

MULTIPLE ORGAN FAILURE AS A COMPLICATION IN INTENSIVE CARE PATIENTS

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Introduction

Multiple organ failure is a clinical syndrome characterized by functional deterioration of two or more organs or organ systems. The most frequent cause of multiple organ failure is inflammatory process - sepsis, but noninfectious causes are also possible: burns, severe pancreatitis, and politrauma. Development of multiple organ failure in intensive care units is as a rule a complication of such inflammatory or noninflammatory events. This clinical syndrome is a continuing challenge to intensivists, because only its appropriate management gives the acknowledgment to the settings in which it occurs. However, despite advanced technology in intensive care units, possibilities of management of multiple organ failure are limited, and are mostly related to supportive therapy. The success of multiple organ failure management is associated with the number of failing organs - if more organs fail, less is the likelihood of patient survival.

Pathophysiology

It has been repeatedly stated that sepsis is the most frequent cause of organ function deterioration. Monocyte stimulation by exotoxins and endotoxins results in the release of proinflammatory cytokines - mostly tumor necrosis factor alpha and interleukin 1, increased susceptibility of monocytes to aggregation on the endothelium, and enhanced release of other inflammatory and anti-inflammatory cytokines. Stimulation of platelet aggregation and thrombin formation results in thrombosis in microvasculature and impaired circulation in individual organ or organ system. This pathophysiological process causes a cascade of events in multiple organ failure.

Patients and methods of measurement

Patients with multiple organ failure are characterized by impaired function of an individual organ or organ system. As a rule, numerical evaluation of organ functions is avoided, due to a high likelihood of misinterpretation of the degree of failure. Scoring systems to obtain information on clinical condition and the outcome are much more frequently employed. The use of scoring systems reduces the risk of error in the assessment of organ function, and increases the knowledge about the entire condition. Enclosed are the tables of some frequently used scoring systems, i.e. APACHE 2, MOF score, and SOFA score. Place and role of scoring systems will be discussed elsewhere, but their role in organ function assessment is helpful because they consider the function of organs as a whole, and not in isolation.

However, along with the utilization of scoring systems, parameters of individual organ systems must be observed. It should be emphasized that prior to individual numerical indicators determination, clinical examination is obligatory and of major importance - from inspection to further physical methods of gaining insight into a patient's clinical condition.

Respiratory function is assessed by rate of respiration, blood gas analysis, oxygen saturation, and capnometry. Radiological follow-up, whether by x-ray or CT findings, provides additional information on lung parenchyma.

For assessment of circulation, in addition to clinical examination we employ noninvasive blood pressure and pulse measurements, central venous pressure and pulmonary capillary pressure measurements and vasoactive therapy.

Central nervous system is most often assessed by Glasgow Coma Score. Electroencephalography can provide additional information on central nervous system function and possible focal lesions. Along with the assessment of central nervous system function, the importance of evaluation of peripheral neurological disorders should be stressed. The latter is performed by clinical examination, and in case of mild impairments by electromyography.

Hepatic function is assessed by measurement of biochemical parameters, bilirubin, transaminases, liver enzymes, and by monitoring of coagulation and hematologic parameters. Again, clinical examination and noninvasive methods like ultrasonography provide important additional information. Viral markers levels are of major importance because they point to possible pre-existing chronic hepatic impairment, which has essential repercussion on the recovery of liver function and survival in case of acute exacerbation. It should be stressed that irreversibly damaged hepatic tissue has no chances of recovery, and that levels of biochemical markers will be permanently below the expected ones, which might mask the degree of damage and chances of survival.

In addition to clinical parameters, renal function can be monitored by measuring azotemia, electrolytes, acid-base status, and hematologic parameters. Red blood count, calcium, and phosphate values give important information for differentiation of chronic from acute organ failure. In general, azotemia with normal red blood count suggests acute renal failure, because the period for loss of erythropoietin and development of renal anemia is as a rule longer. However, decreased red blood count does not rule out acute renal failure, because other factors, primarily bleeding and infections, can contribute to the development of anemia. Decreased calcium and increased phosphate levels generally point to chronic azotemia.

Measurements of individual hematologic, biochemical, and other parameters gain additional value in the course of their comprehensive monitoring and checking in time units.

Discussion

Multiple organ failure is a clinical syndrome representing a great challenge to intensive care units personnel. Organ failure most commonly occurs in hospital settings, and it is particularly frequent in intensive care units. The development of this syndrome is more common in surgical units as a consequence of septic complications, but it is also encountered in medical units in conjunction with heart failure, diverse shock conditions, poisonings, pancreatitis etc. Despite major technological advances, possibilities of management of this syndrome remain limited technically and pharmacologically. Management of the underlying process which led to multiple organ failure is of utmost importance. Only the control of underlying process, most often sepsis, offers chances for successful management of multiple organ failure. Choice of method in management of organ failure, and likelihood of favorable outcome, are frequent concerns. Most often, the selected method of replacement of failing organ function is of less importance, more important being the control of the process which led to the failure. This can be applied to the choice of vasoactive therapy (dopamine, dobutamine, dopexamine, norepinephrine), to the choice of renal function replacement (continued versus intermittent procedures of depuration), and choice of ventilation (noninvasive versus invasive). It is not disputable that certain techniques or drugs have advantages in particular conditions, but the truth is that control over the process which resulted in organ function deterioration is of major importance. Since the main cause of multiple organ failure is sepsis, only the control of burning sepsis allows successful management of multiple organ failure. As a rule these are inflammatory processes caused by pseudomonas, acinetobacter, and staphylococci, often methicillin resistant. An uncommon event, when primary bacterial inflammation is not the

cause of multiple organ failure, is initially chemical inflammation - pancreatitis, and large burns. However, pancreatitis and burns are often intertwined with bacterial superinfection which additionally increases the risk of multiple organ failure.

The number of failing organs essentially affects the outcome. The higher the number of failing organs, the higher is mortality. However, it should be stressed that recovery of an individual organ system does not guarantee survival, because only control of the underlying process allows better prognosis. Major issues in the management of multiple organ failure are prevention of sepsis by control of infections in intensive care units, and appropriate choice of antibiotic therapy for frequently resistant hospital strains.

APACHE II

	4	3	2	1	0	1	2	3	4
temp	<30	30-31.9	32-33.9	34-35.9	36-38.4	38.5-38.9		39-40.9	>41
MAP (S+2D)/3	<50		50-69		70-109		110-129	130-159	>160
pulse	<40	40-54	55-69		70-109		110-139	140-179	>180
resp	<6		6-9	10-11	12-24	25-34		35-49	>49
PaO ₂ ,(A-a)*	<7.3	7.3-8		8.1-9.3	>9.3		26.7-46.6*	46.7-66.6*	>66.7*
pH	<7.15	7.15-7.24	7.25-7.32		7.33-7.49	7.5-7.59		7.6-7.69	>7.7
bicarb.	<15	15-17.9	18-22.9		23-31.9	32-40.9		41-51.9	>52
Na	<111	111-119	120-129		130-149	150-154	155-159	160-179	>180
K	<2.5		2.5-2.9	3-3.4	3.5-5.4	5.5-5.9		6-6.9	>7
creatinine			<53		53-132		133-175	176-299	>300
Htc	<20		20-29.9		30-45.9	46-49.9	50-50.9		>60
L	<1		1-2.9		3-14.9	15-19.9	20-39.9		>40

* (by FiO₂ >50 %) APS:

(best response)	1	2	3	4	5	6
Eyes opening	none	to pain	to voice	spont.		
Motor response	none	extension	flexion	withdraws	local pain	obeys c.
Verbal response	none	incomprehen- sive words	inadequate words	confused	oriented	

GSC:

	0	2	3	5	6
Dob	<44	45-54	55-64	65-74	>75

Kronična obolenja	2	5 (pri urgentni kirurgiji)
Ciroza jeter		
NYHA IV		
Težka COBP		
Kronična dijaliza		
Imunocomprommisation		

APACHE II = APS () + (15 - GCS ()) + Age () + chron. illness () =