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Evolution of Bacterial Diversity in Burned Victims

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Abstract:

The death of a burned patient is most often caused by an infection, the vast majority of which are bacterial.The loss of the skin barrier, invasive devices and immunosuppression associated with the burn are three mechanisms contributing to the occurrence of these infections. In an inflammatory patient, the general infectious signs of infection are not very discriminating. Because of the severity of infections in this patient, their prevention is an essential management parameter. Due to the pharmacokinetic characteristics of burned patients, antibiotic dosages must be adapted and blood tests must be systematic. At a time when resistance is becoming obvious, research into alternative therapies (including virulence factor counteractants, antimicrobial peptides, polyphenols, immunotherapy, etc.) is crucial. We conducted a retrospective study over a period of 8 years, involving 30 burned patients hospitalized in the general surgery department of the regional military university hospital of Oran who all the patients showed an infection confirmed either by skin or blood sampling. Several germs were found with a clear predominance of *Pseudomonas aeregunisa* sensitive to ceftazidime and aother germs to imipenem. In the light of our results, we can say that in the burned patient, the antibiotic therapy must never be probabilistic, itmust be adapted to the bacteriological studies and the antibiograms.

Key words: Burns, Infection, Prevention, Treatment, Antibiotic Therapy.

الملخص

غالبا ما يكون سبب وفاة المريض الحروق عن طريق العدوى ولكن الغالبية منها هي البكتيريا. ان فقدان حاجز الجلد والأجهزة الغازية والتثبيط المناعي المرتب بالحرق هي ثلاث اليات تساهم في حدوث هذه الالتهابات. المريض الذي يكون تحت الالتهاب العلامات ليست شديدة العدوى. فان الوقاية منه هي ملمة أساسية للادارة. بسبب الحصائص الدوائية لمرضى الحروق، يجب تكييف جرعات المضادات الحيوية ويجب ان تكون اختبارات الدم منتظمة. في الوقت الذي أصبحت فيه المقاومة مقلقة، فان البحث في العلاجات البديلة (بما في دالك مضادات عامل الفوعة، البيتيدات المضادة للميكروبات، البوليفينول، العلاج المناعي، إلخ) أمر بالغ الأهمية. لتنضيم مقالنا، اجرينا دراسة بأثر رجعي على مدار 8 سنوات، شملت 30 مريضًا محترقًا في المستشفى في قسم الجراحة العامة في المسشفى الجامعي العسكري الإقليمي في وهران م الذين اصيبوا بعدوى مؤكدة إما عن طريق الجلد أو أخذ عينات الدم. تم العثور على العديد من الجراثيم ذات الغلبة الواضحة للازائفة الحساسة للسيفتازيديم وجميع الجراثيم الاخرى للإيميبينيم. بعد النتائج التي اليها، يمكننا القول إنه في المريض المصاب بالحرق، يجب ألا يكون العلاج بالمضادات الحيوية احتماليًا ابدا، ويجب أن يتكيف مع الدراسات البكتريولوجية و المضادات الحيوية.

الكلمات المفتاحية الحروق، العدوي، الوقاية، العلاج، العلاج بالمضادات الحيوية

Introduction

A burn is defined as a more or less extensive and more or less deep destructive of the "skin organ", under theaction of a thermal, electrical, chemical or radioactive agent (1). The severity of a burn is determined by three essential parameters: the total extent and proportion of a deep burn, the site of the lesion (in particular the faceand the perineum) and the physiological age of the patient (2). Infection in general and nosocomial infection in particular is a particularly frequent complication in severe burns, defined as any infection occurring more than 48 hours after the patient's admission. In addition to their frequency, infectious complications represent one of the first causes of mortality, once the acute phase of the serious burns patient is over. This high susceptibility to infection is mainly due to the loss of the skin covering, the first line of immune defense, andthe immune disorders induced by the initial aggression. The frequency of certain germs compared to others inpatients depends on the patient's normal resident bacterial flora, the length of hospitalisation, and the site of the infection. Initially sterile, the wound is rapidly contaminated after 48 hours by Gram-positive bacteria presenton the skin flora such as *Staphylococcus aureus*. After 72 hours, the wound is colonised by Gram-negative bacteria such as *Pseudomonas aeruginosa, Klebsiella pneumoniae* and *Escherichia coli.* And we are currentlywitnessing the appearance of increasingly virulent and antibiotic-resistant germs (3) (4).

In view of the frequency, severity and diversity of the germs and infection sites, we therefore carried out aretrospective study, the aim of which were to draw up a bacteriological profile of the infections found in ourpatients, then to evaluate the resistance to the used antibiotics , and finally to establish a protocol for the diagnosis and management of burn’s infections, both locally and generally.

Materials and methods

This is a retrospective study over a period of 8 years, from 1 January 2010 to 31 December 2017. We proposed to study infections in 30 patients admitted to the general surgery department of the regional military university hospital of Oran.

We included in this study all patients hospitalised for burns, infected during their hospitalisation or admittedwith infection.

We excluded all patients with signs of local and/or general infection that were not bacteriologically confirmed.

The 20 men and 10 women ranged between 18 and 74 years, with an average of 26 years. The burned bodysurface area varied between 15 and 30%, with an average of 20%. Flame burns accounted for 70% of the aetiologies.

The different types of samples are not systematically taken at the time of hospitalisation, generally carried outbetween d6 and d10 after installation of the SIRS. Local swabs are taken at the time of dressing-up each day.

When there are clinical signs of local infection, including changes in the appearance of the burn,The change in color of the burned area and adjacent areas, or the deepening of the burn.
Blood cultures were taken if at least 2 SIRS criteria were present.

Table 1. The French Society for the Study andTreatment of Burns SFET

	The French Society for the Study and Treatment of Burns (SFETB)2 criteria at least
Temperature	<35.5°C or >39.5°C
FC cardiac frequency	> 150% of base frequency
EN respiratory frequency	> 150% of base frequency
Leucocytes	Duplicated or deviated /2
CRP creativity protein	Not quoted

Bacterial identification

In our study, bacterial identification was performed by the VITEK2 compact 15 (Bio Mérieux).

Orientation tests

For each positive culture, we performed a few orientation tests to help us choose the correct identification cards for the VITEK2 machine.

Gram stain

In the present study, the main purpose of Gram staining is to distinguish Gram+ bacteria from Gram- bacteria in order to choose the correct colorimetric identification reagent card used by the VITEK2 (GP card or GN card).[2]

Oxidase test

This test enables the cytochrome oxidase enzyme of bacteria to be identified. This test has been performed onall Gram-bacilli in order to determine whether we are dealing with fermentative bacteria (enterobacteria; oxidase negative) or non-fermentative bacteria (genus *Pseudomonas*, genus *Bulkholderia*...).

Catalase test

The enzyme catalase is used to catalyse the degradation of hydrogen peroxide (H_2O_2) to H_2O and 1⁄2 O_2. This test involves reacting a bacterial colony with a drop of HO_2 hydrogen peroxide solution. The appearance of air bubbles indicates that the bacteria are catalase +. We have used this test with Gram+ cocci isolates in order to differentiate between *staphylococci* (catalase +) and *streptococci* (catalase -).

Principle

The VITEK 2 is a fully automated system for rapid and accurate identification and susceptibility testing. This equipment is fully automatic and allows the identification of bacteria and yeasts through biochemical tests that it performs by itself.

Technical

After ensuring the purity of the strains, we proceeded to their identification by preparing a bacterial suspension. The preparation of the inoculum is always manual and we will list it as follows:

- Take dry tubes for Vitek 2 and insert them into the wells of the cassette
- Place 3ml of the Vitek 2 saline solution into each tube using the dispenser set to 3ml.
- From the pure agar culture (young culture 24 h), with a pipette, the isolated colonies are selected andput in homogeneous suspension in the previous tubes and mixed well with the vortex.
- This bacterial suspension is standardised according to the appropriate methods using the DensichekPlus,

measuring the bacterial concentration at 0.5 McFarland.

- Take an Identification card (GP or GN) and place it on the cassette by dipping the transfer straw intothe tube.
- Load the cassette into the incubation chamber and close the door.
- The "start filling" button is pressed; an light indicator shows after 70 seconds that the filling cycle is complete.
- The cassette is removed from the inoculation chamber and placed inside the incubator reader (within10 minutes).
- A light indicator shows that card loading is complete. The empty cassette is removed from the reader-incubator.
- Enter the isolate information (number assigned to the laboratory).

The results are read by software associated with the Vitek 2 compact machine (Advanced Expert System ™ (AES™)). The results come out in the form of a table printed by the Vitek computer.

Results

During this period, 146 bacteriological samples were taken. 30 patients received a sample, i.e. 43% of all hospitalized patients. These included 56 blood cultures and 90 local samples

A- Blood cultures

Table 2: Sensitivity of the main germs found in blood cultures

Germs	Peni M	CEFTAZIDINE	IMIPENEME	GENTA	AMIKA	CIPRO
Staphylococcus	R	R	S	R	S	S
Pseudomonas*aeruginosa*	-	S	S	R	S	S
Enterococcusfeacalis	-	R	S	R	R	R

On the blood cultures taken from the 15 patients who presented with SIRS[i]. The antibiotic susceptibility of the different germs most frequently isolated is reported in table 1.

The most common germ found was S *taphylococcus* in 8 patients, our strain was sensitive to imepeneme,amikacyne and ciprolon and resistant to ceftazidine, beta lactamine and gentamicyne.

Pseudomonas was found in 5 patients; our strain was sensitive to almost all antibiotics tested.*Enterococcus feacalis* was found in 2 patients, our strain was sensitive to imipenem.

B-Skin samples

Germs	CEFTAZIDINE	IMIPENEME	AMIKA	CIPRO
Pseudomonasaeruginosa	S	S	S	S
Staphylococcus	R	S	**S**	S
Acenitobacter	R	S	-	R

All samples were positive. The incidence of the different germs found in the local samples is summarised inTable 2. The most frequently found germs were *pseudomonas* and *staphylococcus*, and our strains were sensitive to most of the antibiotics tested.
Acenitobacter was present in 4 patients, our strain was sensitive to imipenem.

The correlation between the results of blood cultures and local samples, only 25 blood cultures were associated with a skin sample and only one third of the positive blood cultures were associated with the presence of the same germ in the local sample. Over the study period, there were 01 case of death from infection.

Probabilistic antibiotic therapy, when necessary, should be started as soon as bacteriological samples are taken, at best within one hour of the development of infectious signs.

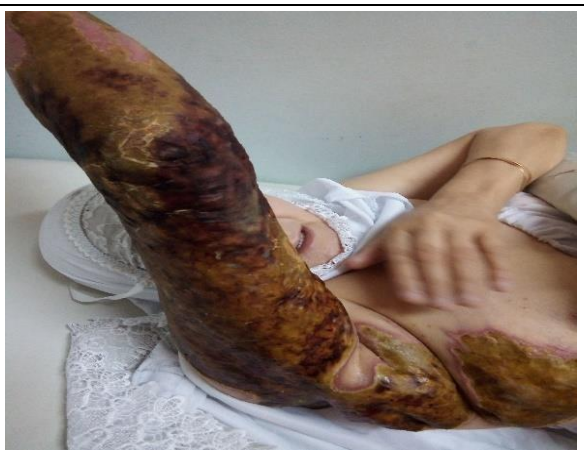

Fig 1. HMRUO (oran military hospital) Neglected burn infected after traditional treatment (egg yolk)

Discussion

The burn is an excellent medium for bacterial culture. B urn infection is therefore a necessary and inevitable phenomenon.
But when it is excessive, it must be feared because of its local and general consequences. It is therefore important to determine the invasive or non-invasive nature of the microbial outbreak.

The microbiological surface of the skin examinations carried out in this study, while providing rapid qualitative results, are not very reliable because of the high frequency of false positives and false negatives.

Infection diagnosis in burns as well as infection detection in burns requires a great deal of practice and dailyexamination of the burn and the burn, without which there is no hope of treating a burn (5).

Clinical and biological signs of infection are the change in the equilibrium obtained that indicates an infectious complication. Fever is always present in the evolution of a burn, as well as hyperleukocytosis, the sudden variation of both are interesting signs (5).

Bacteriological sampling is therefore essential. Swabbing is the most widespread practice, and it allows the germs on the surface to be identified, especially when the lesion is exuding. On the other hand, in dry lesions, these samples may be falsely negative or unrepresentative of the infecting flora that lies beneath the

eschar. For this reason, tissue biopsies are taken and the product is ground up: the presence of 10 (5) germs per gram of tissue is correlated with a significant sepsis risk (6-7).

The microbial flora varies from one centre to another. However, there are two predominant genera: *staphylococci* (38%) and pyocyanins (22%). Chronologically, the first infection is most often pulmonary, themedian time of onset being 10 days (8).

Antibiotic therapy prescribed to prevent burn infection does not prevent burn infection and promotes the emergence of multidrug-resistant bacteria. Local topicals are effective in preventing or treating burn infection. Therefore, local infection should only be treated locally, with surgery being an important part ofthe treatment. When accompanied by general signs of infection, the infectious process is no longer considered to be purely local and the use of antibiotics may be justified (9)

Furthermore, in the results obtained, it was noted the high incidence of positive blood cultures (55%) as

well asthe frequency of *staphylococcus* (37.5%) seconded by *Pseudomonas aeruginosa* (18.5%), for the antibiotic treatment chosen it was according to the sensitivity of the germs found.

Badetti (10) reported in a similar study that *staphylococcus* are most frequently isolated in the first week ofhospitalisation, with a clear predominance of *Pseudomonas aeruginosa* after the eightth day.

With regard to the resistance of germs to different antibiotics, it is difficult to compare our results with thoseof the literature.

However, there is some concordance as less than 83% of pyocyanins remain susceptible to cephalosporins.

Z.darfaoui (11) in his study on infections in burn victims had objectified the dominance of *staphylococcus*(53%) followed by P*seudomonas aeruginosa* (27%) which is in agreement with our study, these germswere sensitive to glycopeptides and aminosides.

Conclusion

In total, the mortality of burn patients who are supposed to be salvageable is mostly due to bacterial infectious causes. The most effective way to avoid them is to cure the patient of his burn by early excision and grafting. While the implementation of "standard" hygiene precautions is unanimously supported, "protective" isolation is more often mentioned. The diagnosis of infection is difficult in these patients whose intense inflammatory reaction mimics the clinic of sepsis, while the classic biological markers are often missed. Antibiotic therapy requires dosages and modes of administration (continuous infusion) that go far beyond the usual recommendations, and must be backed up by monitoring of plasma concentrations. At a time when therapeutic impasse is becoming not exceptional in the face of increasingly resistant bacteria, the use of other therapeutic means should be considered. However, their clinical application is not to be expected in the very near future, although phagotherapy seems to have a head start here

References

Arturson G. 1992 Analysis of severe disasters. In: Massellis M, Gunn S,editors. The Management of Mass Burn Casualties and Fire Disasters:Proceedings of the First International Conference on Burns and FireDisasters. Dordrecht: Kluwer AcademicPublishers; pp. 24-33.

Badetti C, Beyiha G, Garabedian M, Bernini V, Nicoli E, Gombert A, Manelli JC. 1993. Abstract sur la la surveillance des infections nosocomiales dans un centre des brules sur 19 mois. [Google Scholar]

Berrocal Revueltas M, Mendoza IE, Patron Go mez A. 1998. Ana'lisisestadýstico de pacientes con quemaduras, asistidos en la consulta deurgenciasdelHospitalUniversitario de Cartagena (Colombia). CirurgýaPlasticaIberoLatinoamericana.; pp. 403-407.

Dai T, Huang Y, Sharma S, Hashmi J, Kurup D, Hamblin M. 2010. Topical antimicrobials for burn wound infections. Recent Pat Antiinfect Drug Discov.;5:124-51.

Derfaoui.z .2018. Infections in burn patients: epidemiological, clinical and therapeutic data Thesis No. 065 pp:20-23.

Hanlon G. 2007. Bacteriophages: An appraisal of their role in the treatment of infection. Int J Antimicrob Agents. 30:118-28.

H. Carsin ', L. Bargues, J. Stéphanazzi, A. Paris, P. Aubert, H. Le Béver Centre de traitement des hrfilés. h6pilal d 'inst,-ucricn des armées Pet-cv. 92140 Clamart,

Lyngdorf P. 1986 Epidemiology of severe burn injuries. Burns InclThermInj;12(7) pp:491-5

Marco A, Hoyos F. 2006. Epidemiological and clinical profile of burn victims Hospital Universitario San Vicentede Paul, Medellın, 1994-2004, Burns; 32: 1044-51

Magnotti L, Deitch E. Burns, 2005. bacterial translocation, gut barrier, and failure. J Burn Care Res.; 26:383-pp11.

Oleksiewicz M, Nagy G, Nagy E. 2012. Anti-bacterial monoclonal antibodies: Back to the future? ArchBiochem Biophys.;526:124-31.

Wurtz R, Karajovic M, Dacumos E, Jovanovic B, Hanumadass M. 1995.Nosomial infections in a burn intensive care unit. Burns 21:181