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CASE REPORT

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Cardiac tamponade in COVID-19 patients: Management and outcomes

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Abstract

Importance: Cardiac tamponade requiring emergent intervention is a possible complication of coronavirus disease 2019 (COVID-19) infection. Favorable clinical outcomes are possible if timely management and drainage are performed unless ventricular failure develops.

Observation: Cardiac tamponade in COVID-19, based on the limited reported cases, seems to be more common among middle-aged men with observed complications in black and ethnic minorities. Prognosis is worse amongst patients with concomitant ventricular failure.

Design and Methods: This is a case series of three COVID-19 patients complicated by cardiac tamponade, requiring surgical intervention at a single institution in New York.

Intervention: Pericardial window, Pericardiocentesis.

Outcome: One patient had recurrence of cardiac tamponade with hemorrhagic component but fully recovered and was discharged home. Two patients developed cardiac tamponade with concomitant biventricular failure, resulting in death.

Conclusion and Relevance: Cardiac tamponade with possible concomitant biventricular failure can develop in COVID-19 patients; incidence seems to be highest at the point of marked inflammatory response. Concomitant ventricular failure seems to be a predictor of poor prognosis.

KEYWORDS

COVID-19, pericardial effusion, tamponade, ventricular failure

Key points

Question: When should cardiac tamponade be suspected in COVID-19 patients?

Findings: Patients may have cardiac tamponade and cardiac failure on presentation, or develop tamponade during their hospital course. It is important to rule out cardiac tamponade in patients with COVID-19 experiencing cardiac arrest or worsening hemodynamic status. Up-trending inflammatory markers, signifying systemic inflammatory response, may be associated with cardiac tamponade and concomitant ventricular failure.

Question: What can be done to prevent cardiac tamponade and ventricular failure in patients with COVID-19?

Findings: While data is limited, systemic inflammation and hyper-coagulopathy constitute the underlying pathophysiology of

Abbreviations: ABG, PH, PCO_2, PaO_2, arterial blood gas, potential hydrogen, partial pressure of carbon dioxide, and partial pressure of oxygen; AKI, acute kidney injury; ARDS, acute respiratory distress syndrome; BMI, body mass index; COVID-19, coronavirus disease 2019; CXR, chest X-ray; ECG, electrocardiography; ECHO, echocardiogram; ECMO, extracorporeal membrane oxygenation; ED, emergency department; NSAIDs, nonsteroidal anti-inflammatory drugs.

disease in COVID-19 infection. Thus, therapeutic dose anticoagulation and anti-inflammatory medications could possibly play a role in treatment of patients with COVID-19. However, it is important to be cognizant of hemorrhagic sequalae and immunosuppressive side effects of these medications.

1 | INTRODUCTION

The World Health Organization declared the coronavirus disease 2019 (COVID-19) outbreak as a pandemic on 11 March 2020.[1] With its rapid spread across the globe, physicians have encountered a broad spectrum of early and late complications—mostly resulting from the underlying inflammatory process—including acute respiratory distress syndrome, acute kidney injury, and secondary infection.[2]

Mortality among hospitalized patients with COVID-19 has been reported to be as high as 17%, with the figure being 20.5% in patients undergoing elective surgeries.[3,4]

There is paucity of data regarding the cardiovascular effects of COVID-19. Arrhythmias, myocarditis, hypercoagulability, congestive heart failure, and, surprisingly, two cases of cardiac tamponade physiology, both resulting from hemorrhagic pericardial effusion, have been reported.[5-8] We further report three cases of COVID-19 complicated by cardiac tamponade requiring emergent surgical intervention.

2 | CASE SERIES

Case 1. A 48-year-old Hispanic male with a past medical history of type 2 diabetes mellitus, morbid obesity (body mass index [BMI]: 39.5 kg/m^2), with normal cardiac function and a known chronic small pericardial effusion of unknown etiology (not amenable to pericardiocentesis), presented with complaints of dyspnea and fatigue for 1 month, and acute deterioration for the past 24 hours. Nasopharyngeal swab was positive for COVID-19, serum troponins were within normal range, and arterial blood gas (ABG) analysis showed a pH of 7.26, partial pressure of carbon dioxide (pCO_2) of 83mm Hg, and partial pressure of oxygen (pO2) of 187mm Hg. Chest x-ray demonstrated clear lung fields and a large cardiac silhouette. Echocardiogram (ECHO) showed a moderate-to-large pericardial effusion with tamponade physiology (Figure 1).

He underwent emergent percutaneous drainage of 1500 mL of clear pericardial fluid which was sent for cytology (Table 1) and was started on therapeutic anticoagulation. On postoperative day (POD) 8, the patient developed acute cardiovascular decompensation. An ECHO showed a large pericardial fluid collection with signs of obstructive shock (Figure 2). He underwent another pericardial drainage with 900 mL of sanguineous fluid drained (no associated bacterial organisms on fluid culture), and was switched to prophylactic dose anticoagulation. Postoperative course was unremarkable, and patient was discharged 10 days after the second procedure in stable condition with drop in his inflammatory markers after Pericardiocentesis (Table 2).

Case 2. A 56-year-old Hispanic overweight (BMI of 27.1 kg/m^2) male with no significant medical history presented with a one-week history of cough, chest pain, fever and chills. Laboratory investigations showed a serum troponin-I level of 1.2 ng/dL and nasopharyngeal swab confirmed COVID-19 infection. Over the next few hours, the patient developed progressive hypotension. ECHO showed a large pericardial effusion with tamponade physiology (no images recorded in system, as it was an emergent bedside ECHO), and a left ventricular ejection fraction of 20%. He underwent emergent drainage of 400 mL of serous

TABLE 1 Pt 1 pericardial fluid cytology after first drainage

Component	Data
Amylase fluid	46
Appearance, body F	Clear
Baso fluid	0
Cells counted body	100
Eosinophils fluid	0
Fluid type	Pericardial fluid
Fluid type	Pericardial
Fluid type	Pericardial
Fluid type	Pericardial fluid
Fluid type	Pericardial fluid
Glucose, fluid	108
Lymphs fluid	50
Mesothelial fluid	11
Monocyte fluid	6
Nucleated cells fluid	198
PH, pleural fluid	8.9
Protein, fluid	5.0
Red blood cell cou.	555
Macrophage body	11
Color fluid	Yellow
Comment	No established
Neutrophils %, fluid	22
Fluid lining cells	0
Unidentified cells	0

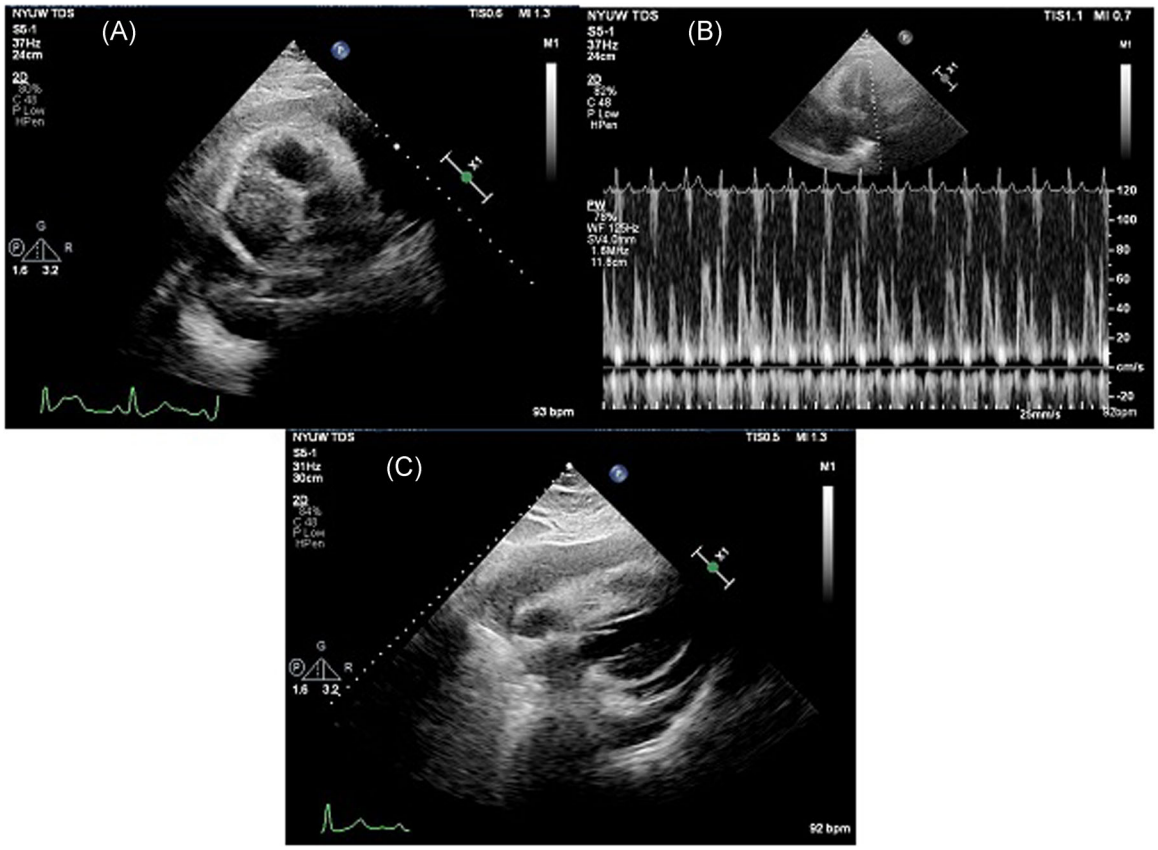

FIGURE 1 A, Top left, subcostal view: There is a large circumferential pericardial effusion. B, Top right, continuous wave Doppler through the mitral valve in the four chamber apical view: This image shows significant (>30%) respiratory variation in mitral inflow velocity. C, Bottom, subcostal view: There is a large circumferential pericardial effusion with visible partial collapse of the right ventricle and right atrium

pericardial fluid. On POD 0, the patient experienced cardio-pulmonary arrest and expired. It's presumed that that cause of death was cardiogenic shock due to severe ventricular dysfunction resulting from COVID-19 inflammatory storm or post-drainage pericardial decompression syndrome. 9) Inflammatory markers can be seen in (Table 3).

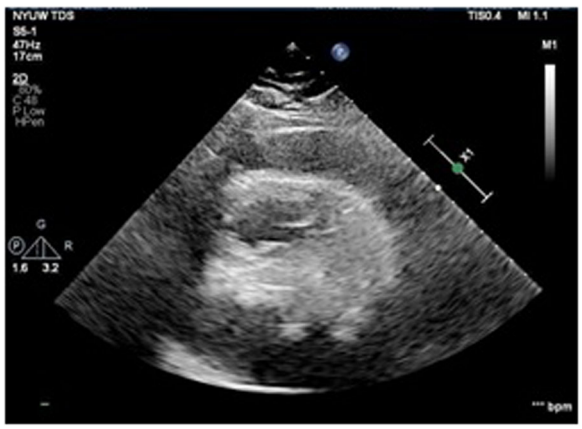

FIGURE 2 Subcostal view: There is a large circumferential pericardial effusion

Case 3. A 55-year-old African American male with a past medical history of hypertension and obesity (BMI of 30.5 kg/m^2) presented with 2 weeks of dyspnea, productive cough, myalgias, fatigue, fever, and chills. ABG showed a pH of 7.46, pCO_2 of 32mm Hg, and pO2 of 67mm Hg on 15 L of oxygen. Serum troponin-I was within normal range, serum creatinine was 2.3 mg/d, and nasopharyngeal swab was positive for COVID-19. CXR showed bilateral lung opacities and a mildly enlarged cardiac silhouette. He was started on prophylactic dose anti-coagulation, intubated on hospital day 2 for progressive hypoxia, subsequently on hospital day 5, required Veno-venous extra corporeal membrane oxygenation (ECMO) and was transitioned to prophylactic dose anti-coagulation. Laboratory investigations during hospitalization are seen in (Table 4). On hospital day 7, the patient developed pulseless electrical activity cardiac arrest. A bedside echocardiogram revealed a large pericardial effusion with tamponade physiology, and 800 mL of sanguineous fluid

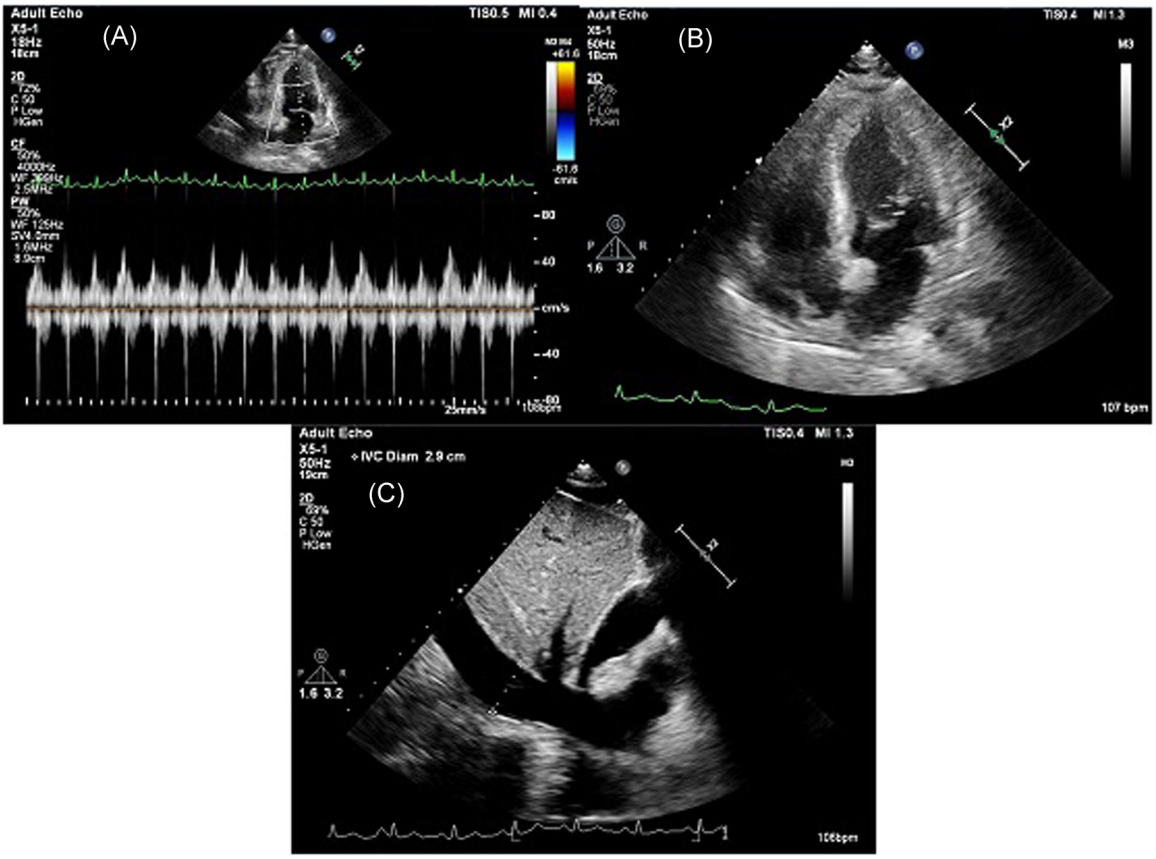

FIGURE 3 A, Top left, continuous wave Doppler through the mitral valve in the two chamber apical view: This image shows significant (>30%) respiratory variation in mitral inflow velocity. B, Top right, apical four chamber view: There is a moderate size effusion adjacent to the right atrium with partial right atrial collapse. C, Bottom, subcostal view: The inferior vena cava is plethoric measuring 2.9 cm

was drained percutaneously, with return of spontaneous circulation. On hospital day 8, due to increasing vasopressor and inotropic requirements, a repeat echocardiogram was performed which demonstrated biventricular failure, likely from post-drainage pericardial decompression syndrome (Figure 3). Further aggressive management was deemed futile, and all interventions were withdrawn per family's wishes; the patient underwent ECMO decannulation and was pronounced dead.

3 | DISCUSSION

COVID-19 was confirmed in a cluster of patients by the Chinese health authorities on 7 January 2020. The first case in the United States was reported on 20 January 2020[1] With its continued spread globally, a range of complications affecting almost every organ system have been reported.

Cardiac tamponade is most commonly idiopathic, with a smaller subset of cases resulting from infectious causes. It can be due to collection of transudate, exudate or blood in the pericardium.[9,10] Viral pericarditis is usually characterized by a gradual accumulation of transudate. Interestingly, in COVID-19 patients with cardiac tamponade, they can develop sanguineous, exudative or transudative pericardial fluid, as evidenced in our patients.[6,7]

The pathophysiology of COVID-19 cardiac tamponade is possibly a result of the marked systemic inflammatory response to the virus, leading to myocarditis and pericarditis. Pericardial drainage can be followed by severe pericardial decompression syndrome, leading to paradoxical hemodynamic instability and/or pulmonary edema following an otherwise noncomplicated pericardial drainage.[11] Myocardial ischemia, can also be explained by the hyper-coagulability (resulting from endothelial damage and the inflammatory response) in these patients.[12,13] Thus, prophylactic use of corticosteroids and therapeutic anti-coagulation have been proposed in patients with COVID-19.[14]

TABLE 2 Pt 1 inflammatory markers during admission

	04/15	04/16	04/17	04/18	04/19	04/20	04/21	04/22	04/23	04/24	04/25	04/26	04/27	04/28	04/29	04/30	05/01
24 h	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001	0001
Biomarkers																	
D-dimer quantitative	404		1.597	935	502		277		1.039		691	567		592	653	962	
C-reactive protein	19.74			151.86		37.86			35.20		32.97	15.06		31.25	15.03	10.84	
Erythrocyte sedimentati			35	57		49			3		3				7		
Lactate dehydrogenase	317		372	330	319	297			1.585		350	328			267		
AST	62	59	46:	38	42	40			1.592		1.290	374	133	83	68	69	69
ALT	229	184	104	65	60	62			1.964		3.581	2.563	1.602	1.090	834	626	427
Ferritin	22		61E		105	80	54		1.307		620	228			55		
Procalcitonin	<0.05								0.76		0.20			<0.05			
Troponini	<0.1	<0.1							5.0	3.3	0.9	0.6					
Creatine kinase total			84	82					134								
Triglycerides			84						72						78:		
White blood cell count	10.2	9.6	9.0	14.2	11.7	9.5	9.3	11.2	14.4		11.2	10.5	17.0	15.1	16.4	16.2	9.7
Neutrophils absolute	8.6	8.1	8.3			9.0		9.7	29.5				15.7		15.3		8.4
Lymphocytes absolute	0.6	0.4	0.2			0.2		0.6	0.5				0.3		0.2		0.5
Lymphocytes %	6	4	2			2		5	2				2		2		5
Platelet count	139	108	101	107	81	104	98	112	184		192+	186	226	218	242	245	191
Interleukin 6			<5														

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase.

TABLE 3 Pt 2 Inflammatory markers during admission

	03/28	03/29	03/30
24 h	0001	0001	0001
Biomarkers			
D-dimer quantitative		<202	
C-reactive protein		24.90	
Lactate dehygenase		240	
AST	26	55	24
ALT	26	65	12
Ferritin		1.008	
Procalcitonin		0.08	
Troponin I	1.2	0.5	
Creatine kinase total		110	
White blood cell count	5.5	4.5	
Neutrophils absolute	4.5	3.6	
Lymphocyte absolute	0.5	0.5	
Lymphocyte %	9	12	
Platelet count	95	82	

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase.

Cardiac tamponade should be suspected in COVID-19 patients with progressive hemodynamic compromise. An echocardiogram showing late diastolic collapse of the right atrium and early diastolic collapse of the right ventricle (occurring when the intra-pericardial pressure exceeds intracavitary pressure) is diagnostic.

After pericardial fluid drainage management is largely supportive, and recurrent cardiac tamponade is possible. While the use of therapeutic anticoagulation has been shown to improve prognosis in severe patients with COVID-19[14] the development of sanguineous cardiac tamponade (as seen in Case 1) may point towards increased risks of therapeutic anticoagulation after initial drainage. However, given the documented benefits of therapeutic anticoagulation in patients with COVID-19, particularly in patients with elevated D-dimers, it might be judicious to resume anti-coagulation 12 hours postoperatively, with a high index of suspicion for rebleeding in case of progressive worsening of hemodynamic status.[12,15]) The role of NSAIDs in this subpopulation is yet to be determined.[16,17] High suspicion for recurrence of cardiac tamponade is warranted as risk of recurrence is as high as 20%.[18]

While current data on cardiac tamponade, with possible concomitant biventricular failure, in patients with COVID-19 are limited, we hypothesize that short-term prognosis in this subpopulation is primarily dependent upon ventricular function at the time of development of tamponade, and also by development of post-drainage pericardial decompression syndrome. In our case series, the two patients with cardiac tamponade with concomitant biventricular failure (Cases 2 and 3) experienced rapid deterioration leading to death, while the patient with preserved ventricular function (Case 1) survived. However, the observed ventricular dysfunction—possibly stress cardiomyopathy or cytokine-related myocardial dysfunction—may just be a manifestation of the overall severity of inflammatory response and the associated fulminant cytokine release (Tables 1-3).[19] Further, the long-term prognosis, sequelae and predictors of survival in patients with COVID-19 developing transient myocarditis or cardiac tamponade remain yet to be observed.[20,21]

Importantly, surgical intervention and drainage of pericardial fluid in patients with COVID-19, while allowing for rapid relief from tamponade physiology, is associated with intra- and postoperative risks.

As mentioned in our study, all three patients who developed cardiac tamponade, a severely morbid complication, were of either African American, or Hispanic race. COVID-19 has been more deadly for African American and Hispanic people as seen in studies published in the US and the UK.[22]

4 | LIMITATIONS

Our study is limited by the small sample size, and inability to test pericardial fluid for COVID-19 antibodies and fluid composition.

5 | CONCLUSION

Cardiac tamponade is a rare complication of COVID-19 infection. Physicians must be cognizant of this possibility in patients with cardiovascular decompensation. Ultrasonography can aid in rapid diagnosis, and drainage of pericardial fluid and can result in clinical improvement. Short-term prognosis appears to depend upon ventricular function. Role of corticosteroids, NSAIDs, and immune modulators remains unclear in this subgroup of COVID-19 patients, but may have a role in preventing the development of ventricular failure caused by the marked inflammatory response and stress cardiomyopathy.

TABLE 4 Pt 3 inflammatory markers during admission

	03/25	03/26	03/27	03/28	03/29	03/30	03/31	04/01	04/02
24 h	0001	0001	0001	0001	0001	0001	0001	0001	0001
Biomarkers									
D-dimer, quantitative			15.212	11.119		43.874			
C-reactive protein			205.23	324.44		363.53	230.99		
Erythrocyte sedimentati			66	62		65			
Lactate dehydrogenase	Specim		2.445	2.303		1.997	1.941		
AST			Speci	143:		66	262		>6.600
ALT			90	77		55	133		3.306
Ferrtin			12.310	6.880		4.578			
Procalcitonin		0.94							
Troponin	0.4		0.3				0.4		
Creative kinase total	3.744		3.158	6.547	6.875	4.417	1.218		1.411
Triglycerides							479		
White blood count	10.7	12.3	14.1	15.6	22.0	27.6	27.9	28.2	31.4
Lymphocyte absolute cal	1.7	1.6	0.8	0.5	0.9	0.7			
Lymphocytes %	13	12	6	3	4	3			
Platelet count	255	236	231	228	200	141	92	60	51
Interferon gama				<5		<5		<5+	
Interleukin 10				44		39		128	
Interleukin 12				<5		<5		<5+	
Interleukin 13				<5		<5		<5+	
Interleukin-1 beta				<5		<5		<5+	
Interleukin 2 receptor (CD25) sol				524		See no		1.578+	
Interleukin 2				<5		<5		<5+	
Interleukin 4				<5		<5		<5+	
Interleukin 5				<5		<5		<5+	
Interleukin 6				130		See no	See no	See n +	
Interleukin 8				<5		<5		35	
Tumor necrosis factor-alpha				<5:		<5		<5–	

Abbreviations: ALT, alanine aminotransferase; AST, aspartate transaminase.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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