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Clinical Experiences of Multiple Organ Failure after Surgery for Acquired Cardiovascular Disease

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—Abstract—

A serious problem after cardiovascular surgery known as Multiple Organ Failure(MOF) whereby several vital organs successively demonstrate dysfunction in spite of intensive postoperative treatment has recently arisen.

We have made a retrospective study of the clinical records of 137 patients who underwent cardiovascular surgery during past two years (1987-1988).

Fourteen patients (10%) developed multi-organ failure postoperatively with the results of seven death (50%).

In fatal group, preoperative poor cardiac function (Cardiac Index≦2.0L / min. / ㎡) was considered important prognostic factor and infection & disseminated intravascular coagulation complicating gastrointestinal bleeding were the leading cause of death.

In conclusion, evaluation of multiple factors concerning multi-organ failure demonstrates preoperative poor functional preservation of vital organs is the main factor. So early diagnosis & management for each of the failing organs & prevention of infection are mandatory of the treatment of these critically ill patients.

Following major surgery for acquired cardiovascular disease, the cardiac output may be depressed initially but usually improves if the surgical correction has been successful.

In certain patients, particularly those with longstanding pulmonary hypertension or biventricular failure at rest, the cardiac output may remain depressed postoperatively despite satisfactory surgical correction.

The operative mortality in this group of patients has remained comparatively high in spite of continuing improvements in surgical technique & management. Past experience has been that many patients with persistent postoperative low cardiac output develop intractable heart failure and die in a low perfusion acidotic state within 24 to 36 hours after operation.

As improved methods of cardiopulmonary support have deen developed and utilized, many of patients in this group have survived beyond this time, some recovering, and others dying one to several weeks postoperatively from the effects of prolonged low cardiac output.

Theses cases have a clinical courses manifesting progressive vital organ failure in association with a persistent low cardiac output. So called multiple organ failure(MOF).

Recently we have been experienced postoperative

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low cardiac output status and sequential multiple system organ failure followed by increasing number of old aged patients and preoperative poor functional status of heart.

This report describes the clinical courses, concerning factors of multiple organ failure and the comparison between survival & fatal groups.

Method

(1) Materials(Table 1).

From Jan. 1987, to Dec. 1988, 137 patients underwent major cardiovascular surgery for acquired cardiovascular disease in our institute.

Among them, 14 cases of MOF were developed, so the incidence was about 10%.

After valve replacement : 10 cases, after redovalve replacement : 1 case, after operation of abdominal aortic aneurysm : 1 case, after pericardiectomy : 1 case, after removal of LA myxoma : 1 case, were developed.

In the case of LA myxoma, emergency operation was done due to sudden cardiac arrest resulted from mitral valve orifice obstruction by tumor.

We reviewed retrospectively the records of 14 patients who were managed intensively under the diagnosis of MOF.

Table 1. Operative cases of acquired cardiovascular disease(1987, Jan.−1988, Dec.)

Disease	Operation	Cases
Valvular heart disease	Valve replacement	95(10)
	Open commissurotomy	9
	Valve rereplacement	9 (1)
	Valve reconstruction	8
Annuloaortic ectasia	Bentall procedure	1
Ischmic heart disease	CABG	3
	LV aneurysmectomy	1
Aortic aneurysm	Graft replacement	6 (1)
Constrictive pericarditis	Pericardiectomy	4 (1)
Cardiac tumor	Removal of myxoma	1 (1)
Total		137(14)

( ) : cases of multiple organ failure.

(2) Definition of organ failure(Table 2-A, 2-B).

Multiple organ failure occurs when more than one organ system cannot support its activity spontaneously. Our criteria of organ failure is obtained from a review of the some clinical literature[5,7,10,15] because the criteria used to define organ failure is vary, arbitrary. Failure of cardiac system is defined as low cardiac index($\leqq$2.0L / min / m^2), the days of inotropic support($\geqq$7 days), and requirement of assist circulation(IABP).

The criterion to establish pulmonary failure is hypoxia that warranted respirator-assisted ventilation

Table 2-A. Defintion of organ failure

Organ	Criterion
Heart	C.I.$\leqq$2.0 L / min / m^2
	Catecholamine(+)$\geqq$7 days
	IABP (+)
Lung	Respirator (+)$\geqq$5 days
Liver	Bilirubin$\geqq$3 mg / dl
	SGOT$\geqq$100 units
Kidney	Bun$\geqq$50 mg / dl
	Creatinine$\geqq$3 mg / dl
	Dialysis (+)

*C.I. : Cardiac Index.
IABP : Intra Aortic Balloon Pump.

Table 2-B. Definition of organ failure

Organ	Criterion
Coagulation (DIC)	Platelet $\leqq$ 70,000 / mm^3
	Fibrinogen $\leqq$ 150 mg / dl
	FDP $\geqq$ 40 μg / ml
Gastrointestinal	Ulcer (confirmed)
	Hematemesis, melena
Brain	Painful response only to stimuli

*DIC : Disseminated Intravascular Coagulation.
FDP : Fibrin Degradation Products.

for at least 5 days postoperatively or until death.

Hepatic failure is defined as a serum bilirubin level greater than 3㎎/dl with elevation of the SGOT level greater than 100 units.

Renal failure is defined as an elevation of BUN (≧50㎎/dl), Creatinine (≧3㎎/dl) and requirement of peritoneal dialysis or hemodialysis.

Failure of coagulation system(Disseminated Intravascular Coagulation) is defined as low platelet count(≦70,000/㎣), low fibrinogen level(≦150㎎/dl) and high FDP level(≧40㎍/ml).

Gastrointestinal bleeding is an endoscopic confirmation of ulcer with hematemesis or melena.

CNS failure is defined as painful response only to stimuli.

We consider it as an organ failure in case of more the one item in each organ.

Results

Of the 137 patients examined in this study, 14 cases were diagnosed as MOF.

The age of patients ranged from 32 to 67 years.

The majority were in 5th decade of life and male against female was 4:3.

In our 14 cases of MOF, 7 cases survived and 7 cases expired.

So we analized and compared two groups each other.

Among survival 7 cases(Table 3), valvular heart disease were 6 cases and one case was abdominal aortic aneurysm.

Age was variable but 5th decade was most frequent. Preoperative functional class was poor, almost all cases belonged to NYHA functional class Ⅲ or Ⅳ.

In cardiac index, majority of cases were above. 2.0L/min/㎡ but only one case was below 2.0 L/min/㎡.

Postoperative inotropic support needed from 7 days to 27 days.

But circulatory assist device(IABP) used in 2 cases, which C.I. were very poor(Case No.2:1.87 L/min/㎡, Case No.5:2.07L/min/㎡).

Table 4 showed fatal cases of MOF.

Five cases were valvular heart disease, one case was Lt. atrial myxoma and another one case was constrictive pericarditis.

Age incidence and preoperative functional class were similar to survival group.

But preoperative Cardiac Index was worse than survival group. Majority of cases were below 2.0 L/min/㎡ and postoperative inotropic supportive days were much longer than survival group. Circu-

Table 3. Survival cases of multiple organ failure

Cases	Age/Sex	Diagnosis	Operation	Preoperative		Postoperative
				NYHA Class	C.I.(L/min/m²)	Inotropic support
1.	40/F	MSR+TR +GLA	MVR+TAP +LA plication	Ⅳ	2.68	14 days
2.	43/M	MSR+TR +GLA	MVR+TAP +LA plication	Ⅳ	1.87	27 days IABP (+)
3.	41/M	MSR+TR	MVR+TAP	Ⅲ	2.45	10 days
4.	34/M	PVF(M)+TR	RedoMVR+TAP	Ⅲ	2.44	7 days
5.	46/F	MSR+ARS	MVR+AVR	Ⅳ	2.07	21 days IABP(+)
6.	41/M	MSR+ARS	MVR+AVR	Ⅳ	2.50	7 days
7.	67/M	Ao. aneurysm (Abdominal)	Graft replacement	Ⅲ	2.42	(−)

*GLA : Giant Lt. Atrium PVF : Prosthetic Valve Failure.

latory assist device(IABP) was used in 4 cases.

At the same time, degree of preoperative pulmonary hypertension was higher than survival group.

So we thought that longstanding pulmonary hypertension was an another important prognostic factor of postoperative hemodynamic findings.

In survival group(Table 5), duration of respiratory support was from 5 days to 15 days.

Hepatic failure was noted in all cases and ++ grade failure, which means that elevation of serum bilirubin was over 10mg / dl, was noted in 2 cases.

Renal failure was noted in 4 cases and its degree was all ++ grade failure, which were managed with peritoneal dialysis and hemodialysis.

DIC was noted in 1 case, which was managed with platelet transfusion and heparin therapy.

Sepsis was noted in 1 case, causative organism was Nonhemolytic streptococci.

Compared with survival group, fatal group(Table 6) showed severe degree of hepatic & renal failure.

Almost all cases showed ++ grade organ failure.

And also, incidence of DIC & Sepsis was higher than survival group.

Causative organisms were revealed Pseudomonas, Klebsiella, Serratia species.

Severe hepatic & renal failure with superimposed DIC & sepsis was revealed the leading cause of death in our series.

Table 4. Fatal case of multiple organ failure

Cases	Age / Sex	Diagnosis	Operation	Preoperative		Postoperative	
				NYHA Class	C.I.	Inotropic	support
1.	47 / M	MS+AS	MVR+AVR	Ⅳ	1.38	7 days	IABP (+)
2.	43 / F	MR+AR	MVR+AVR	Ⅳ	2.0	25 days	
3.	55 / F	MS+AS	MVR+AVR	Ⅲ	1.72	24 days	IABP (+)
4.	44 / F	MSr+Tr	MVR+TAP	Ⅲ	1.44	13 days	
5.	52 / M	MS+COPD	MVR	Ⅳ	1.50	17 days	IABP (+)
6.	32 / F	LA Myxoma	Removal	Ⅳ	2.0	7 days	
7.	45 / M	Constrictive pericarditis	Pericar-diectomy	Ⅲ	1.75	50 days	IABP (+)

*COPD……Chronic Obstructive Pulmonary Disease

Table 5. Grade of multiple organ failure(survival cases)

Case No.	Respiratory support	Hepatic failure	Renal failure	CNS failure	G−I bleeding	DIC	Sepsis (organism)
1.	14 days	+	++	drowsy	−	+	−
2.	15 days	++	++	drowsy	−	−	−
3.	7 days	+	++	−	−	−	−
4.	5 days	+	−	−	−	−	−
5.	14 days	+	−	−	−	−	−
6.	5 days	++	−	−	−	−	−
7.	15 days	+	++	drowsy	−	−	+
					−	−	Non-hemolytic Streptococci

*++……Need Dialysis in Renal failure
Bilirubin≧10 mg / dl in Hepatic failure.

Table 6. Grade of Multiple organ failure(fatal cases)

Case No.	Respiratory support	Hepatic failure	Renal failure	CNS failure	G-I bleeding	DIC	Sepsis (organism)
1.	7 days	++	++	Drowsy	−	−	−
2.	25 days	+	++	−	−	−	+ (Pseudomonas)
3.	24 days	+	++	Comatous	+	+	−
4.	13 days	++	++	Drowsy	−	+	−
5.	17 days	++	++	Drowsy	−	−	+ (Pseudomonas)
6.	7 days	++	+	−	−	−	+ (Klebsiella)
7.	50 days	++	++	Drowsy	−	+	+ (Serratia)

*++……Need dialysis in renal failure.
Bilirubin≧10 mg / dl in Hepatic failure.

Table 7. MOF by organ system

Lung : 14 / 14 (100%), Liver : 14 / 14 (100%),
Kidney : 11 / 14 (78.6%), DIC : 4 / 14 (28.6%)
Brain : 2 / 14 (14.3%), G-I tract : 1 / 14 (7%)

Table 8. Incidence & Mortality of MOF

No. of organ failures	Cases	No. of Death	Mortality (%)
2	3	0	0
3	7	4	57.1
4	3	2	66.7
6	1	1	100
Total	14	7	50.0

MOF by organ system was revealed in Table 7.

Lung, Liver, kidney were the major failing organs and stress gastro-intesitnal bleeding was the least frequent failing organ.

Table 8 showed incidence and mortality of MOF.

The mortality of two organ system failure was zero, while the mortality of three organ system failure was 57.1%, that of four organ system failure was 66.7%, and that of six organ failure was 100 %.

The mortality increased by the increasing number of organ failure. So the overall mortality was 50%.

All MOF cases were revealed hepatic failure. So the course of hepatic failure was reviewed.

In survival group(Table 9), 2 cases showed ++ grade failure but 5 cases showed ++ grade failure in fatal group(Table 10).

Onset of hepatic failure was variable from POD lst to POD 12th. But maximum level of Bilirubin & SGOT was revealed at 3-4 days after onset in majority of cases.

There was no difference between survival & fatal groups.

Maximum level of bilirubin was much higher in fatal group compared with survival group.

But maximum level of SGOT was no difference between two groups.

In survival group, all cases were returned to normal range with general supportive care within

Table 9. Course of hepatic failure(survival cases)

Case No.	Onset	Maximum Level		
		Bilirubin (mg / dl)	SGOT (U / L)	SGPT (U / L)
1.	4 POD	5.7	60	75
2.	1 POD	14.6	196	86
3.	4 POD	3.7	187	105
4.	2 POD	5.0	108	52
5.	1 POD	8.3	143	97
6.	1 POD	10.8	112	48
7.	12 POD	8.4	84	84

*POD……Post Operative Day.

Table 10. Course of hepatic failure(fatal cases)

Case No.	Onset	Maximum level		
		Bilirubin (mg / dl)	SGOT U / L)	SGPT (U / L)
1.	3 POD	13.2	185	127
2.	12 POD	6.2	85	60
3.	7 POD	3.6	63	23
4.	1 POD	21.2	122	98
5.	1 POD	13.7	159	132
6.	4 POD	11.4	179	126
7.	1 POD	23.6	146	106

*POD……Postoperative Day

2 weeks.

But in fatal group, hepatic failure was progressively deteriorated despite general supportive care.

So the plasmapheresis was applied in 2 cases, which showed the level of bilirubin over 20mg / dl, but there's no effect.

Other literatures[5,11], also commented that there's no effective management for severe hepatic failure.

We thought that it was serious problem for management of severe, progressive hepatic failure.

Course of renal failure was also reviewed.

In survival group(Table. 11), renal failure was noted in 4 cases.

These cases showed high elevation of BUN, Creatinine. But anuria was noted in only 1 case and its duration was short.

These cases were managed with peritoneal dialysis & hemodialysis.

Mean the while, the course of renal failure was severe in fatal group(Table 12).

Six cases showed anuria and its duration was from

Table 11. Course of renal failure(survival cases)

Case No.	Anuria		Maximum level		Dialysis
	Onset	Duration	BUN (mg / dl)	Creatinine (mg / dl)	
1.	6 POD	1 day	122.1	3.0	HD
2.	–	–	155.8	5.3	PD+HD
3.	–	–	160.2	4.7	PD+HD
4.	–	–	29.7	1.8	–
5.	–	–	58.1	1.8	–
6.	–	–	47.4	1.5	–
7.	–	–	100.7	3.6	HD

*PD : Peritoneal Dialysis HD : Hemodialysis POD : Post Operative Day.

Table 12. Course of renal failure(fatal cases)

Case No.	Anuria		Maximum level		Dialysis
	Onset	Duration	BUN (mg / dl)	Creatinine (mg / dl)	
1.	6 POD	1 dya	87.8	4.2	PD+HD
2.	14 POD	11 days	150.0	5.5	PD+HD
3.	13 POD	11 days	117.4	4.8	PD+HD
4.	10 POD	3 days	106.7	5.3	PD+HD
5.	13 POD	4 days	99.5	5.5	PD+HD
6.	–	–	68.1	2.4	PD
7.	40 POD	10 days	74.9	4.2	PD+HD

*HD……Hemodialysis PD……Peritoneal Dialysis

1 day to 11 days.

Maximum level of BUN, Creatinine was much higher than survival group and all cases were managed with peritoneal dialysis & hemodialysis.

But every cases failed.

Discussion

Patients with longstanding pulmonary hypertension and right heart failure and those with biventricular failure at rest frequently will manifest a low cardiac output syndrome preoperatively.

This may be associated with depressed renal and hepatic function, presumably on the basis of reduced perfusion, and further contribute to the poor risk status of the patient for open heart surgery.

Subsequent hypoxic injury as a result of a low perfusion state, either during or following cardiovascular surgery, will result in succesive dysfunction of several vital organs in spite of intensive postoperative management[8,19].

Recently considerable effort is required to solve the patho-physiology of vital organ failure.

It is due to the advanced management of critically ill patients and the developement of clinical investigation for dysfunction of various organs.

Progress in the development of organ support systems has created the clinical syndrome ; Other investigators have termed multlple organ failure (MOF)[2,6,10].

In this report, we studied the preoperative hemodynamic status and postoperative dysfunction of various organs at the viewpoint of MOF after surgery for acquired cardiovascular disease.

Incidence of MOF after emergency surgery for trauma patients has been reported 6.9% by Fry and associates[7], 7.7% by Borzotta and associates[5]. While the incidence of MOF after open heart surgery has been reported slightly high, 8.9% by Koyanagi and associates[11] & 15% by Mori and associates[15].

In general, incidence of MOF after open heart surgery has been reported below 10% by Japanese Association of Thoracic Surgery(1986).

In our country, exact incidence has not been reported. But our incidence of MOF after surgery for acquired cardiovascular disease was revealed about 10%. It was similar to other reported series in JAPAN.

If patient had fallen in MOF, mortality has been reported very high (70-80%) by other reports.[6,7].

After open heart surgery, it has been reported 40-70% by Japanese Association of Thoracic Surgery (1986).

Mortality of our series was revealed 50%.

Many other clinicians[2,6,7].has been reported that the mortality from failure of two organ system was 53-80%, three organ system failure was 79-96%, four organ system failure was 83-100%.

The mortality increased by the increasing number of organ failure.

In our series, it was similar to above report.

So we could hypothesize that mortality would cumulatively rise with the numbers of failing organs involved.

Although the mechanism for the development of hepatic failure as a consequence of prolonged low cardiac output is not completely understood, decreased hepatic perfusion and decreased portal venous flow have been cited as primary etiologic factors[12,16,17,18].

Longerbeam and associates[13]have ascribed that support of the systemic blood pressure with vasopressors under these conditions further decreased hepatic perfusion and hastens liver damage.

Although hepatic failure has not been frequently described as a complication of open heart surgery, renal failure as a result of acute tubular necrosis has[20].

Prolonged hypotension, acidosis, excessive hemolysis, low flow rates during E.C.C. and prolonged perfusion times have been involved as etiologic agents in causing acute tubular necrosis following open heart surgery.

Oliguria is an early manifestation of low cardiac

output occuring after open heart surgery but early death often precludes the development of acute tubular necrosis and renal failure[20].

Prolonged low cardiac output associated with decreased renal perfusion results in progressive renal tubular damage.

Metabolic acidosis resulting from prolonged low cardiac output additionally may enhance the renal tubular damage[16].

Baue and Associates[3]have described that hepatorenal syndrome is a clear example of the organ associative phenomenon in which failure of one organ contributes to failure in another.

In the patients with severe liver disease there is often high cardiac output or hyperdynamic circulation with low peripheral vascular resistance which tends to shift blood flow away from the kidney.

These patients also have secondary hyperaldosteronism because the sick liver cannot breakdown aldosterone. This may decrease renal cortical blood flow.

The simultaneous appearance of both renal and hepatic failure suggests a common etiologic factor, which is decreasing hepatic and renal perfusion with subsequent ischemic damage.

It is apparent that the effectiveness of cardiovascular support in the postoperative period must be sufficient to provide adequate perfusion to vital organs to gain survival[4].

Another organ-associative phenomenon is the development of high pulmonary vascular resistance in patients with acute respiratory failure[21].

High right sided cardiac pressure increases the venous pressure of the kidney and liver and may suggest the need for decreased volumes of fluid, whereas left sided cardiac pressure may actually be low and cardiac output could be decreasing.

Jenkins and associates[9]had been reported that persistent low cardiac output and high CVP over 24 hours after open heart surgery were very harmful factors for function of liver and kidney.

Recognition of this phenomenon will allow the surgeon to better support the circulation.

Knowledge of organ-associative phenomena and the relationships of one system to another alert us in certain circumstances to the need for support of various specific organs.

Use of this knowledge may allow interruption of the cycle of deterioration.

Considerable effort is required to support failing organ systems in these critically ill patients[7].

Hemodilaysis may be required in the patient with renal failure to control hyperkalemia, metabolic acidosis and rising BUN & Creatinine values.

Volume respirator and positive end expiratory pressure(PEEP) are often necessary to treat progressive hypoxemia.

Uncontrolled stress hemorrhage may necessitate emergency operation that can be futile.

The high mortality attending organ failure has prompted many efforts to prevent organ dysfunction by specific prophylactic maneuver. Antacid therapy for stress gastrointestinal bleeding has been used effectively in reducing incidence of stress bleeding.

The best prevention or treatment for MOF requires correction of the fundamental pathophysiologic feature that has disrupted homeostasis.

In addition, better control of surgical infection or post-operative infection is essential for preventing MOF and fatal outcome.

Many other investigators[6,7]had been reported that relationship between MOF & infection was demonstrated 69%-89%. Unfortunately,.MOF with sepsis in our series had invasive pulmonary infection as the primary septic source. Infection in the lung is not amenable to drainage and debridement in the traditional sense.

These patients underscore that improved fundamental understanding of the host-pathogen and organ system pathophysiologic features are required before means of intervention can be improved further. Baue and associates[3] had emphasized that host resistance or the ability of an individual to resist infection is depressed after injury or operation

by a number of factors.

First factor is circulating immunosuppressive factor after injury or operation[1].

Second factor that alters host resistance is shock or hypotension, resulting in depression of the reticuloendothelial system and depression in white cell function.

Third factor is nutritional deficiency which is now recognized as being associated with depression of both T & B lymphocyte function. All these factors tend to decrease resistance in the operated patient[14].

Recent studies have demonstrated that stimulation of non-specific host defenses may have promise in providing a means of attacking uncontrolled infection. Until such time as additional means become part of the therapeutic armamentarium, application of surgical drainage, debridement and judicious antibiotic therapy remain the mainstays both prevention and treatment of postoperative organ failure.

Although we must strive to provide better means of support for organs that have failed, especially long-term support of failed kidneys, lungs, circulatory and other organ systems, the primary approach is prevention of multiple systems failure during the initial phase of treatment, operation or injury.

Baue and associates[3] had emphasized that a preventive attack on this syndrome requires increased understanding in four areas;

1) Definition and recognition of the problem.

2) Understanding the setting and circumstances in which multiple or sequential organ failure occurs.

3) Understanding the relationships between the various systems that can lead to the domino effect of one system trigerring problems in another.

4) Prevention of multiple systems failure by support of organs and systems before they fail.

He concluded that our ultimate goal must be to prevent organ failure and, in particular, to prevent the sequence of multiple organ failure so that this syndrome will gradually be decreased in frequency and severity.

If we are able to do this, increased numbers of patients will survive and fare better.

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