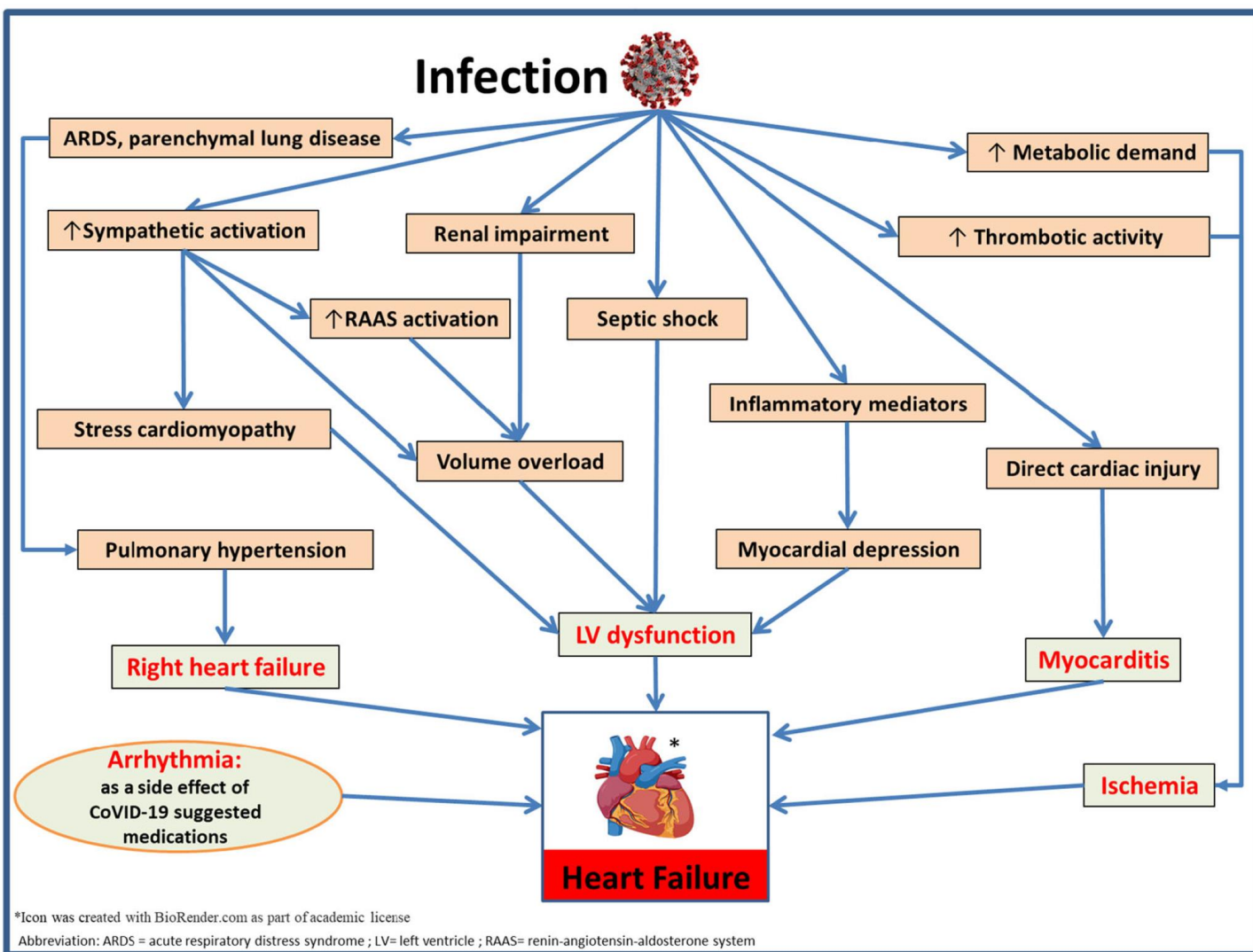
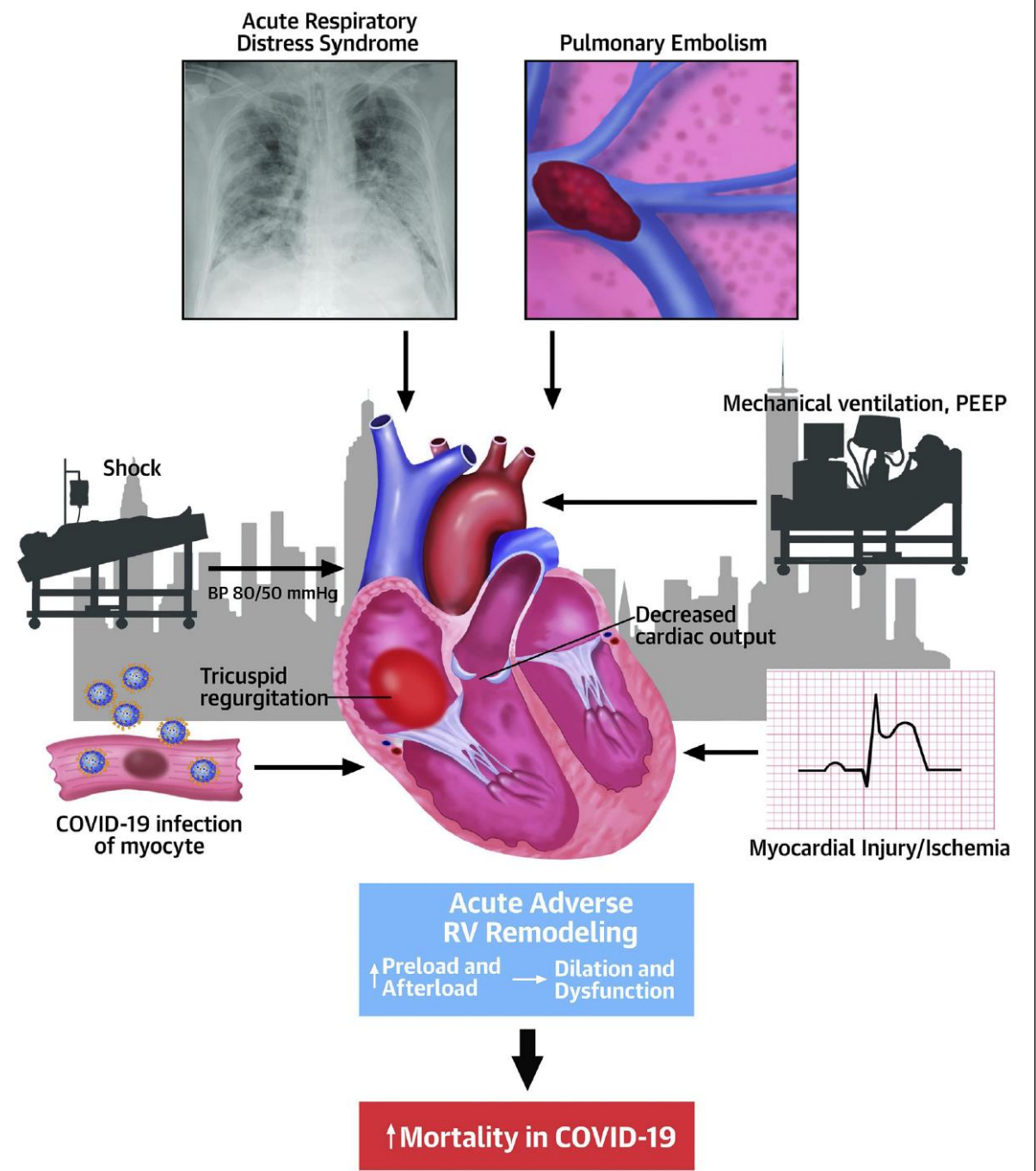


Fig. 1 COVID-19 and heart failure



Dysfonction Ventriculaire Droite Mécanisme

FIGURE 1 Adverse RV Remodeling By Echocardiography Was Associated With Doubling of Mortality in Patients With COVID-19



Insuffisance Cardiaque Pré-existante

Impact Pronostic

Étude retrospective

USA - 5 Centres du réseau Mount Sinai
Tous les patients consécutifs admis pour COVID-19 +

Âge moyen 63 ans

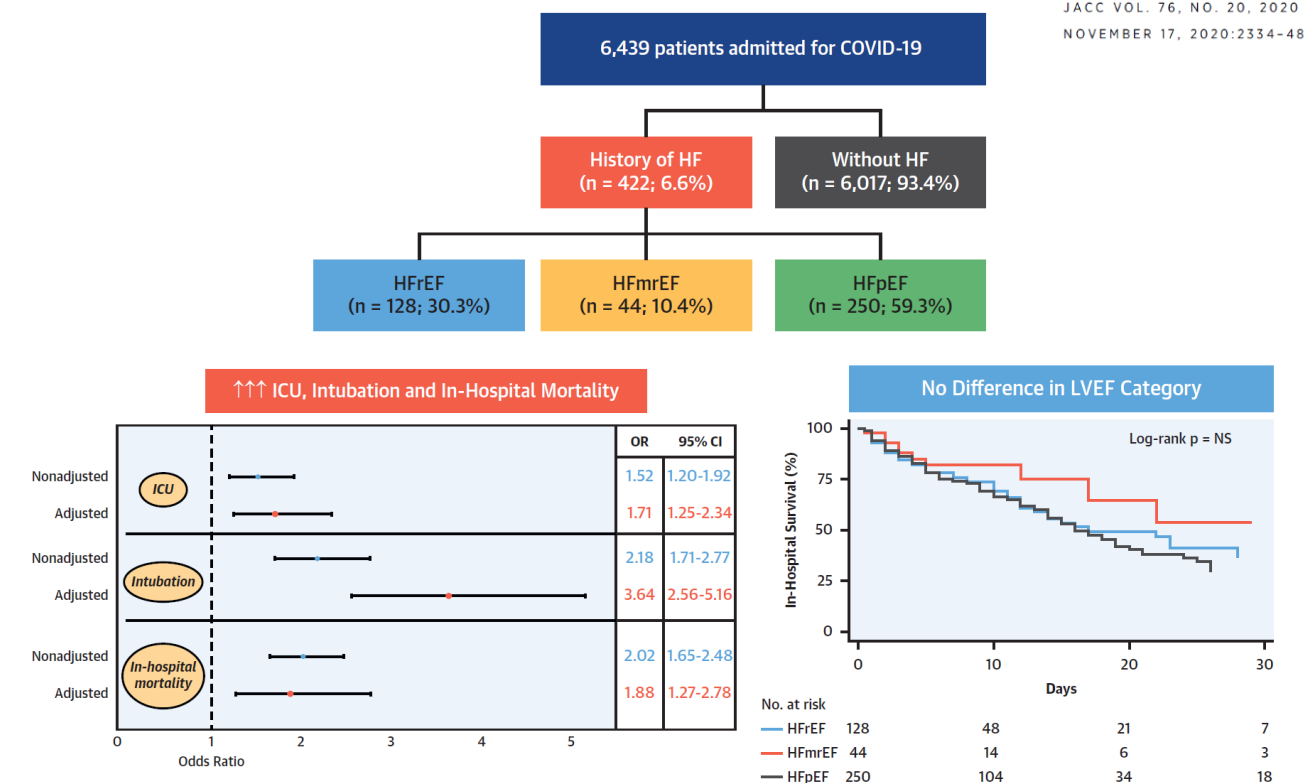
Homme 55%

Multiples comorbidités CV

NYHA 2 & 3 principalement

CENTRAL ILLUSTRATION History of Heart Failure and Coronavirus Disease-2019

JACC VOL. 76, NO. 20, 2020
NOVEMBER 17, 2020:2334-48



Alvarez-Garcia, J. et al. J Am Coll Cardiol. 2020;76(20):2334-48.

Patients with pre-existing heart failure (HF) are at nearly twice the risk of mortality and 3 times the risk of mechanical ventilation compared with patients without HF when hospitalized for coronavirus disease-2019 (COVID-19), yet outcomes among patients with HF were similar regardless of left ventricular ejection fraction (LVEF). (Top panel) Consort diagram of the study population. (Bottom right panel) Kaplan-Meier survival curves in patients hospitalized with COVID-19 according to LVEF category. (Bottom left panel) Forest plot of the effect of history of HF on outcomes in patients admitted for COVID-19. CI = confidence interval; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; HR = hazard ratio; ICU = intensive care unit.

Dysfonction ventriculaire

Impact Pronostic

Étude prospective

98 patients consécutifs admis pour COVID-19
ETT dans les 24h de l'admission
USI 46 % | Mortalité 13 %

FEVG Moyenne: survivant: 58%
FEVG Moyenne : décédés: 47%
Dysfontion VD chez 14 %

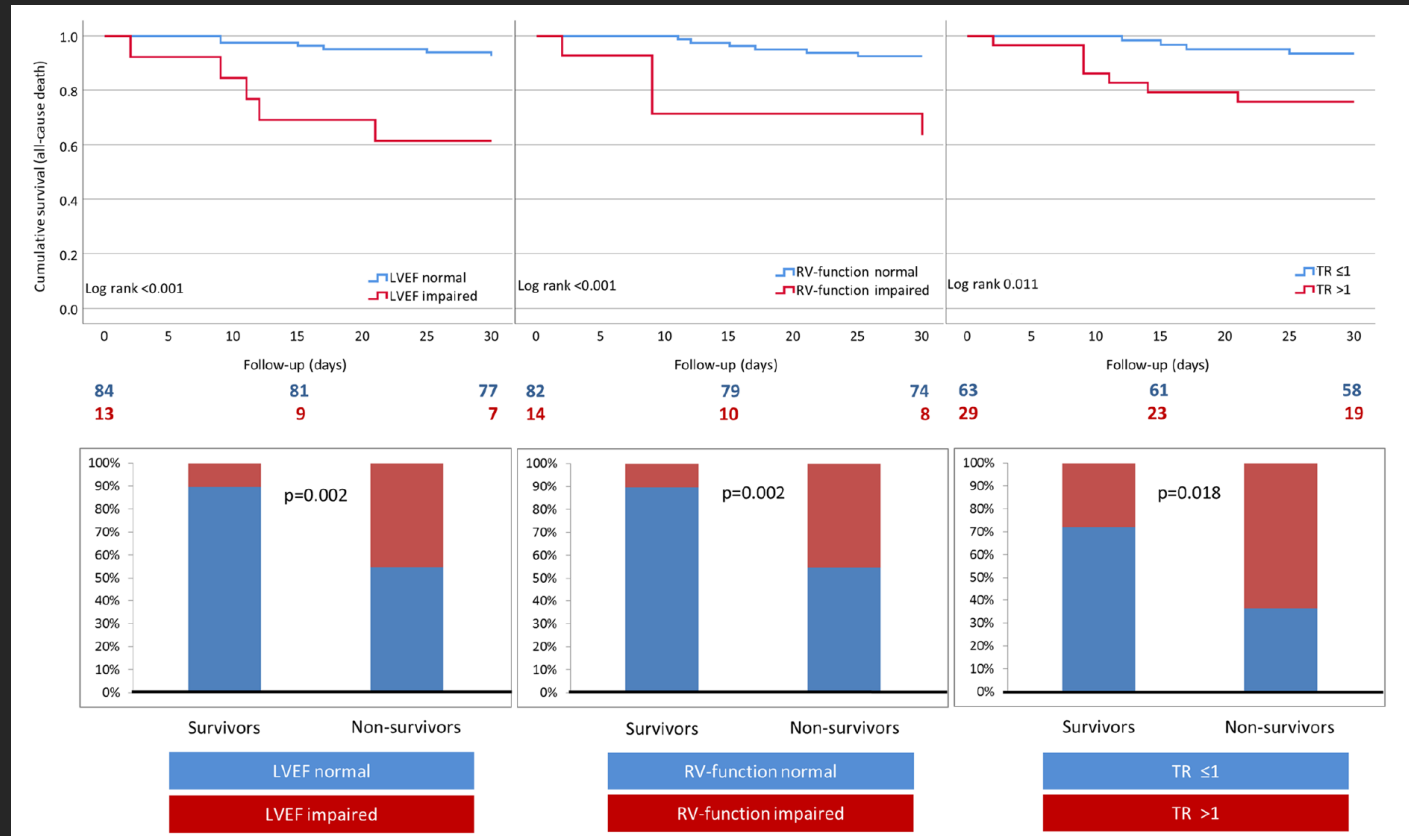
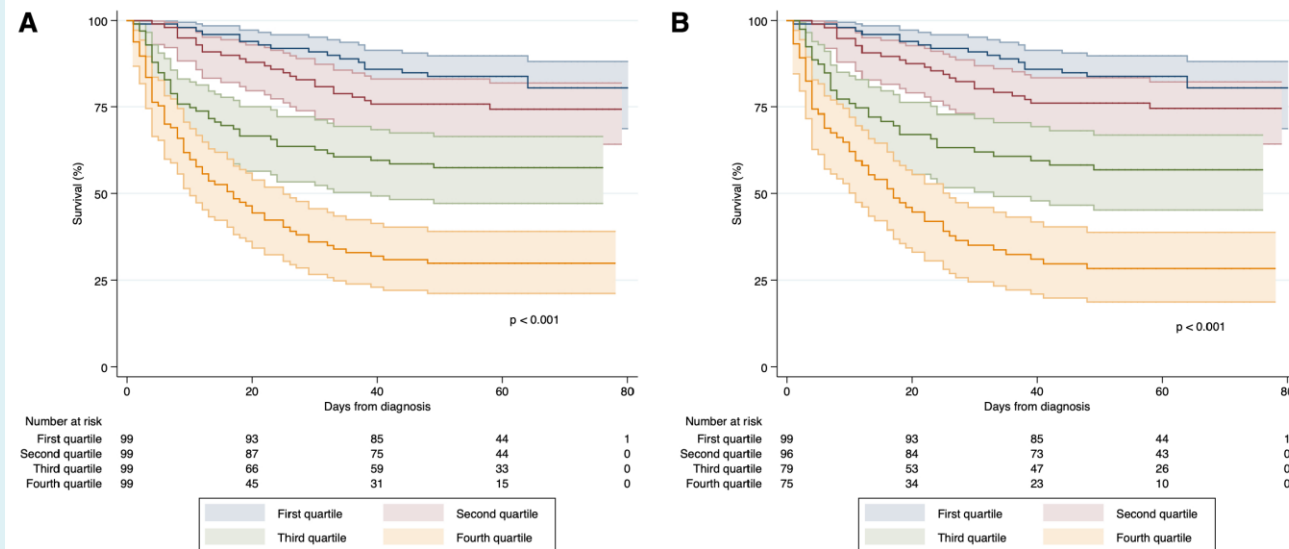


Fig. 1 Upper row: Kaplan–Meier curves showing cumulative event-free survival for the endpoint all-cause death stratified according to LVEF, RV function and TR. Lower row: bar diagrams showing dis-

tribution of LVEF, RV-function and TR between survivors and non-survivors. *TR* tricuspid regurgitation
Clinical Research in Cardiology (2020) 109:1491–1499

BNP

Valeur Pronostique



Étude de cohorte prospective - Espagne

396 patients consécutifs avec COVID-19 et
Dosage de NT-pro-BNP per-hospitalization

Suivi moyen 53 jours

- NT-pro-BNP élevés : 48.5 %
- Manifestations cliniques d'IC: 11.8 %
- Mortalité: 38.5 %



Myocardite

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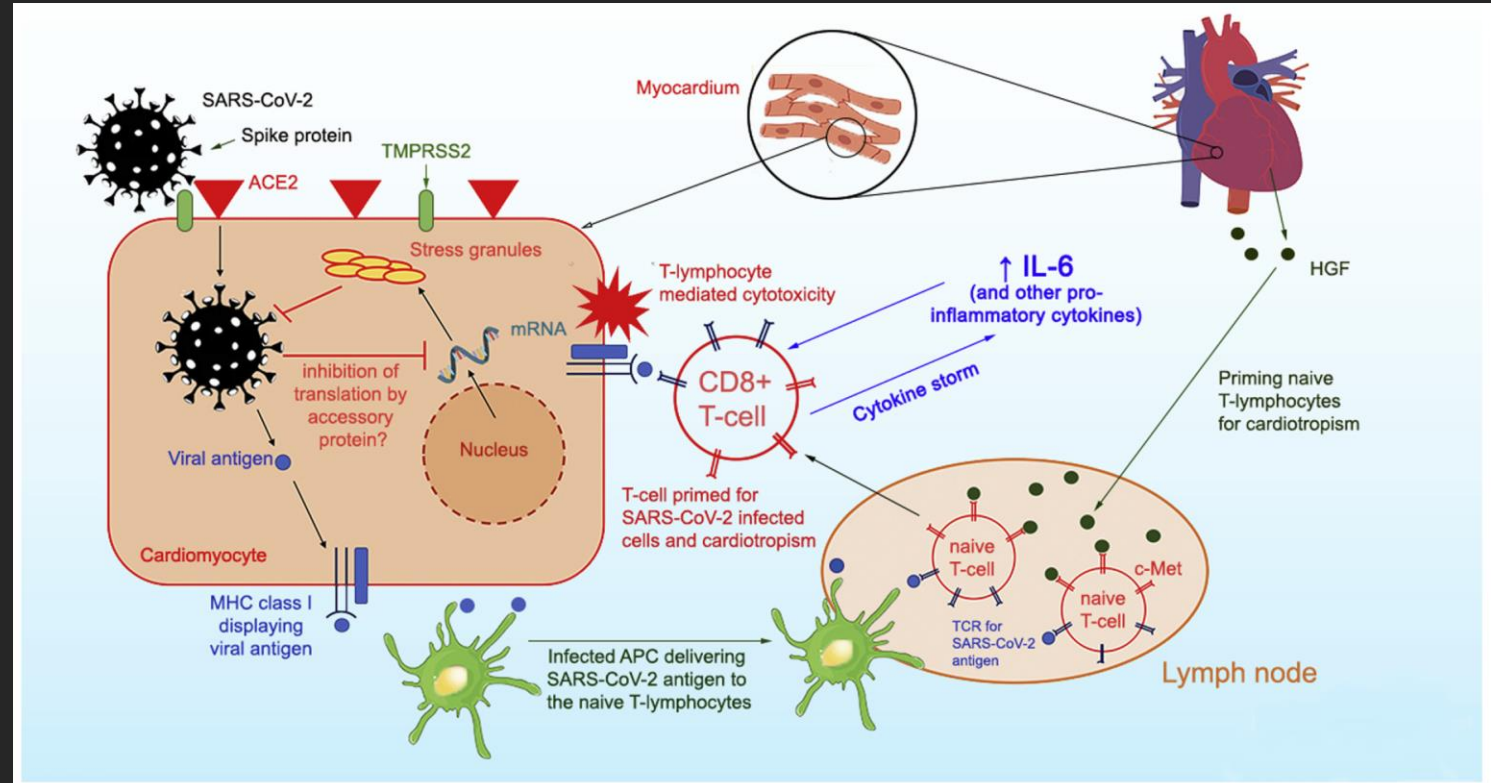
Myocardite

Hypothèses Pathophysiologiques

43

Infection directe des cardiomyocytes

Hyperactivation du système immunitaire et tempête cytokinique



Détection du COVID-19

a/n cardiaque



Présence de virus a/n cardiaque : 61.5 %

A/n des cellules interstitielles
(et non a/n des cardiomyocytes)

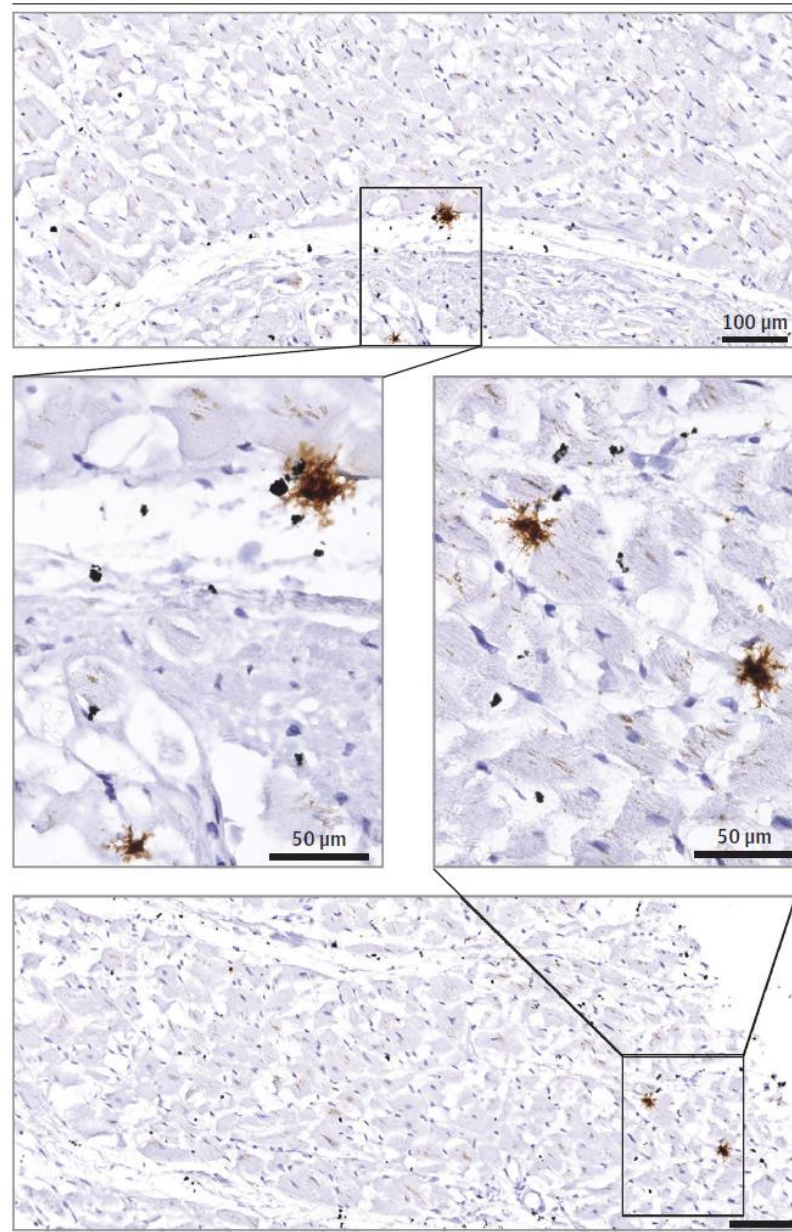


Réplication virale a/n cardiaque : 13 %



Aucun critère de myocardite

Figure 2. In-situ Hybridization to Detect Virus RNA in SARS-CoV-2-Infected Cardiac Tissue

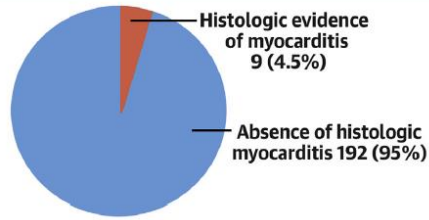


Paraffin-embedded cardiac tissue section of a patient with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection revealed interstitial cells carrying virus RNA detected.

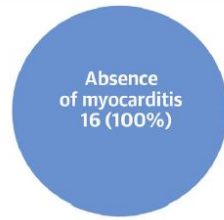
39 autopsies consecutives de patients avec COVID-19:

La présence du COVID-19 a/n cardiaque ne cause pas nécessairement de réaction inflammatoire et de myocardite clinique

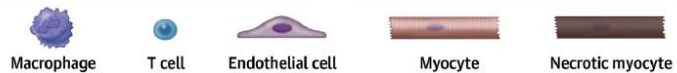
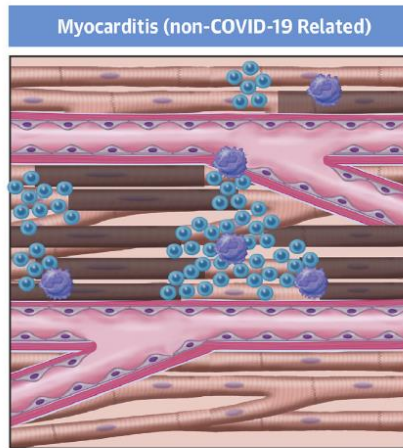
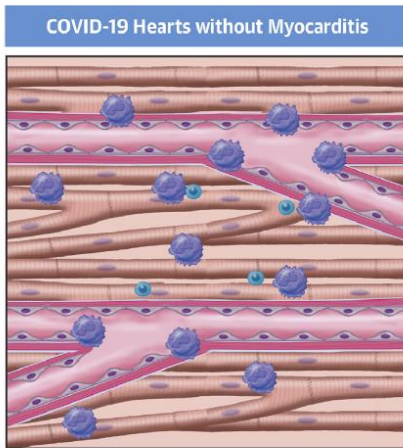
Histologic Assessment in the Literature for Myocarditis in COVID-19 Cases



Histologic Assessment of Myocarditis in COVID-19 Victims, CVPATH Experience



Pattern of Inflammatory Cell Infiltration



Kawakami, R. et al. *J Am Coll Cardiol.* 2021;77(3):314-25.

According to published data on pathological evidence for myocarditis in subjects with coronavirus disease-2019 (COVID-19), the rate of myocarditis is 4.5%. In our experience with 16 COVID-19 autopsy cases, no case met diagnostic criteria for myocarditis. In a comparison of inflammatory cells in the myocardium of subjects dying from traumatic versus COVID-19 deaths (but without a diagnosis of myocarditis), there were less cluster of differentiation (CD) 3-positive cells in COVID-19 cases and more CD68-positive cells.

Myocardite

45

Une entité surestimée ?

Peu des cas publiés rencontrent réellement les critères histologiques de myocardite.

Kawakami R & al. *JACC.* 2021;77:314-25

Myocardite

Une entité peu fréquente

46

Morbidity and Mortality Weekly Report

Association Between COVID-19 and Myocarditis Using Hospital-Based Administrative Data — United States, March 2020–January 2021

Banque de données du CDC: 900 hôpitaux /36 005 294 hospitalisations

- Comparaison entre 2019 (pré-COVID) et 2020:
 - ↑ risque de myocardite de 42%
- Augmentation X15 du risque de myocardite chez les patients avec COVID:
 - Risque de myocardite chez patient AVEC COVID: 0.146 %
 - Risque de myocardite chez patient SANS COVID: 0.009 %
- Le risque de myocardite demeure très bas, avec ou sans COVID.

Myocardite post-vaccin

Une entité peu fréquente

Morbidity and Mortality Weekly Report

Use of mRNA COVID-19 Vaccine After Reports of Myocarditis among Vaccine Recipients: Update from the Advisory Committee on Immunization Practices — United States, June 2021

Banque de données du VAERS (vaccine adverse event reporting system)

- 1226 cas de myocardite pour plus de 296 millions de doses de vaccin = 0.0004 %
- Surtout chez les jeunes hommes (âge median = 26 ans)

Myocardite Investigation



Figure 1. Tools used to diagnose COVID-19 myocarditis. COVID-19: Coronavirus-19; ECG: Routine 12-lead electrocardiogram.

Échocardiographie

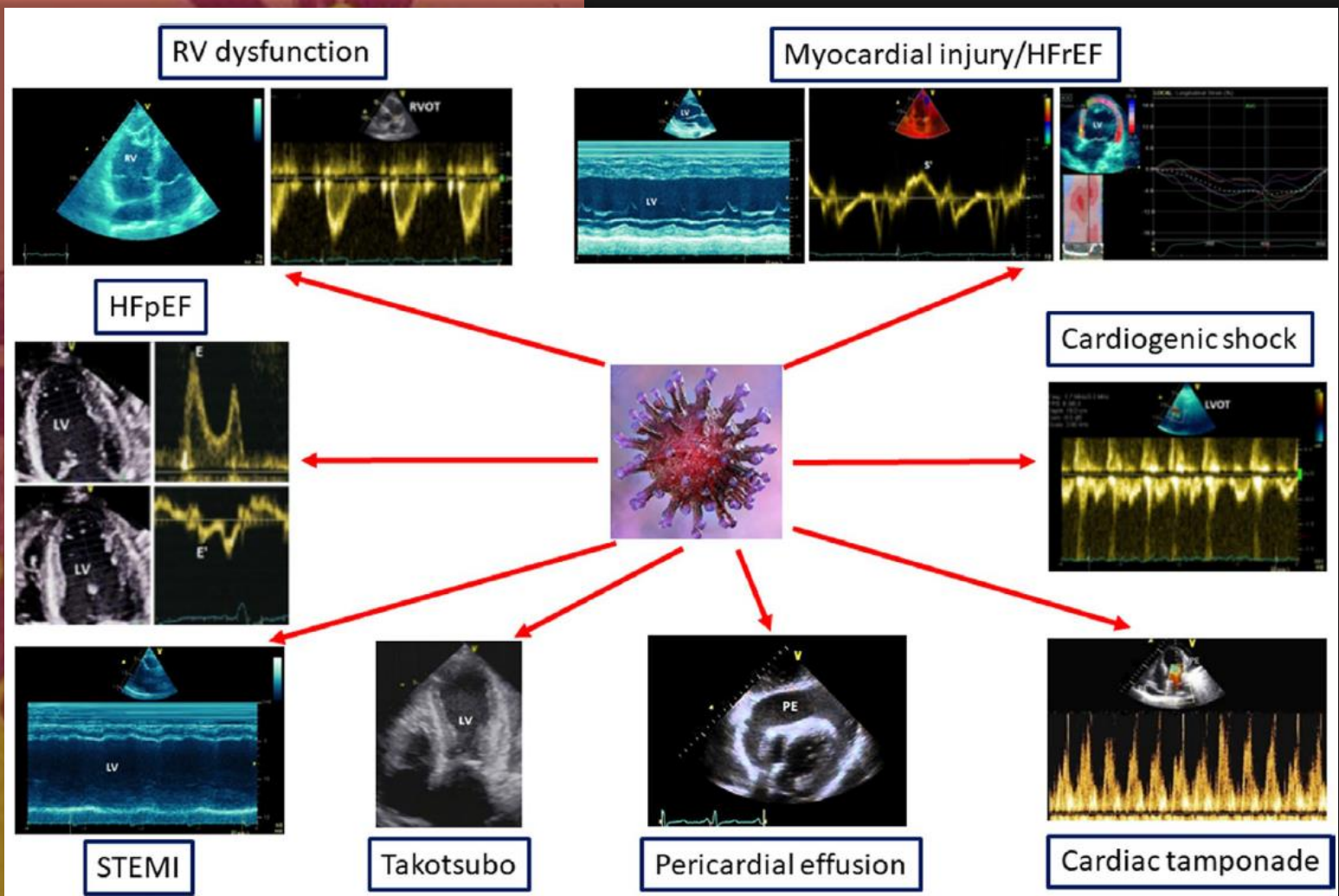


FIGURE 2 Spectrum of cardiovascular syndromes and echocardiographic abnormalities in patients with COVID-19 infection. *Top panel*



AMERICAN COLLEGE of CARDIOLOGY
 «Patients demonstrating heart failure, arrhythmia, ECG changes or cardiomegaly should have echocardiography»



Faible sensibilité
de la Bx myocardique
pour myocardite virale

10-35%



Faible prévalence
de la myocardite
à COVID-19



Risques de la procédure

- Procédure invasive: 6% complications
- Risques de transmission du COVID-19



Impact sur la prise en
charge du patient ?

Considérer tx empirique

Biopsie myocardique

À réserver au "worst-case scenario"

Myocardite

IRM cardiaque

Nécessite stabilité hémodynamique et ventilatoire

Déplacement du patient / Risque de contamination

Désinfection de l'environnement IRM

Changement de la conduite ?



Myocardite

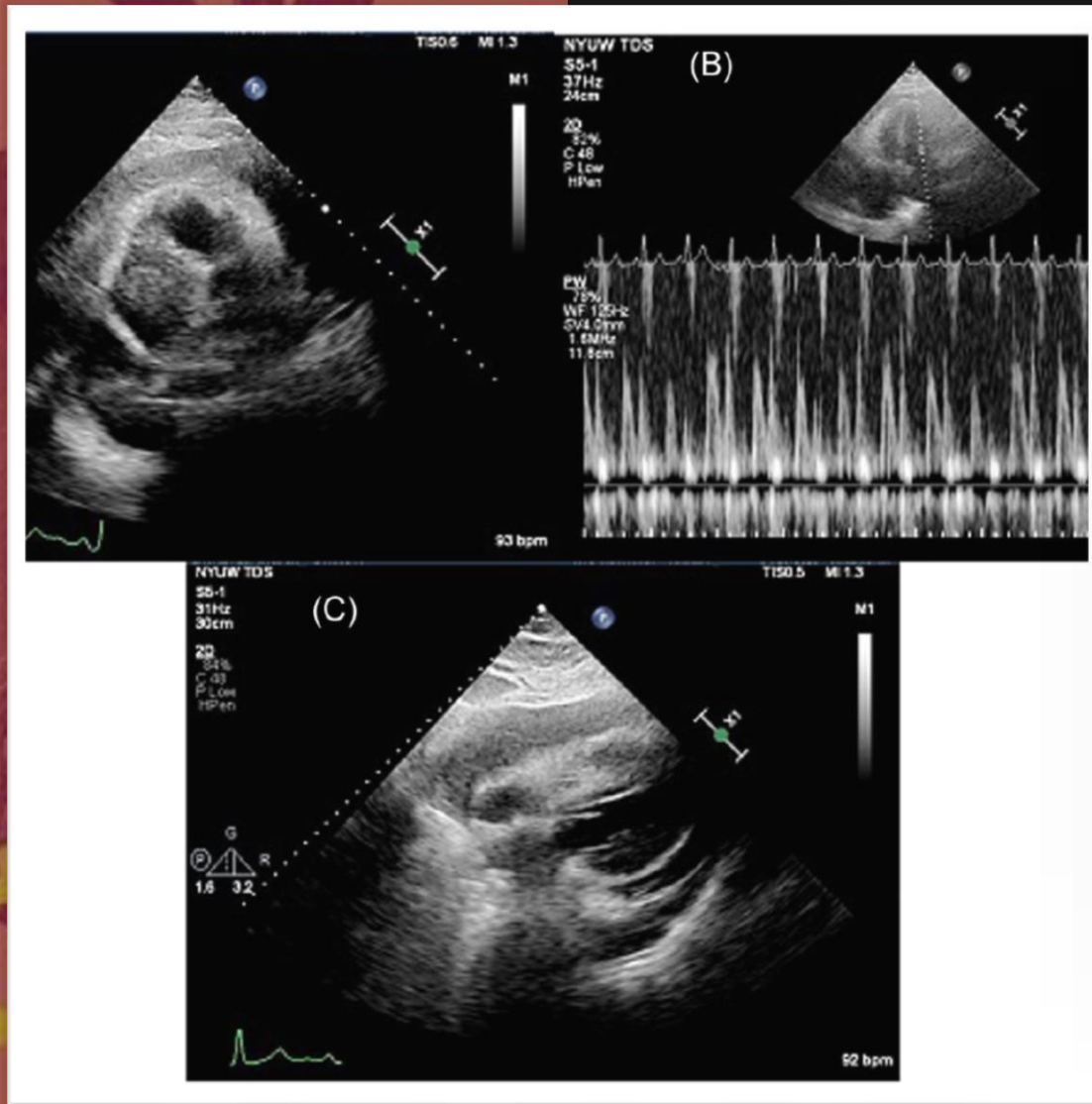
Prise en charge

Traitement de support pour tous

Si myocardite fulminante:


Stéroïdes haute dose et Immunoglobulines IV à considérer





De la Péricardite à la Tamponade... en passant par la Myopéricardite

- Le diagnostic de péricardite aiguë est sans doute sous-estimé.
- Quelques rapports de cas de péricardite aiguë
- Tx usuel de la péricardite aiguë
- Quelques rapport de cas de tamponade
- Association variable avec \uparrow troponines ou \downarrow FEVG



Syndrome Inflammatoire Multisystème de l'Adulte (MIS-A)



MIS-A

Table 2: CDC criteria for multisystem inflammatory syndrome in adults and children with SARS-CoV-2 infection and Kawasaki disease²⁻⁴

Characteristic	Multisystem inflammatory syndrome in adults	Multisystem inflammatory syndrome in children	Kawasaki disease
Case definition	Hospital admission of a patient aged ≥ 21 yr without evidence of severe respiratory illness and no alternative plausible diagnosis and involvement of 1 or more extrapulmonary organ systems: <ul style="list-style-type: none"> • Hypotension or shock • Cardiac dysfunction • Arterial or venous thromboembolism • Acute liver injury and laboratory evidence of acute inflammation	Patient aged < 21 yr with fevers $> 38.0^{\circ}\text{C}$ for ≥ 24 h, or report of subjective fever lasting ≥ 24 h with laboratory evidence of clinically severe illness requiring hospital admission with multisystem (≥ 2) organ involvement: <ul style="list-style-type: none"> • Cardiac • Renal • Respiratory • Hematologic • Gastrointestinal • Dermatologic • Neurologic 	Complete Kawasaki disease: <ul style="list-style-type: none"> • Fevers ≥ 5 d • AND ≥ 4 principal clinical features <ul style="list-style-type: none"> • Extremity changes* • Rash† • Conjunctivitis‡ • Oral changes§ • Cervical lymphadenopathy (at least 1.5 cm in diameter, usually unilateral) Suspected incomplete Kawasaki disease: <ul style="list-style-type: none"> • Fevers ≥ 5 d • AND • 2–3 compatible clinical criteria, or infants with fevers ≥ 7 d without other explanation.
Supportive investigations and laboratory finding	Elevated CRP, ferritin, D dimer or IL-6	Elevated CRP, ESR, fibrinogen, procalcitonin, D dimer, ferritin, lactate dehydrogenase or IL-6, elevated neutrophils, reduced lymphocytes and low albumin	CRP ≥ 3.0 mg/dL (or) ESR ≥ 40 mm/hr AND <ul style="list-style-type: none"> • 1) Positive echocardiogram: From AHA criteria,² echocardiography is considered positive if any of 3 conditions are met: <ul style="list-style-type: none"> • Z score of left anterior descending coronary artery or right coronary artery ≥ 2.5; coronary artery aneurysm is observed; or ≥ 3 other suggestive features exist, including decreased left ventricular function, mitral regurgitation, pericardial effusion • or Z scores in left anterior descending coronary artery • or right coronary artery of 2–2.5 • OR • 2) ≥ 3 supportive laboratory findings (anemia for age, platelet count $\geq 450\ 000$ after 7th day of fevers, albumin ≤ 3.0 g/dL, elevated alanine aminotransferase, leukocytes $\geq 15\ 000/\text{mm}^3$, or urine with ≥ 10 leukocyte/hpf)
SARS-CoV-2	Positive for current or previous SARS-CoV-2 infection (nucleic acid, antigen, or antibody) during admission or in the previous 12 wk	Positive for current or recent SARS-CoV-2 infection by RT-PCR, serology or antigen test, or exposure to a suspected or confirmed COVID-19 case within 4 wk before onset of symptoms	NA

Note: AHA = American Heart Association, CDC = Centers for Disease Control and Prevention, CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, hpf = high power field, IL-6 = interleukin 6, NA = not applicable, RT-PCR = reverse transcription–polymerase chain reaction.

*Erythema and edema of the hands and feet in acute phase or periungual desquamation in subacute phase, or both.

†Maculopapular, diffuse erythroderma or erythema multiforme-like.

‡Bilateral bulbar conjunctival injection without exudate.

§Erythema and cracking of lips, strawberry tongue, or erythema of oral and pharyngeal mucosa.

Arythmies



Arythmies

Table 1

Cardiac Arrhythmias Occurring in Patients with COVID-19 Infection.

Sinus tachycardia
Sinus bradycardia
Conduction disturbances (AVB/BBB)
Atrial premature complexes
Atrial fibrillation
Supraventricular tachycardia
Ventricular premature complexes
Non-sustained ventricular tachycardia
Polymorphic ventricular tachycardia (Torsade des pointes)
Sustained ventricular tachycardia
Ventricular fibrillation
Pulseless electrical activity

AVB = atrioventricular block; BBB = bundle branch block.

Les plus fréquentes:

- Tachycardie sinusale (1e)
- Fibrillation auriculaire (2e)

Arythmies

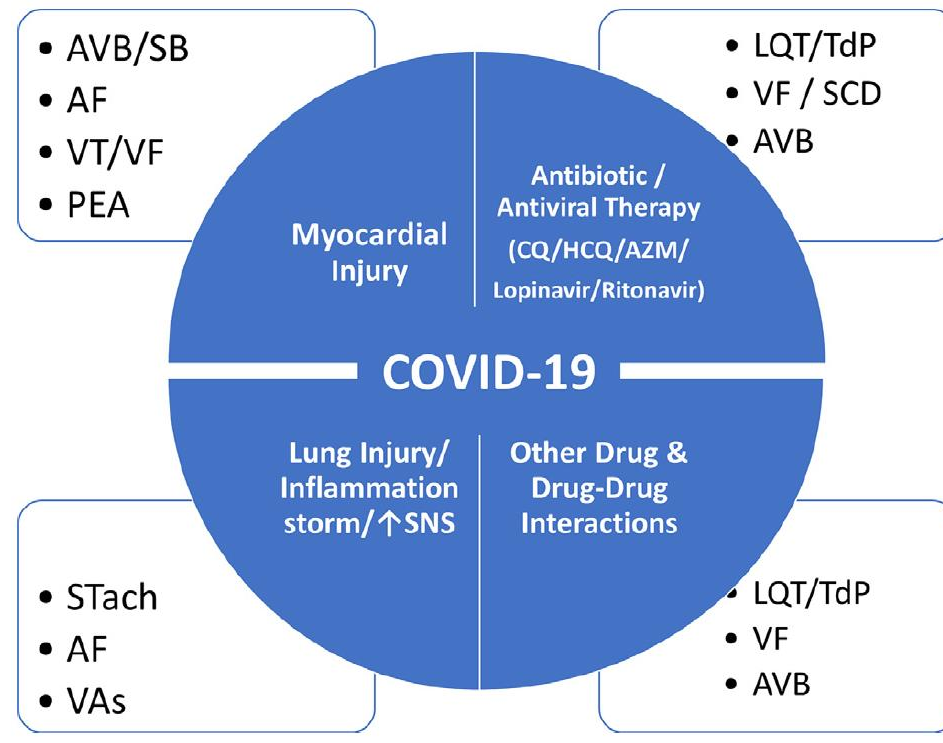
Mécanismes

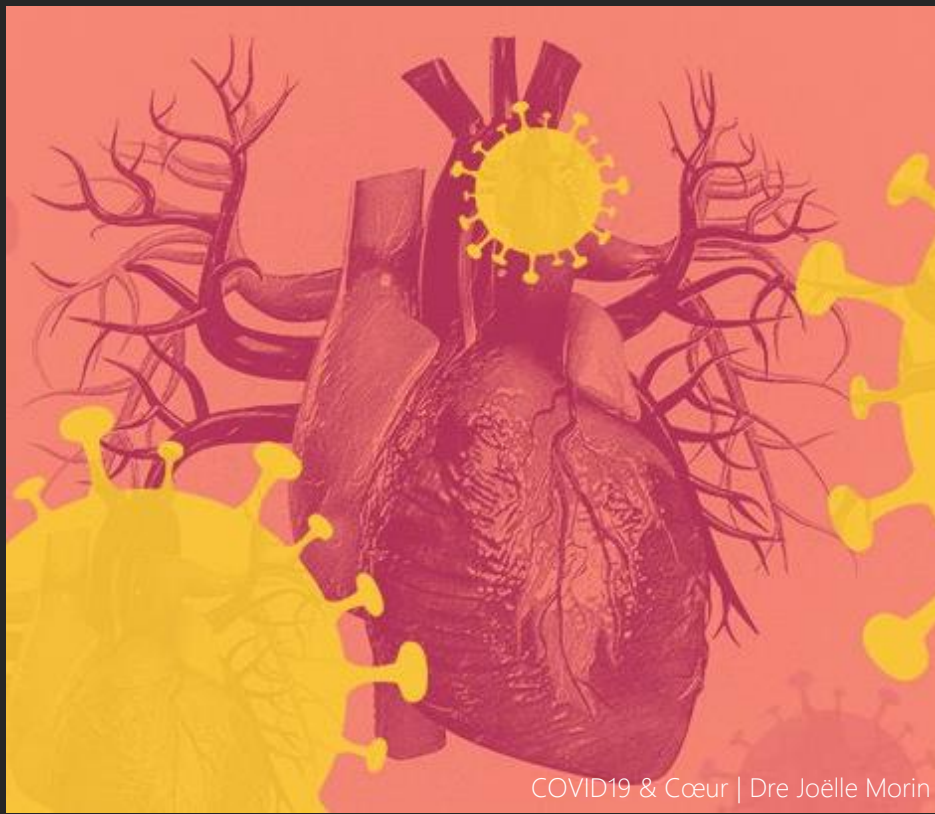
Table 2

Mechanisms of Arrhythmogenicity in Patients with COVID-19 Infection.

- Acute myocardial injury / Myocarditis
- Hypoxia
- Systemic inflammation
- Autonomic imbalance (SNS overactivity / virus-induced vagal nerve injury)
- Electrolyte abnormalities
- QT prolonging drugs (anti-COVID pharmacotherapies / AADs / other agents)
- Drug-drug interactions
- Cardiovascular comorbidities (hypertension, coronary artery disease, cardiomyopathy)

AADs = antiarrhythmic drugs; SNS = sympathetic nervous system.






Arythmies

Prévalence

59

Low prevalence of arrhythmias in clinically stable COVID-19 patients

Simone Sala MD¹ | Giovanni Peretto MD^{1,2}  | Giacomo De Luca MD^{2,3} | Nicola Farina MD^{2,3} | Corrado Campochiaro MD³ | Moreno Tresoldi MD⁴ | Lorenzo Dagna MD^{2,3} | Alberto Zangrillo MD⁵ | Simone Gulletta MD^{1,#} | Paolo Della Bella MD^{1,#}

“Snapshot” sur une journée du fardeau d’arythmie sur les unités non-intensives COVID-19 d’un centre tertiaire

132 patients inclus

12 patients avec arythmies (9%)

- 8 FA (4 paroxystiques et 4 permanentes)
- 12 TSVP
- Aucune prolongation QT > 450 ms
- Aucune arythmie ventriculaire

COVID-19 and cardiac arrhythmias

Anjali Bhatla, BA,^{*1} Michael M. Mayer, BS,^{*1} Srinath Adusumalli, MD, MSc,^{*}
 Matthew C. Hyman, MD, PhD,^{*} Eric Oh, MS,[†] Ann Tierney, MS,[†] Juwann Moss, BS,^{*}
 Anwar A. Chahal, MD, PhD,^{*} George Anesi, MD, MSCE, MBE,[‡] Srinivas Denduluri, PhD,^{*}
 Christopher M. Domenico, PharmD,^{*} Jeffrey Arkles, MD,^{*} Benjamin S. Abella, MD, MPhil,[§]
 John R. Bullinga, MD,^{*} David J. Callans, MD, FHRS,^{*} Sanjay Dixit, MD, FHRS,^{*}
 Andrew E. Epstein, MD, FHRS,^{*} David S. Frankel, MD, FHRS,^{*} Fermin C. Garcia, MD,^{*}
 Ramanan Kumareswaram, MD,^{*} Saman Nazarian, MD, PhD, FHRS,^{*}
 Michael P. Riley, MD, PhD,^{*} Pasquale Santangeli, MD, PhD,^{*}
 Robert D. Schaller, DO, FHRS,^{*} Gregory E. Supple, MD, FHRS,^{*} David Lin, MD, FHRS,^{*}
 Francis Marchlinski, MD, FHRS,^{*} Rajat Deo, MD, MTR^{*}

Tous les patients COVID-19 admis dans un même centre sur une période de 9 semaines

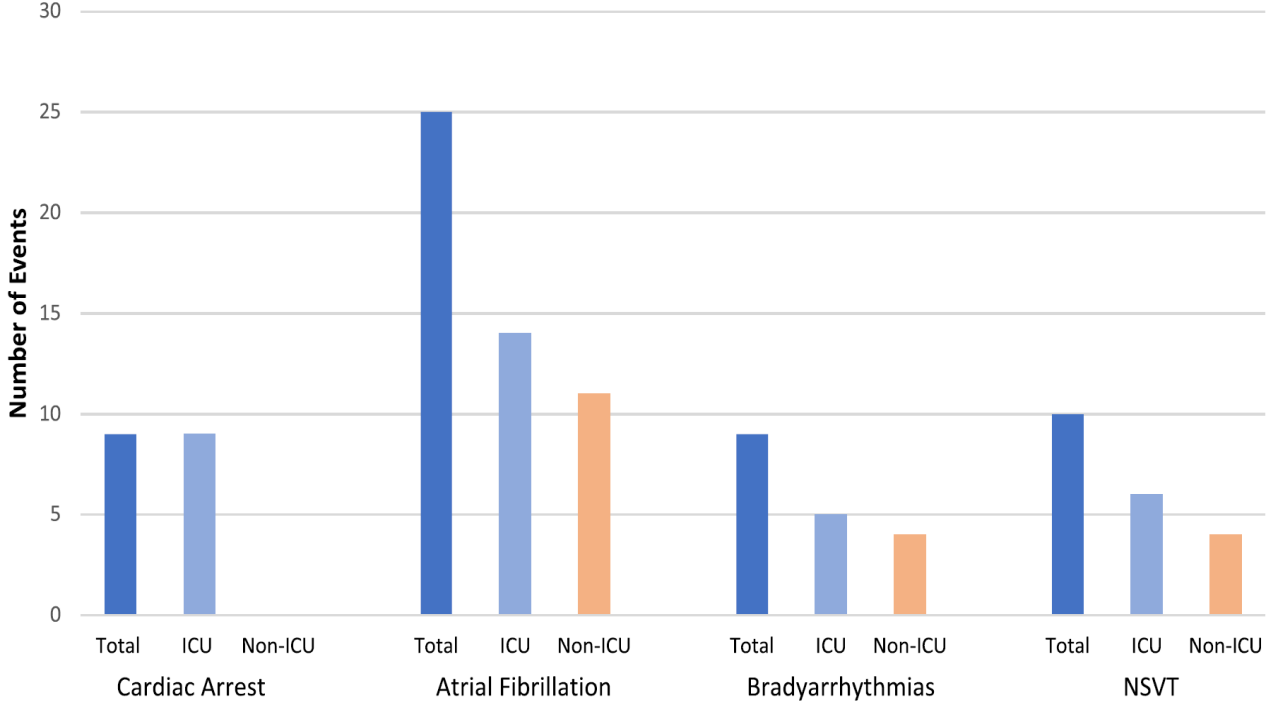
- 700 patients inclus
- 11% USI
- 4 % décès

Arythmies Prévalence

COVID19 & Cœur | Dre Joëlle Morin

Arythmies = 7.5 %

- 9 arrêts cardiorespiratoires (6 DEM, 2 asystolies, 1 TdP)
- 25 FA
- 9 brady-arythmies significatives
- 10 TVNS



Bhatla A et al. *Heart Rhythm* 2020;17:1439–1444

Les **facteurs de risque**
& **comorbidités** ♥ **vasculaires**
confèrent un risque augmenté
de souffrir d'une forme sévère
de COVID-19 ou d'en mourir.

La **mortalité** chez ces patients
est augmentée de 5 à 10 X (selon le
facteur de risque / la comorbidité)

01

La complication cardiaque
la plus fréquente du COVID-19
est le **dommage myocardique aigu**: est
associée de manière constante à un
moins bon pronostic.

Cependant **toutes les complications**
cardiaques confèrent un moins bon
prognostic.

03

Conclusion

02

Les **Rx antagonistes du SRAA**
n'ont pas été démontrés délétères
chez les patients souffrant
du COVID-19.

Les bénéfices prouvés de ces agents
dépassent les risques théoriques

04

Le **dosage des troponines**
doit être effectué avec discernement
pour éviter des investigations
potentiellement inutiles en découlant.



QUESTIONS ?



LA FIN

Aurevoir !