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Antibiogram-derived radial decision trees: Innovative visual educational tools for discussing empirical antibiotic selections

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Abstract

This article describes a unique visual pedagogical tool that displays appropriate empirical antimicrobial treatment choices for anticipated pathogens. This new tool, a probability-based radial decision tree (RDT), transforms data from a traditional hospital antibiogram into a format that can be used as an epidemiologic tool, a guide to empiric antimicrobial therapy and a robust educational tool for display of therapy options for pan-susceptible and drug-resistant isolates. As well-described resistance mechanisms serve the basis for the antimicrobial treatment choices in the RDT, use of this tool provides a means for both displaying treatment choices and explaining the rationale for such options in a logical and systematic manner. Teaching fellow pharmacists and pharmacy students with this tool provides both verbal and visual cues that may allow more efficient conveying of key concepts important to understanding options for treatment of select pathogens.

Keywords: *Antibiogram, decision-trees, pedagogy, susceptibility*

Introduction

Pharmacists often function as front-line educators and enforcers of antimicrobial management policies in institutional settings (Owens, Fraser, & Stogsdill, 2004; MacDougall & Polk, 2005). Because of their therapeutic knowledge and drug dispensing function, their role in antimicrobial resistance control and prevention efforts is well recognized (Goldmann et al., 1996; Shlaes et al., 1997; Lawton, Fridkin, Gaynes, & McGowan, 2000). Additionally, pharmacists are increasingly being recognized for their ability to provide input in infectious diseases pharmacotherapy choices (Ibrahim, Gunderson, & Rotschafer, 2001; Rapp, 2006). In light of the increasing infectious diseases-related pharmacy practice opportunities, it is important for pharmacists to develop an intimate understanding of the information guiding infectious diseases pharmacotherapy decisions that impact on patient care.

If a pharmacist is to gain mastery of infectious diseases pharmacotherapy, knowledge of information in the areas of infectious diseases, microbiology and antimicrobials is required. (Moellering & Eliopoulos, 2005). Each of these areas contains large volumes of complex information which is increasingly becoming complicated by the escalating crisis of drug-resistant organisms in both hospital and community settings (Goldmann et al., 1996; Shlaes et al., 1997; Chambers, 2005; Levy & OBrien, 2005). Hence, when assisting with the development of infection-related patient care plans, pharmacists must possess an understanding of anticipated antimicrobial resistant mechanisms and their impact on anticipated susceptibility profiles, particularly when a pathogen has yet to be identified (Moellering & Eliopoulos, 2005).

Because institution-specific antibiograms are used by clinicians to guide empirical antimicrobial selections, they theoretically could be used by pharmacy educators to incorporate practical and clinically-relevant information into their discussions of antimicrobial pharmacotherapy. However, antibiogram

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displays (typically large two-factor tables comparing organism–antimicrobial susceptibility combinations) allow discussion of important epidemiological trends (characterizing susceptibility patterns of bacterial species over time) (Clinical Laboratory and Standards Institute/NCCLS, 2005a), but they have less utility as educational tools that provide a rationale for well-known alternative therapy options in situations where resistant subpopulations must be considered. For example, if an educator wishes to describe the rationale behind treatment options for a patient in whom there is concern about infection with a ceftazidime-resistant *Pseudomonas aeruginosa* isolate, the typical antibiogram will not display information that allows prediction of the susceptibility of such an isolate to a carbapenem.

In an attempt to increase the practical and educational utility of antibiogram data, we developed a strategy to transform hospital antibiogram data into a probability-based radial decision tree (RDT). Previously, we described the methods involved in developing the RDT and detailed its utility as an epidemiological tool and a guide to empirical therapy choices for drug-resistant subpopulations (Perla & Belliveau, 2005). However, the RDT also serves as a robust visual educational tool that displays first and second-line antimicrobial options for specific organisms. Since the educational value of the RDT is maximized when these options are discussed in the context of the resistance mechanisms that guide them, the current article includes a discussion of the resistance mechanisms that serves as the basis for the RDT structures. In this discussion, key "take home" concepts are identified, supporting information for these concepts is provided and the therapeutic implications of this information is presented in the context of the RDT.

Materials and methods

RDTs were developed for *Staphylococcus aureus* and *P. aeruginosa*, microbial species known to exhibit resistance to different classes of antimicrobials and are often associated with difficult to treat nosocomial infections. RDTs could also be developed for other pathogens to be used as tools for educators. Susceptibility information from HealthAlliance Hospital (a medium-sized, non-urban, community hospital in Central Massachusetts) was incorporated into the RDTs for demonstration purposes. This institution uses standard susceptibility testing and reporting procedures (Clinical Laboratory and Standards Institute/NCCLS, 2005a,b; Perla & Belliveau, 2005).

The rationale for the selection and reporting of each antimicrobial agent (and its placement in the RDT) is discussed in detail in the text below. However, since the purpose of this article is to demonstrate the educational utility of the RDT as a guide to help explain empirical therapy of suspected drug-resistant subpopulations when teaching pharmacists and pharmacy students, we have not attempted to address all clinically relevant findings revealed in a typical antibiogram. Indeed, antimicrobial resistance is a complex phenomenon that can be discussed from many different viewpoints (Courvalin, 2005) and our descriptions of antibiotic resistance are representative (but certainly not exhaustive) of the types of issues that should be considered in making empirical antimicrobial selections.

Results

Radial structure and decision trees

The radial structure of the antibiogram-derived decision tree addresses the fact that empiric (as well as culture-guided) antimicrobial therapy can have different starting points relative to the types of agents considered first-line and second-line therapy. Figure 1 demonstrates the basic structure of the RDT model. The center circle represents the microbial species in question. The first circular level around the microbial species (level 1) represents antimicrobials that are typically considered first-line agents or agents whose susceptibility should be considered during the initial selection of an antimicrobial agent for that organism. For example, when treating a suspected or known *S. aureus* infection, the oxacillin susceptibility should be an initial concern that influences therapy selection. The second level in the RDT (level 2) addresses therapeutic options when resistance to the first line agents is known or suspected. In the RDT, the susceptibility values (i.e. percent susceptible or resistant) are provided on the branches in the tree. In as much as level 2 addresses resistant microbial subpopulations of a given species, this level represents the most difficult and challenging therapeutic situations. Although not presented here, a third level of empirical decision making could be added to the RDT to explore and teach more complex treatment scenarios both in the classroom and in practice.

With the RDT, the anticipated prevalence of a pathogen's susceptibility profile as it relates to more than one antibiotic can be determined. The *in vitro* (*a posteriori*) probability of encountering an isolate with a specific phenotype can be calculated by multiplying the susceptibility percentages of any branch on the tree together. Additionally, if a clinician suspects resistance to a level 1 agent (i.e. based on patient location or history), the RDT allows for an educated prediction of susceptibility to level 2 agents for that resistant subpopulation. To demonstrate this principle, let us look at a simple example, the *S. aureus* RDT. Observation of this RDT reveals that the probability of encountering a *S. aureus* isolate that is oxacillin-resistant *and* vancomycin-susceptible

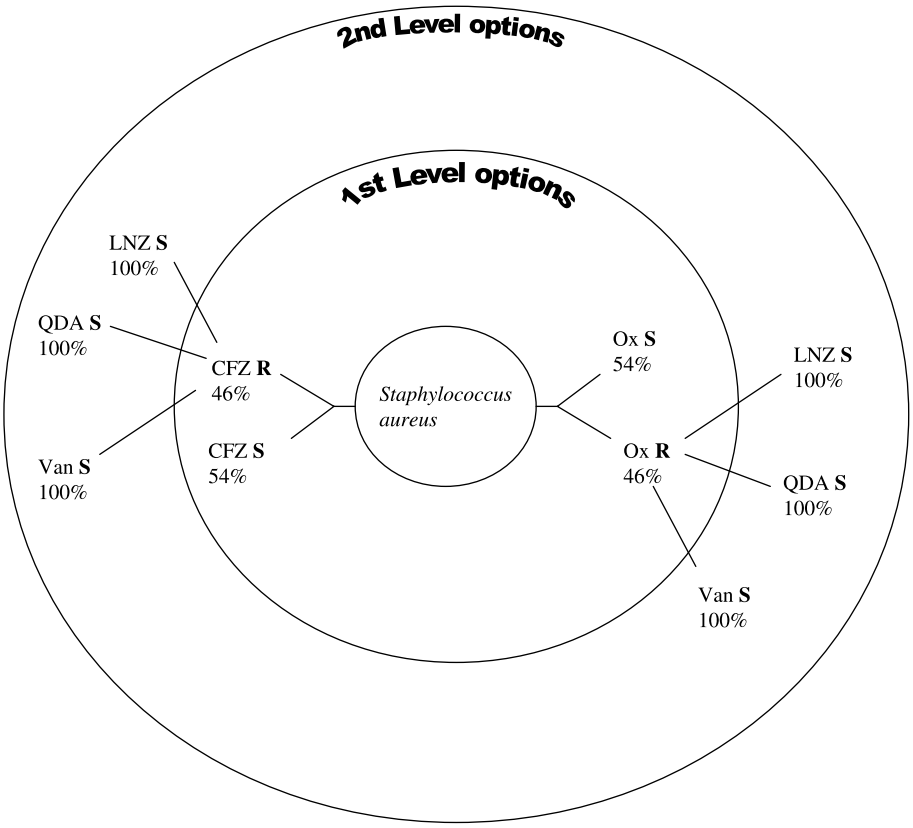

Figure 1. RDT for *S. aureus*. Ox, oxacillin; CFZ, cefazolin; Van, vancomycin; QDA, quinupristin/dalfopristin; LNZ, linezolid; S, susceptible; and R, resistant. Reproduced from Perla & Belliveau 2005, with Permission of Science Publications.

in Figure 1 is determined by the following calculation:

$$\text{oxacillin resistance } (0.46)$$
$$\times \text{ vancomycin susceptible } (1.0)$$
$$= 0.46$$

Alternatively, there is little chance of encountering an oxacillin-resistant, vancomycin-resistant isolate (oxacillin resistance (0.46) × vancomycin resistant (0) = 0). While these examples provide information that is simple and somewhat intuitive (and does not necessarily require a calculation) since resistance to level 2 antibiotics in the *S. aureus* RDT is presently considered a rare event (Jones, 2003; Wilson et al., 2003; Ruef, 2004; Peeters & Sarria, 2005), this calculation demonstrates the multiplicative probability susceptibility profiling that is a feature of the RDT (but is not addressed in traditional antibiograms).

The *P. aeruginosa* RDT is more complex from a therapeutic decision-making and *in vitro* susceptibility testing standpoint and may better demonstrate the value of multiplicative probability as it relates to susceptibility data (see Figure 2). Utilizing the same type of calculation described with the *S. aureus* RDT, the probability of encountering a *P. aeruginosa* isolate that is ceftazidime-resistant and imipenem-susceptible is 0.21. This could be compared to the probability of encountering-a ceftazidime-resistant, imipenem-resistant isolate (probability of 0) or a ceftazidime-susceptible isolate (0.79) to help assist with empirical treatment decisions. Alternatively, when choosing a second gram-negative antibiotic to combine with ceftazidime, one could determine the probability of encountering an isolate that is: gentamicin-susceptible (0.74); gentamicin-resistant and amikacin-susceptible (0.23); levofloxacin-susceptible (0.70); or levofloxacin-resistant and ciprofloxacin-susceptible (0).

Educational utility; the rationale for antibiotic choices

The selection of specific antibiotics and their placement in the *S. aureus* or *P. aeruginosa* RDT are based on the interrelatedness of resistance mechanisms among different antimicrobials, established practice guidelines and the peer reviewed literature in clinical microbiology and infectious diseases. Consideration of each antibiotic in the RDT was based on the antibiotic's indication to treat infections typical for each pathogen. As with traditional antibiogram data, the RDT does not account for infection location, severity of infection, or other important patient-dependent factors (such as β-lactam allergies) that may affect treatment choices. Our intention is to introduce a different and unique model for representing susceptibility data for use

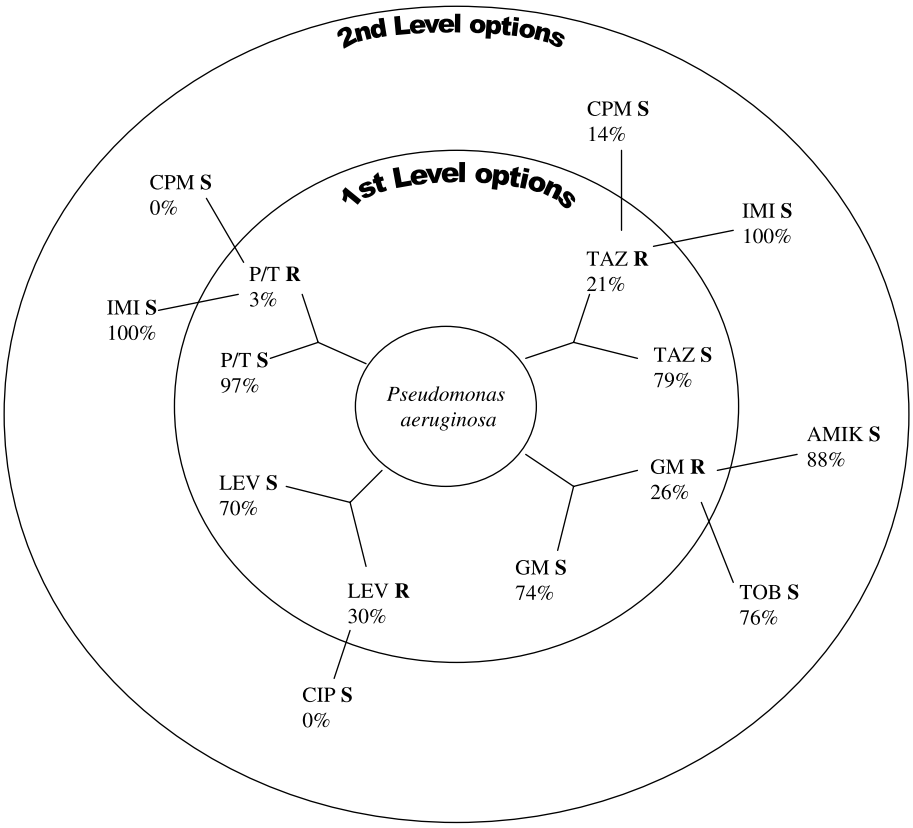

Figure 2. RDT for *P. aeruginosa*. LEV, levofloxacin; GM, gentamicin; TAZ, ceftazidime; P/T, piperacillin/tazobactam; CIP, ciprofloxacin; TOB, tobramycin; AMIK, amikacin; IMI, imipenem; CPM, cefepime; S, susceptible; and R, resistant. Reproduced from Perla & Belliveau 2005 with permission of Science Publications.

in pedagogical settings. As other clinicians and epidemiologists may choose different antibiotics in a particular RDT based on institutional variations in resistance trends, clinical experiences, and formulary preferences, our model is one of many possible RDT models that may be developed and modified over time.

The RDTs were developed to convey several key concepts related to choosing antimicrobial therapy for *S. aureus* and *P. aeruginosa* infections. The following section highlights theses concepts, supporting information and their therapeutic implications as they might be presented in pedagogical situations.

Concept #1. Staphylococcus aureus *expresses a β-lactamase enzyme which inactivates many, but not all, β-lactam antimicrobials*

Supporting information. *S. aureus* produces a β-lactamase enzyme that hydrolyzes the β-lactam ring of susceptible β-lactam antimicrobials, rendering them devoid of clinically useful antimicrobial activity (Livermore, 1995a; Moreillon, Que, & Glauser, 2005). Although all β-lactam antimicrobials are substrates for this enzyme, the extent of susceptibility to hydrolysis varies among different β-lactam antibiotics. Natural penicillins, aminopenicillins and anti-pseudomonal penicillins are rendered inactive by this enzyme. Conversely, penicillinase-resistant penicillins (nafcillin, oxacillin) and cefazolin (one of the more commonly prescribed first-generation inpatient IV cephalosporins) are relatively stable in the presence of this enzyme (Livermore, 1995a; Kucers, Crowe, Grayson, & Hoy, 1997). Despite qualitative differences in the degree of stability for these latter antibiotics, the clinical significance of such differences is of dubious consequence as suggested by the performance standards for antimicrobial susceptibility testing developed by the Clinical and Laboratory Standards Institute (CLSI) (Clinical Laboratory and Standards Institute/NCCLS, 2005b). The CLSI considers penicillin-resistant, oxacillin-susceptible strains to be susceptible to penicillinase-resistant penicillins, β-lactamase inhibitor combinations and cephalosporin antibiotics (Clinical Laboratory and Standards Institute/NCCLS, 2005b). While the activity of penicillinase-resistant penicillins and cefazolin are based on their intrinsic stability, β-lactamase mediated resistance to β-lactamase labile antimicrobials may be circumvented by the administration of products that contain a β-lactamase inhibitor (i.e. sulbactam) that irreversibly binds these enzymes (Kucers et al., 1997).

Therapeutic implications. Although β-lactam antimicrobials are typical first line options for

antibiotic treatment of *S. aureus* infections (included as first level options in the RDT) (Mylonakis & Calderwood, 2001; Mermel et al., 2001; Moreillon et al., 2005; Stevens et al., 2005), the aforementioned well-described resistance mechanisms and patterns must be considered when prescribing such therapy, since the prevalence of β-lactamase production exceeds 80% and is widespread in both hospital and community strains of *S. aureus* (Livermore, 1995a; Chambers, 2001; Moreillon et al., 2005). Therefore, among the β-lactams, intravenous treatment options for methicillin-susceptible *S. aureus* infections consist of a penicillinase resistant penicillin, a first-generation gram-positive cephalosporin, or a β-lactamase inhibitor containing product. The first two groups are represented as level 1 options in the RDT (see Figure 1). It could be argued that β-lactamase inhibitor combinations (i.e. ampicillin/sulbactam) and other agents (i.e. clindamycin, macrolides) should be included in the *S. aureus* RDT. However, as these are typically not included among first-line agents in well respected sources (Mylonakis & Calderwood, 2001; Mermel et al., 2001; Moreillon et al., 2005; Stevens et al., 2005), it may be prudent to avoid the appearance of placing such agents on an equal standing with those that are typically used to treat *S. aureus* infections in hospitalized patients.

Concept #2. Target site changes is the mechanism by which S. aureus *isolates express resistance to penicillinase-resistant penicillins and cephalosporins*

Supporting information. Even though β-lactamase production among *S. aureus* isolates has long been considered a substantial therapy-limiting consideration, a greater and increasing concern is the problem of *S. aureus* resistance to penicillinase-resistant penicillins (Chambers, 2005; Moreillon et al., 2005). Organisms sharing this resistance phenotype were originally confined primarily to hospital environments. However, this problem is extending into the community and reaching epidemic proportions (Chambers, 2005). As can be seen from the RDT in Figure 1, the rate of oxacillin resistance at our institution is 46% making oxacillin resistance an important consideration in the selection of a therapeutic agent when treating known or suspected *S. aureus* infections.

S. aureus resistance to penicillinase-resistant penicillins is mediated by the *mec*A gene contained in a fragment of DNA (referred to as SCC*mec*; SCC stands for staphylococcal cassette chromosome) that has been integrated into the chromosome of these methicillin/oxacillin-resistant *S. aureus* (M/ORSA) isolates. *mec*A encodes for penicillin-binding protein 2a (PBP2a), an altered form of penicillin-binding protein 2 which can take over the cell wall formation activities of this organism. PBP2a has a lower affinity for and higher dissociation rates with penicillins, allowing cell wall formation to proceed efficiently enough to ensure survival in the presence of penicillin antimicrobials (Chambers, 1997; Stapleton & Taylor, 2002; Moreillon et al., 2005). Since these penicillin-binding protein changes affect binding by other β-lactam antimicrobials such as cephalosporins, the presence of this resistance mechanism also confers resistance to all agents in this class (Chambers, 1997; Stapleton & Taylor, 2002; Moreillon et al., 2005). Although *in vitro* susceptibility testing systems may sometimes report M/ORSA isolates as susceptible to some cephalosporins, these results are overridden by expert rule-based algorithms because of concerns with the heterogeneous expression of methicillin-resistance and a lack of clinical support for use of such agents for M/ORSA infections (Clinical Laboratory and Standards Institute/NCCLS, 2005b). This is why we see, and would expect to see, the same percentage of *S. aureus* resistance to oxacillin and cefazolin as we do in Figure 1. Since a β-lactamase inhibitor does not circumvent the issues posed by the presence of PBP2a, M/ORSA isolates are also resistant to such products (Chambers, 1997; Clinical Laboratory and Standards Institute/NCCLS, 2005b). Furthermore, because SCC*mec* also often contains genetic information encoding for resistance to non-β-lactam antimicrobials, a considerable amount of co-resistance to M/ORSA is exhibited with multiple classes of antimicrobials (Chambers, 1997; Diekema et al., 2001).

Therapeutic implications. In light of the resistance phenotypes observed in M/ORSA, treatment options become limited. With fewer treatment options, clinicians must consider a second level of therapeutic options that include vancomycin, linezolid, or quinupristin/dalfopristin (Mylonakis & Calderwood, 2001; Mermel et al., 2001; Moreillon et al., 2005; Stevens et al., 2005). These second level treatment options are represented in the outer circle of the RDT in Figure 1. Other potential treatment options (macrolides, clindamycin) may be added to this second level for M/ORSA isolates depending on local susceptibility profiles and prescribing patterns for treatment of M/ORSA infections.

Concept #3. Pseudomonas aeruginosa *resistance to β-lactam antimicrobials is typically mediated by β-lactamase production*

Supporting information. *P. aeruginosa* displays numerous resistance mechanisms, some of which are considered more common than others. Expression of resistance may be mediated by β-lactamase production, antimicrobial target site changes, as well as by mechanisms that reduce antimicrobial

accumulation within the organism (Livermore, 2002; Pier & Ramphal, 2005; Rossolini & Mantengoli, 2005). Resistance to the anti-pseudomonal β-lactam antimicrobials (piperacillin, ceftazidime, ticarcillin) is usually mediated by the chromosomal AmpC β-lactamase (Kucers et al., 1997; Hancock, 1998; Pier & Ramphal, 2005; Rossolini & Mantengoli, 2005), an enzyme which demonstrates varying substrate specificity for different antibiotics and inconsistent inhibition by β-lactamase inhibitors. Hence, *P. aeruginosa* resistance to piperacillin may not be circumvented with the use of piperacillin–tazobactam (Akova, Yang, & Livermore, 1990; Bryson & Brogden, 1994; Livermore, 1995b; Pfaller et al., 1997). Additionally, because of a greater resistance to AmpC β-lactamase hydrolysis, ceftazidime-resistant isolates may still be susceptible to cefepime and antipseudomonal carbapenems (Livermore & Yang, 1987, 1989; Fung-Tomc, Huczko, Pearce, & Kessler, 1988; Sanders C. C., Gates, & Sanders W. E., 1988; Yang & Livermore, 1989; Hancock & Bellido, 1992; Livermore, 1995b; Pfaller et al., 1997). However, significant qualitative differences in the stability of these agents exist. Cefepime stability can be overwhelmed by inoculum effects and high-levels of AmpC (Fung-Tomc et al., 1988; Fung-Tomc, Dougherty, DeOrio, Simich-Jacobson, & Kessler, 1989; Johnson et al., 1995; Limaye, Gautom, Black, & Fritsche, 1997). Carbapenems are much more stable in the presence of AmpC, and resistance is typically expressed as a result of an interplay of several resistance mechanisms (AmpC production, loss of porin channels, efflux pump expression, penicillin binding protein changes) (Livermore, 1992, 2001; Masuda et al., 1999; Pai et al., 2001; El Amin et al., 2005). This is consistent with the susceptibility data at our institution (level 2 in Figure 2) where only 14% of ceftazidime-resistant *P. aeruginosa* are susceptible to cefepime, while 100% of this *P. aeruginosa* ceftazidime-resistant subpopulation is susceptible to imipenem.

Therapeutic implications. Several β-lactam and non-β-lactam options exist for treatment of infections due to documented or suspected *P. aeruginosa* infections (Mermel et al., 2001; Hughes et al., 2002; American Thoracic Society (ATS) & Infectious Diseases Society of America (IDSA), 2005; Sobel & Kaye, 2005). The rationale for these choices can be explained by the resistance mechanisms described above. Initial choices typically involve either a piperacillin-containing product (piperacillin or piperacillin/tazobactam) or ceftazidime (see level 1 in Figure 2). For *P. aeruginosa* isolates resistant to piperacillin, piperacillin–tazobactam, or ceftazidime, the fourth-generation cephalosporin, cefepime (although unlikely to be susceptible), or an antipseudomonal carbapenem (such as imipenem) may be considered.

Concept #4. Aminoglycosides or fluoroquinolones are commonly combined with a β-lactam antimicrobial to treat P. aeruginosa infections. Known resistance mechanisms expressed to these antimicrobials allow prediction of the susceptibility to other agents in these classes when resistance to a first level option is anticipated

Supporting data and therapeutic implications. Although the need for dual antimicrobial therapy of *P. aeruginosa* infections has not been clearly established (Hilf et al., 1989; Siegman-Igra, Rovona, Primerman, & Giladi, 1998; Chatzinikolaou et al., 2000; Chamot, El Amari, Rohner, & Van Delden, 2003; Safdar, Handelsman, & Maki, 2004; Paul & Leibovici, 2005; Micek et al., 2005), such a practice is often listed as an option (Mermel et al., 2001; Hughes et al., 2002; ATS & IDSA, 2005; Sobel & Kaye, 2005). When indicated, an aminoglycoside is typically combined with a β-lactam antibiotic. Because of the better intrinsic activity against *P. aeruginosa* (Kucers et al., 1997), and the slightly better susceptibility observed in reports on clinical isolates of this organism (Poole, 2005), some institutions may prefer tobramycin over gentamicin when *P. aeruginosa* is documented or strongly suspected. However, recommendations for treatment of *P. aeruginosa* infections typically include either aminoglycoside as an option (Mermel et al., 2001; Hughes et al., 2002; ATS & IDSA, 2005; Sobel & Kaye, 2005). *P. aeruginosa* resistance to the aminoglycosides typically occurs as a result of plasmid- or chromosome-encoded aminoglycoside-modifying enzymes that phosphorylate, adenylate, or acetylate these antimicrobials (Poole, 2005). As with the β-lactamases, these enzymes demonstrate substrate specificity. The enzymes most commonly observed have specificity for gentamicin and tobramycin, leaving amikacin as an alternative for isolates expressing resistance via this mechanism (Poole, 2005). As can be seen in Figure 2, 88% of gentamicin-resistant *P. aeruginosa* in our institution are susceptible to amikacin. However, when resistance occurs as a result of aminoglycoside-efflux pumps (the second most common resistance mechanism in *P. aeruginosa* isolates), discrimination among the aminoglycosides may be less significant, resulting in panaminoglycoside resistant strains (Poole, 2005). In such situations, or for patients whose renal function precludes aminoglycoside therapy, a fluoroquinolone, such as levofloxacin or ciprofloxacin, might be combined with one of the above β-lactams (Hughes et al., 2002; ATS & IDSA, 2005; Sobel & Kaye, 2005). Some institutions may prefer ciprofloxacin as its first-line antipseudomonal fluoroquinolone because of its better intrinsic activity against *P. aeruginosa* (Kucers et al., 1997). However,

susceptibility, synergy and pharmacodynamic studies show these products to be similar in terms of their ability to kill this organism *in vitro* (Isenberg, Alperstein, & France, 1999; MacGowan, Wootton, & Holt, 1999; Jones, Beach, & Pfaller, 2001; Burgess & Nathisuwan, 2002; Pendland, Messick, & Jung, 2002; Friedland, Gallagher, King, & Woods, 2004) Consistent with the susceptibility information at our institution, published susceptibility surveys indicate a high degree of fluoroquinolone cross-resistance among *P. aeruginosa* strains (Jones et al., 2001; Friedland et al., 2004). When resistance is expressed to the fluoroquinolones, it typically is the result of chromosomal mutations causing changes in the fluoroquinolone target site (DNA gyrase) or the activation of efflux pumps (Jalal & Wretlind, 1998; Akasaka, Tanaka, Yamaguchi, & Sato, 2001).

Discussion

The RDT model and concept discussed in this article addresses important educational limitations inherent in typical antibiogram data displays that are routinely collected and analyzed in clinical microbiology laboratories and disseminated hospital-wide to assist with empirical antimicrobial selections. The RDTs presented here could easily be modified to reflect local susceptibility patterns, prescribing patterns, formulary choices and educational objectives. Below is a review of the benefits and limitations of the antibiogram-derived RDT.

Benefits of the radial decision tree

1. Reflects the logical thought process of antimicrobial selection that is influenced by knowledge of known resistance mechanisms and institution-specific resistance trends;
2. provides a quick visual "gestalt" of how clinically important drugs respond—and are likely to respond—to clinically significant microbial species;
3. provides a robust visual representation of complex data that could not be efficiently communicated in a traditional antibiogram without overcomplicating the display of information in the typical 2-factor table;
4. provides an opportunity to educate pharmacists and pharmacy students about logical empirical therapeutic decisions and resistance trends in their local environment;
5. provides additional epidemiological information regarding the prevalence of resistant microbial species to more than one agent at a time.

Limitations of the radial decision tree

1. Like traditional antibiograms, the RDT provides information (susceptibility data) generated from *in vitro* testing and serves as only a guide or reference point for the empirical treatment of infectious diseases;
2. as with traditional antibiogram data, the RDT does not account for infection location, severity of infection, or other important patient-dependent factors (such as β-lactam allergies) that may affect treatment choices;
3. the RDT developed and discussed here is not unit or location specific, but like traditional antibiograms the RDT could be constructed to reflect the susceptibility data and patterns in a particular unit or location;
4. like traditional antibiogram data, the RDT data and structure is limited to the agents tested in the clinical microbiology laboratory;
5. the RDT may not easily be used to address all clinically relevant treatment options as the degree of complexity that would be required in such a display may outweigh the pedagogical value of the model.

With the increasing challenge and complexity of antimicrobial susceptibility testing, analysis and reporting, communication of accurate and useful susceptibility data requires the interdisciplinary collaboration and efforts of microbiologists, epidemiologists, clinicians and pharmacists (Clinical Laboratory and Standards Institute/NCCLS, 2005a; Larson et al., 2005; Zapantis et al., 2005). The RDT model developed here is an example of this type of interdisciplinary approach, one that provides a useful therapeutic decision-making guide for drug-resistant subpopulations and serves as a relatively straightforward yet dynamic educational tool which allows pharmacists and pharmacy students to develop an intimate knowledge behind the rationale for these treatment choices.

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