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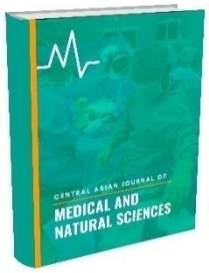

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IRAQIBACTER AS A NEW EMERGING PATHOGEN

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Abstract: As a result of its rapid development of resistance to routinely used antimicrobials, Iraqibacter (Acinetobacter baumannii) has emerged as a major hospital infection. It has lately come to light as a major reason why troops returning from Iraq and Afghanistan suffer from crippling soft tissue infections. It is widely believed, and supported by the available research, that the increased rates of infection are attributable to the widespread contamination of hospitals worldwide by A. baumannii, which is becoming more resistant to antimicrobials.

Key words: Iraqibacter, MDR, Hospital.

Introduction

In the aftermath of the 2003 US invasion of Iraq, reports emerged from US military surgeons of an "invisible enemy" they dubbed "Iraqibacter." The name related to Acinetobacter boumannii, a bacteria responsible for life-threatening illnesses among injured American service members. Following allegations that the bacterium had been carried back to the United States from U.S. field hospitals in Iraq via military evacuation lines, there was a media frenzy. Non-military patients in the US were also infected with Iraqibacter (1).

The United States military had been the primary source of information on A. baumannii in Iraq until very recently. In a somewhat perplexing turn of events, the armed forces have blamed the lower

wounded-to-killed ratio of previous conflicts for the increase in such diseases among the armed services as a whole. In hospitals, environmental organisms are more often brought in by patients with wounds (2). Some have hypothesised that the contamination of hospitals treating wounded service members and civilians has contributed significantly to the spread of this disease and its subsequent evolution towards drug resistance (3)

History

In order to get chemotherapy or staged reconstructive surgery for injuries sustained in Iraq, some patients were making the monthly journey between Iraq and Beirut. Patients and their loved ones were searching for reliable medical treatment outside of Iraq's damaged healthcare system (4).

These trips were part of a larger pattern of medical tourism that helped define a "therapeutic geography" of war by linking areas of conflict in Iraq, Libya, Gaza, Syria, as well as Humanitarian medical facilities in Yemen in neighboring countries like Lebanon, Turkey, Jordan, Iran, and India (5).

According to the medical records, at least half of the Iraqi patients at this Beirut hospital had multidrug-resistant illnesses., the most prevalent of which was caused by Acinetobacter baumannii. Similar infection patterns were seen among patients wounded in the current Syrian civil conflict who were treated at the same hospital, with A. baumannii being the only exception. War zones, such as Syria (6), Gaza (7), Yemen (8), and Libya (9), have seen a rapid spread of this disease, according to reports from regional hospitals and humanitarian organisations (9). Long-term financial and public health burdens are placed on patients and health systems due to the widespread problem of drug-resistant bug colonisation, which compounds the difficulty of treating a large number of war-wounded patients in civilian and humanitarian hospitals across the region.

However, the Iraqis I spoke with were conflicted as to what exactly was to blame for these tenacious diseases; they pointed fingers at everything from "pollution" to "insecurity" to "corruption" to "the US" to "sectarianism" to "stress" to "Saddam" and more. Their stories were sometimes vague and hard to follow, but they all pointed to the poisonous past and ecology that had a hand in moulding their daily lives as a contributing factor to their illnesses (10).

Motivated by guesswork, speculation, and new anthropological evidence on the impacts of conflict and damage in the area, a variety of historical and environmental hypotheses might be used to solve the puzzle of Iraqibacter and its evolving resistance to treatment in combat circumstances. Such theories are based on the environmental and microbiological remnants of previous military activities. The "biology of history" theory put out by scientific historian Hannah Landecker serves as the foundation for her investigation of antibiotic resistance in the post-industrial age (11).

Resistant to antimicrobials

Multi-drug resistance in A. baumannii is a major issue for hospitals, physicians, and military health care workers today. When an organism is resistant to three or more antibiotics, the CDC calls it multidrug-resistant (MDR) (12). A. baumannii's resistance to many antibiotics is not surprising. In a medical context, it is surrounded by antibiotics and often found in close proximity to other gram-negative bacteria.

Therefore, it has developed, in addition to its innate talents, a remarkable variety of resistance mechanisms. Plasmids, integrons, and transposons allow Acinetobacter baumannii and other gram-

negative bacteria to gain novel strategies. Studies have shown that many A. baumannii outbreak strains carry a class 1 integron, which is an interesting finding. Eighty-eight percent of A. baumannii strains capable of producing a biofilm were found to have the class 1 integron, which is responsible for the transfer and recruitment of numerous resistance genes. The presence of class 1 integrons in epidemic strains has also been related to treatment centres for returning service members in the United States and the United Kingdom (13,14).

Enzymes are a kind of resistance that is shared by A. baumannii and other gram-negative bacteria.

The processes we've examined so far allow the transmission of the genes that code for these enzymes from one cell to the next. The enzyme beta-lactamase is widely distributed and is responsible for hydrolyzing penicillins, cephalosporins, and carbapenems (15) .

Additionally, A. baumannii may acquire the fluoroquinolone and aminoglycoside resistance-promoting enzymes acetyltransferases, phosphotransferases, and nucleotidyl transferases. It is also possible for bacteria to pick up mutated genes from one another. Antimicrobial drug targets in bacteria may be mutated, decreasing the drug's affinity for the bacterium and raising the drug's MIC. Point mutations may occur anywhere in a gene, for as in the gyrA or parC genes. A higher minimum inhibitory concentration (MIC) for all fluoroquinolones would be expected from the isolate if point mutations were present in both genes (16).

The bacterium Acinetobacter baumannii has its own set of innate defences. Eflux pumps and porins are two such examples. Small metabolites including ions, sugars, and amino acids (17) are transported over the outer membrane by proteins called porins. Antibiotics may pass through cell membranes more easily when there are more porins present.

Researchers have hypothesised that the low quantity and relatively large size of porins in the outer membrane of Acinetobacter baumannii contribute to the bacterium's inherent resilience (18). Three porins (19) are absent in A. baumannii strains that have developed resistance to imipenem, one of the few medicines still effective against the vast majority of strains.

More study is needed to clarify the significance of these various porins and their link to antibiotic resistance. A. baumannii's efflux pump is yet another remarkable mechanism. In order to avoid being killed by antibiotics, bacteria have efflux pumps that aggressively pump them out of the cell. Efflux pumps and porins working together are thought to be a potent resistance mechanism (20). The dread of every clinician may be summarised by considering the consequences of a strain of A. baumannii with mutations in the gyrA and parC genes, efflux overexpression, and a lack of porins (21).

Some medications may be active against the A. baumannii germs that cause such havoc in intensive care units, despite everything going on within and outside the organism's cell walls. Monobactams, and some aminoglycosides, fluoroquinolones, polymixins, sulbactams,carbapenems, as well as glycylcyclines (2) are all examples. However, there have been several instances of resistance due to carbapenem-hydrolyzing beta lactamases (22-23) despite the fact that carbapenems have been the major therapy for MDR A. baumannii isolates. Therefore, it is essential that susceptibility testing be performed on each and every isolate to guarantee timely and appropriate treatment and to avoid excessive morbidity (6).

Diagnosis of this bacteria

When dealing with a hospital-acquired illness, it is crucial to treat the condition promptly and properly.

As soon as possible, the cause of the infection must be determined so that treatment may begin. When Acinetobacter is in its stationary phase, it takes the form of a coccobacillus, but during times of fast development, it becomes a rod-like morphology (15). Negative for oxidase and indole but positive for catalase and hemolytic. Because of its adaptability to different nutrient sources, Acinetobacter may be grown at 44°C on standard laboratory medium (15).

Standard laboratory methods may determine the genus, but not the species (24). Species-level identification of an isolate may now be done rapidly and correctly using automated approaches. Phenotypic techniques use biochemistry and assimilation tests, which are traditionally performed by hand, to identify the genus as well as bacterial species in a shorter amount of time. Microscan WalkAway (25-26) is one example of an analyzer that might be used to detect an A. baumannii isolate.

Although these analyzers will incorrectly identify the isolate as A. baumannii, they will correctly identify the A. baumannii complex, which will assist the practitioner make the best treatment options (25).

Molecular testing is another technique that may be used for identification. The genotype of an isolate may be determined using molecular testing. Pulsed-field gel electrophoresis (PFGE) and 16s rRNA sequencing (26-27) are two examples of modern molecular techniques employed in labs. Gel electrophoresis of the pulsed-field kind can separate DNA molecules up to around 2000 kb in size. In this method, there are three electric fields used rather than 1.58 Similar to standard electrophoresis, one electric field travels along to the gel's centre axis, while the other two run at an angle of 120 degrees on each side. The enormous DNA molecule is gradually disentangled by pulsing the electric fields alternately. With a resolution of up to 1 bp, pulsed-field gel electrophoresis may be used to distinguish between bacterial strains, which is highly valuable in tracking the spread of germs during an epidemic (28).

16s ribosomal DNA (rDNA) sequencing is a second molecular technique used for bacterial identification. This technique involves isolating DNA from bacterial colonies and amplifying it using primers that target just the 16s rDNA. After the rDNA is sequenced, it may be compared to other Acinetobacter genotypes in GenBank and other public sequence databases. In addition to pinpointing the origin of HAIs, the molecular techniques may help hospitals improve their infection control practises by revealing where they are falling short (29).

Conclusion

Because of the overuse and misuse of antibiotics, Acinetobacter baumannii has developed into a dangerous hospital infection. Instances of mass hospital trauma admissions and greater movement of patients and personnel from one hospital to another have been linked to its spread, as have war and natural catastrophes.

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