Intensive Care Med (1988) 14:575–577

Intensive Care Medicine

Bacteremia following cardiac arrest and cardiopulmonary resuscitation

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Received: 15 January 1988; accepted: 8 February 1988

Abstract. After but of hospital CPR thirty three resuscitated patients were studied for bacteremic complications. Thirteen patients (39%) had two or more positive blood cultures during the twelve hours following CPR. Source of superinfection was a central venous catheter in one case (staphylococcus). The twelve other bacteremic patients had fetid diarrhea a few hours after admission. The same organism were found in blood and faeces (streptococcus D, Escherichia coli, Pseudomonas aeruginosa, acinetobacter, enterobacter). Mesenteric ischemia caused by a low cardiac output may explain the diarrhea and the intestinal origin of the septicemia. All patients (12 cases) with diarrhoea and bacteremia died. Patients who recovered without neurologic sequelae (4 cases) had never been septic and never had diarrhea.

Key words: Cardiac arrest – Cardiopulmonary resuscitation – Bacteremia – Diarrhea

Improvement in and wider application of cardiopulmonary resuscitation (CPR) has resulted in an increasing number of patients resuscitated after cardiac arrest (CA) [1]. As a consequence CPR sequelae and complications are more often reported [2]. Neurologic (anoxia) and mechanical (external compression) complications are frequently described [3, 4]. Soppi et al. [5] also describe impaired cell mediated immunity. Prior to this study, we had sometimes observed early after CA and CPR, fetid diarrhoea and septicemia, furthermore the patients with diarrhoea often died.

The aim of this prospective study was to study the relationship between CA, diarrhoea, bacteremia and death.

Patients and methods

Thirty three consecutive patients were studied between January 1983 and September 1985 (30 male, 3 female, mean age, 55±15 years).

All had CA and CPR out of hospital and were transfered within one hour to our intensive care unit.

Asystole (24 cases) and ventricular fibrillation (9 cases) were caused by myocardial disease (16 cases), inhalation (7 cases), idiopathic (4 cases), intoxication (3 cases), drowning (2 cases) and electrocution (1 case).

Estimated arrest time and CPR time were respectively 18±11 min and 6±4 min.

At the time of hospital admission samples were taken to white blood count (WBC) and three blood cultures (aerobic, anaerobic) were taken during the twelve hours following admission (first hour, second hour and later). Blood culture samples were drawn by venepuncture, not through catheters. Patients were treated with antibiotics as soon as the blood culture became positive. Venous catheters used for the CPR were removed and all intravascular tips were sent for bacteriological culture. New lines were inserted in the internal jugular vein. The nursing staff recorded diarrhoea when present. We defined diarrhea as one or more liquid stools per hour during three hours. Plate culture of faeces was made. Only catheters, stools and blood were cultured during the first twelve hours.

Patients with two or more positive blood cultures (for the same microorganism) sampled during the first twelve hours were included in the bacteremic group (B.G).

Results

All patients (33 cases) were comatose at the time of hospital admission (mean Glasgow coma scale = 3).

Thirteen patients had at least two positive blood cultures. The following microorganisms were isolated: streptococcus D (5 cases), Escherichia coli (3 cases), pseudomonas aeruginosa (1 case), acinetobacter (1 case), enterobacter (1 case), enterobacter with clostridium perfringens (1 case) and staphylococcus epidermidis with staphylococcus aureus (1 case). In the last case, the same organisms were found on the intravascular tip of the central venous catheter (subclavian) which was probably the origin of the bacteremia. All other catheter tips were negative. Initial catheters were peripheral lines in 30/33 cases and all new inserted lines were central lines. Insertions outside hospital were made in an emergency with few aseptic precautions. The twelve other bacteremic patients had fetid diarrhoea during three to five hours following CA, in no case was this hemorragic; faeces were not checked for occult blood.

In all cases the same organism was found in both faeces and blood. The cause of cardiac arrest in patients who had diarrhea was: heart disease (7 cases), intoxication (2 cases), inhalation (1 case), drowning (1 case) and idiopathic (1 case). CA and CPR duration are shown in Table 1. The thirty three patients were not infected before CA according to the patients histories. There were no other obvious clinical source of infection exept for one patient who had a drown pneumonia.

The remaining 20 patients had no positive blood culture and no diarrhoea.

During the twelve hours following the admission, 7 patients (3 B.G) had fever (>38.5 °C), 6 patients (3 B.G) hypothermia (<36 °C) and 10 patients (7 B.G) had increased WBC (10000 cells/mm^3). Hypothermia or fever with leucocytosis was found in 6 cases (6 B.G).

Table 1. Comparison between bacteremic and non-bacteremic patients

	Bacteremic	Non-bacteremic
Number of patients	13	20
Mean age (years)	56±15	53±15 NS[a]
Time CPR (min)	6±2	6±3 NS[a]
Epinephrine dose (mg)	9±8	13±7 NS[a]
Ventricular fibrillation	5	4 NS[b]
Diarrhea	12	0 $p^b < 10^{-6}$
Hyperthermia (>38.5 °C)	3	4 NS[b]
Hypothermia (<36 °C)	3	3 NS[b]
Leucocytosis (>10000 cell/mm^3)	7	3 NS[b]
Death	12	15 NS[b]
Survivors		
– without sequelae	0	4 NS[b]
– chronic coma	1	1 NS[b]

[a] = student's *t*-test
[b] = CHI2 test with Yates correction

One day after CA, 22 patients (11 bacteremic) were febrile with a leucocytosis but without diarrhoea and without new bacteriological findings.

Only one patient (drowning pneumonia) received treatment with antibiotics (ureidopenicillin) on admission before the results of bacteriological sample and died 10 days later in spite of antibiotics effective against streptococcus D.

Twenty-seven patients died (mean 6±5 days, range 1–19); Post-morstems were not carried out. The death of the bacteremic patients was not due to septic shock. Six patients recovered (four without any neurologic sequelae). Those who survived never had initial diarrhea. The only one who survived in the bacteremic group (septicemia from catheter) remained in chronic coma.

There was no significant difference (Table 1) concerning age, etiology, estimated arrest time, duration of CPR and dose of epinephrine between the two groups.

Discussion

Resuscitation represents an extreme stress with a profound decrease of cell-mediated immunity [5]. To our knowledge bacteremias following diarrhea have not been reported as a complication of CA with resuscitation. In this study bacteremias occured with high frequency (13/33 = 39%). However, the previously observed decreased cellmediated immunity does not explain the increased risk of diarrhoea and sepsis in these patients.

The indwelling venous catheters, often inserted under unsterile conditions during CPR, may represent the main origin of septicemia. However, in this study catheterassociated bacteremia was the exception, probably because emergency cannulae were replaced early.

The micro-organisms cultured from the blood suggested an intestinal origin. Predominance of streptococcus D (5 cases) and Escherichia coli (3 cases) was compatible with the usual presence of these organisms in normal stools [6].

Bacteremia may be explained by intestinal leakage caused by mesenteric ischemia due to a low cardiac output [7]. During CPR the brain is preferentially perfused relative to other tissues [8]. The ratios of regional blood flow (using radioactive microspheres) during CPR to regional blood flow during sinus rythm were 90% for brain and 33% for small intestine [8]. During CPR the peripheral vascular effects of epinephrine transfer the available blood flow to the heart and brain [9]. Therefore the decreased intestinal blood flow caused by CA and epinephrine can induce mesenteric vascular insufficiency. All patients in our study had received epinephrine and the doses of epinephrine

used during CPR were important (mean 11 ± 7 mg). One case of nonocclusive mesenteric infarction has been reported [10] as a complication of CA with CPR.

Diarrhoea commonly occurs in patients treated in ICU [11] but its relation with septicemia following CA and CPR has not been reported. Further studies of such patients with histopathologic analysis of the gut may confirm the hypothesis of a leaky intestinal barrier.

In this study the 82% mortality (27/33) is quite similar to the published results concerning the same CA time [1]. However septicemias seem to be a bad prognostic factor since all the patients with septicemia following diarrhoea after CA died (12/12), but, the difference in mortality between the two groups is not significant. Bacteremia did not directly cause the death but has probably worsened the outcome. Patients who recovered without neurologic sequelae had never been septic and never had diarrhoea.

We conclude that septicemia during the first twelve hours following CA and CPR with epinephrine is frequent. The intestinal origin of septicemia may be explained by mesenteric ischemia caused by low cardiac output. Diarrhoea after CA seems to be associated with bacteremia and represents a risk of infectious complication and leads us to propose early use of antibiotic therapy. We feel that patients with continued diarrhoea after CA should receive antibiotics at least covering the gut flora. The use of calcium entry blockade proposed [12–14] to improve outcome after CA could possibly prevent mesenteric ischemia and perhaps septicemia, but this hypothesis has not been tested yet.

References

1. Abramson NS, Safar P, Detre KM, Kelsey SF, Monroe J, Reinmuth O, Snyder V (1985) Neurologic recovery after cardiac arrest: effect of duration of ischemia. Crit Care Med 13:930
2. Eisenberg MS, Hallstrom A, Bergner L (1982) Long-term survival after out-of hospital cardiac arrest. N Engl J Med 306:1340
3. Nagel EL, Fine EG, Krischer JP, Davis JH (1981) Complications of cardiopulmonary resuscitation. Crit Care Med 9:424
4. Powner DJ, Holcombe PA, Mello LA (1984) Cardiopulmonary resuscitation-related injuries. Crit Care Med 12:54
5. Soppi E, Lindroos M, Nikoskelainen J, Kalliomaki JL (1984) Effect of cardiopulmonary resuscitation-induced stress on cell-mediated immunity. Intensive Care Med 10:287
6. Gorbach SL (1971) Intestinal microflora. Gastroenterology 60:110
7. Aranhia GV, Goldberg NB (1981) Surgical problems in patients on ventilators. Crit Care Med 9:478
8. Voorhees WD, Babbs CF, Tacker WA (1980) Regional blood flow during cardiopulmonary resuscititation in dogs. Crit Care Med 8:134
9. Holmes HR, Babbs CF, Voorhees WD, Tacker WA, Degaravilla B (1980) Influence of adrenergic drugs upon vital organ perfusion during CPR. Crit Care Med 8:137
10. Morgan RJ, White PW, Temesy-Armos PN, Davis JT (1985) Treatment of nonocclusive mesenteric infarction after cardiopulmonary resuscitation. Crit Care Med 13:53
11. Kelly TWJ, Patrick MR, Hillman KM (1983) Study of diarrhea in critically ill patients. Crit Care Med 11:7
12. Andersson KE, Edvinson L, Mac Kenzie ET, Skarby T (1983) Influence of extracellular calcium and calcium antagonists on contractions induced by potassium and prostaglandin in isolated cerebral and mesenteric arteries of the cat. Br J Pharmacol 79:135
13. Vaagnenes P, Cantadore R, Safar P (1984) Amelioration of brain damage by lidoflazine after prolonged ventricular fibrillation cardiac arrest in dogs. Crit Care Med 12:846
14. White BC, Winegar CD, Wilson RF, Hoehner PJ, Trombley JN (1983) Possible role of calcium blockers in cerebral resuscitation: a review of the literature and synthesis for future studies. Crit Care Med 11:202

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